Postgraduate Orthopaedics

The Candidate's Guide to the FRCS (Tr & Orth) Examination

Third edition

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The third edition of *Postgraduate Orthopaedics* is dedicated to the memory of Professor Andrew P. Sprowson. He was one of the strongest early supporters of *Postgraduate Orthopaedics* and shared the same goals and visions that founded the development of the book series. He intuitively recognised the importance of developing a UK-based orthopaedic textbook for UK graduates sitting their FRCS (Tr & Orth) examination, rather than having to rely on dissimilar North American counterparts. His basic science chapters in the *Postgraduate Orthopaedics Viva Guide* will be fondly remembered for their uniquely differing approach to a difficult dry subject area of the exam syllabus. He was very keen to continue his involvement with the book series for the third edition and co-authored two chapters. He will be sadly, yet fondly, remembered for his enthusiasm, energy and larger-than-life character.

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Foreword

It is a pleasure to be asked to write a foreword for the third edition of this now very well-known textbook. It builds on the tradition of the previous two editions and it is easy to see why *Postgraduate Orthopaedics* is the best-selling orthopaedic text in the UK. There are contributions from over 50 surgeons, and Banaskiewicz and Kader have edited it into a hefty but very readable single volume. The changes and additions make it even more comprehensive than the previous editions. It will continue to be an essential read for orthopaedic trainees especially those with the FRCS Orth looming. Its strength lies in the useful knowledge contained within the text and, moreover, for the invaluable advice on exam techniques that will help in the presentation of that knowledge.

The flavour of the book is unchanged, and the recipe is still very successful. There is freshness about this edition and a confidence in the presentation, which comes from the undoubted success of the first two editions. Dip into it, or read it from cover to cover. Enjoy.

Bill Ledingham, Aberdeen, March 2016

Preface to third edition

There was a big jump-up in detail and quality between the first and second editions of *Postgraduate Orthopaedics*, so when the second edition was released we felt confident we could rest up a little before we undertook a third edition.

After each new book release the general feeling for the first few months is that we will never do another book as we really don't need all the hassle all over again. However, events took over very quickly in that soon after the second edition's release it won a highly commended prize at the BMA book awards in London 2012. We realized that evening that a third edition was inevitable and probably would be needed sooner rather than later!

As time went by and the book's popularity increased we began to notice more closely deficiencies within the second edition. We needed some extra illustrations; some tidying-up of chapters that could have read better; and additional specific details for the trickier areas of the syllabus, like basic science.

The anatomy and surgical approaches section definitely needed more professional illustrations and the text also required polishing-up. The clinical section needed additional cases and more precise exam-focused details. Despite the significant learning potential of the old-style long case, the general consensus was that this material now had to be completely dropped. We decided to include an applied basic science chapter where we specifically worked on basic science viva questions in more detail. The trauma chapter needed expanding and for ease of purpose was broken down into separate sections.

Despite writing a separate paediatric book we still needed to review this section but without any unnecessary repetition of material.

The part one MCQ/EMI section was revised and updated but we decided against including large numbers of MCQ/EMI questions at the end of each chapter. This would have made the book unmanageable and is perhaps best saved for a later date in the future.

An interesting addition was the chapter on what books to read for the FRCS (Tr & Orth) exam. This had always been included in the general introduction chapter but just seemed to have outgrown it. This part of the chapter continually surprises us in how popular it is with candidates.

In short, each chapter has been thoroughly reviewed, revised and updated. We have again included a number of new contributors who have used the Postgraduate Orthopaedics book series to pass the exam. The continued success of the book relies on involving these newly qualified trainees who keep the book relevant and current.

We again make no claim for the originality of the text. We are distilling orthopaedic knowledge from the wider orthopaedic community specifically for exam-related subjects and material.

Our popular examination corner section had to be tweaked. For various reasons we have omitted any new second-hand accounts of specific detailed examination dialogues. This has been compensated for by *Postgraduate Orthopaedics* now running its own courses which provide similar material for use.

A special word of thanks again to Cambridge University Press for their help and support. The grass is not always greener elsewhere.

Finally as we become more established in our clinical practice we may have said 'poacher turned game keeper' a few years ago but now prefer the term 'gatekeeper'. It will be interesting to see if we make it to a fourth edition.

Paul A. Banaszkiewicz
Deiary F. Kader

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Our special thanks to Caitlin Monney and Emily McDougall for drawing illustrations at short notice for the surgical approaches and basic science chapters. Likewise, special thanks to Steve Atkinson who photographed a large portion of the orthopaedic implants, again at very short notice. Publishing a book is much more than just writing the text!

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Abbreviations

1,2-ISCRA	1,2-intercompartmental supraretinacular artery
A&E	Accident and Emergency
AA	ankle arthroplasty
AACP	American College of Chest Physicians
AADI	anterior atlanto-dens interval
AANS	American Association of Neurological Surgeons
AAOS	American Academy of Orthopaedic Surgeons
ABC	airway, breathing, circulation
ABPI	Ankle : Brachial Pressure Index
AC	acromioclavicular
ACDF	anterior cervical decompression and fusion
ACEA	anterior centre edge angle
ACL	anterior cruciate ligament
AD	autosomal dominant
ADI	atlanto-dens interval
ADL	activities of daily living
ADM	abductor digiti minimi
AER	apical ectodermal ridge
AFO	ankle–foot orthosis
AIDS	acquired immunodeficiency syndrome
AIIS	anterior inferior iliac spine
AIN	anterior interosseous nerve
AIS	Abbreviated Injury Scale
AITFL	anterior-inferior tibiofibular ligament
AJCC	American Joint Committee on Cancer
AKP	anterior knee pain
ALL	anterior longitudinal ligament
ALVAL	aseptic lymphocyte-dominated vasculitis-associated lesions
AM	anteromedial
ANOVA	analysis of variance
ANT	artery–nerve–tendon
AOFAS	American Orthopaedic Foot and Ankle Society
AP	anteroposterior
APB	abductor pollicis brevis
APTT	activated partial thromboplastin time
AR	autosomal recessive
ARCO	Association Research Circulation Osseous
ARDIS	adverse reaction to metal debris
ARDS	acute respiratory distress syndrome
ARMD	adverse reactions to metal debris
ARR	absolute risk reduction
AS	ankylosing spondylitis
ASA	American Society of Anesthesiologist
ASB	anatomical snuffbox
ASIA	American Spinal Injury Association
ASIS	anterior superior iliac spine
ATD	articular–trochanteric distance
ATFL	anterior talofibular ligament
ATLS®	Advanced Trauma Life Support®
ATP	adenosine triphosphate
AVN	avascular necrosis
BDGF	bone-derived growth factor
bFGF	basic fibroblast growth factor
BHR	Birmingham Hip Resurfacing
BMD	bone mineral density
BMES	bone marrow oedema syndrome
BMG	bone matrix gelatin
BMI	Body Mass Index
BMP	bone morphogenetic protein
BMU	basic multicellular unit
BOA	British Orthopaedic Association
BOAST	British Orthopaedic Association Standards for Trauma
BP	Buechel–Pappas
BPI	brachial plexus injury
BPTB	bone–patella–tendon–bone
BR	brachioradialis
BW	body weight
CAP	Clubfoot Assessment Protocol
CC	costoclavicular
CCT	Certificate of Completion of Training
CDH	congenital dislocation of the hip
CEA	carcinoembryonic antigen
CEO	common extensor origin
CESR	Certificate of Eligibility for Specialist Registration
CFL	calcaneofibular ligament
CFO	common flexor origin
CI	Clearance Interval
CIA	carpal injury adaptive
CIC	carpal instability complex
CID	carpal instability dissociative
CIND	carpal instability non-dissociative
CJD	Creutzfeldt–Jakob disease
CL	capitolunate
CMAP	compound muscle action potential
CMC	carpometacarpal
CME	continuing medical education
CMT	Charcot–Marie–Tooth
CMV	cytomegalovirus
CNS	central nervous system
CNS	Congress of Neurological Surgeons
CO_2	carbon dioxide
CoC	ceramic on ceramic
CoP	ceramic on polyethylene
COPD	chronic obstructive pulmonary disease
COR	centre of rotation
CORA	centre of rotational angular

Abbreviations

CP	cerebral palsy
CPM	continued passive motion
CPN	common peroneal nerve
CPPD	calcium pyrophosphate dihydrate
CR	cruciate retaining
CRP	C-reactive protein
CRPS	complex regional pain syndrome
CSF	cerebrospinal fluid
CSISS	Cervical Spine Injury Severity Score
CSM	cervical spondylotic myelopathy
CT	computed tomography
CT2	core trainee 2
CTEV	congenital talipes equinovarus
CVA	cerebrovascular accident
CVP	central venous pressure
DAB	dorsal abducts
DCO	damage control orthopaedics
DCP	dynamic compression plate
DCS	dynamic condylar screw
DD	Dupuytren's disease
DDH	developmental dysplasia of the hip
DEXA	dual energy x-ray absorptiometry
DHS	dynamic hip screw
DI	dorsal interosseous
DIC	dorsal intercarpal
DIP	distal interphalangeal
DIPJ	distal interphalangeal joint
DISH	diffuse idiopathic skeletal hyperostosis
DISI	dorsal intercalated segment instability
DMAA	distal metatarsal articular angle
DMARDs	disease modifying anti-rheumatoid drugs
DP	distal phalanx
DRC	dorsal radiocarpal
DRUJ	distal radioulnar joint
DV	dorsoventral
DVT	deep vein thrombosis
ECA	extensor compartment artery
ECM	extracellular matrix
ECR	extensor carpi radialis
ECRB	extensor carpi radialis brevis
ECRL	extensor carpi radialis longus
ECU	extensor carpi ulnaris
ED	emergency department
ED	extensor digitorum
EDB	extensor digitorum brevis
EDC	extensor digitorum communis
EDL	extensor digitorum longus
EDM	extensor digiti minimi
EDQ	extensor digiti quinti
EF	external fixation
EGF	epidermal growth factor
EHL	extensor hallucis longus
EI	extensor indicis
EIP	extensor indicis proprius
EJS	effective joint space
EMG	electromyography
EMIs	extended matching items
ENT	ear, nose, throat
EPB	extensor pollicis brevis
EPI	epicondylitis
EPL	extensor pollicis longus
EQA	Examination Quality Assessment
ER	external rotation
ERCB	extensor carpi radialis longus
ESR	erythrocyte sedimentation rate
ESWL	extracorporeal shock wave lithotripsy
ETC	early total care
ETC	early trauma care
EtO	ethylene oxide
ETO	extended trochanteric osteotomy
EUA	examination under anaesthesia
FABER	flexion, abduction and external rotation
FAI	femoroacetabular impingement
FAST	focussed assessment with sonography in trauma
FBC	full blood count
FCF	four-corner fusion
FCR	flexor carpi radialis
FCU	flexor carpi ulnaris
FDB	flexor digitorum brevis
FDG	fluorodeoxyglucose
FDL	flexor digitorum longus
FDP	flexor digitorum profundus
FDQ	flexor digiti quinti
FDS	flexor digitorum superficialis
FEAR	flexion, extension, abduction and adduction, external and internal rotation
FFD	fixed flexion deformity
FFP	fresh frozen plasma
FGF	fibroblast growth factor
FGF23	Fibroblast Growth Factor 23
FGFR3	Fibroblast Growth Factor Receptor gene 3
FHB	flexor hallucis brevis
FHL	flexor hallucis longus
FPA	foot progression angle
FPB	flexor pollicis brevis
FPL	flexor pollicis longus
FTA	foot–thigh angle
GA	general anaesthetic
GAGs	glycosaminoglycans
GCS	Glasgow Coma Score
GCT	giant cell tumour
GHJ	glenohumeral joint
GHL	glenohumeral ligaments
GI	gastrointestinal
GMC	General Medical Council
GMFCS	Gross Motor Function Classification System
GRAFO	ground reaction ankle foot orthosis
GRF	Ground Reaction Forces
GT	greater trochanter
HA	hyaluronic acid
HA	hydroxyapatite
HAGL	humeral avulsion of inferior glenohumeral ligament
HbA1c	glycated hemoglobin
HBO	hyperbaric oxygen
HEA	Hilgenreiner's epiphyseal angle
HHS	Harris Hip Score
HIV	human immunodeficiency virus
HMSN	hereditary motor sensory neuropathies
HNP	herniated nucleus pulposus
HO	heterotopic ossification
HOOD	hereditary osteo-onychodysplasia
HOTS	higher order thinking skills
HPT	hyperparathyroidism
HPV	human papillomavirus
HRT	hormone replacement therapy
HSMN	hereditary motor and sensory neuropathies
HTO	high tibial osteotomy
HU	Hounsfield units
HV	hallux valgus

HVA	hallux valgus angle
ICD	International Classification of Diseases
ICSRA	intercompartmental supraretinacular artery
IDGF	insulin-derived growth factor
IGF	insulin-like growth factor
IGHL	inferior glenohumeral ligament
Ihh	Indian hedgehog
II	image intensifier
IL	interleukin
IM	intramedullary
IMA	intermetatarsal angle
IMHS	intramedullary hip screw
IMN	intramedullary nailing
IMT	intermetatarsal
INR	International Normalized Ratio
IOTFL	interosseous tibiofibular ligament
IP	interphalangeal
IPJ	interphalangeal joint
IPR	inferior peroneal retinaculum
IQR	interquartile range
IR	internal rotation
ISAKOS	International Society of Arthroscopy, Knee Surgery and Orthopaedic Sports Medicine
ISB	Intercollegiate Specialty Boards
ISS	Injury Severity Score
ITB	iliotibial band
ITU	Intensive Care Unit
IV	intravenous
IVC	inferior vena cava
IVP	intravenous pyelogram
JBJS	*Journal of Bone and Joint Surgery*
JCA	juvenile chronic arthritis
JCIE	Joint Committee on Intercollegiate Examinations
JRA	juvenile rheumatoid arthritis
JRF	joint reaction force
KAFO	knee–ankle–foot orthosis
KD	knee dislocation
LA	local anaesthetic
LAT	lateral
LBP	low back pain
LCDCP	low-contact dynamic compression plates
LCE	lateral centre edge
LCEA	lateral centre-edge angle of Wiberg
LCFA	lateral circumflex femoral artery
LCL	lateral collateral ligament
LCP	low compression plates
LCS	low contact stress
LFTs	liver function tests
LHB	long head of biceps
LIPUS	low intensity pulsed ultrasound
LISS	less invasive stabilisation system
LLA	lateral longitudinal arch
LLD	limb length discrepancy
LMN	lower motor neuron
LOTS	lower order thinking skills
LRL	long radiolunate ligament
LRP5	low-density lipoprotein receptor-related protein 5
LRTI	ligament reconstruction tendon interposition
LTL	lunotriquetral ligament
LUCL	lateral ulnar collateral ligament
MACTAR	McMaster-Toronto Arthritis patient Preference Disability Questionnaire
MARS	metal artifact reduction sequence
MBD	metastatic bone disease
MC	metacarpal
MCFA	medial circumflex femoral artery
MCL	medial collateral ligament
MCP	metacarpophalangeal
MCQs	multiple choice questions
MCSF	macrophage-colony stimulating factor
MDP	methylene diphosphonate
MDT	multidisciplinary team
MED	multiple epiphyseal dysplasia
MEN	multiple endocrine neoplasia
MEPs	motor-evoked potentials
MESS	Mangled Extremity Severity Score
MFC	medial femoral condyle
MFH	malignant fibrous histiocytoma
MH	medial hamstring
MHRA	Medicines and Healthcare products Regulatory Agency
MIC	minimum inhibitory concentration
MIPO	minimally invasive plate osteosynthesis
MLA	medial longitudinal arch
MMP	metalloproteinase
MOA	mechanism of action
MOA	mode of action
MoM	metal on metal
MoP	metal on polyethylene
MP	migration percentage
MPFL	medial patellofemoral ligament
MR	magnetic resonance
MRA	MR arthrogram/ arthroscopy
MRC	Medical Research Council
MRI	magnetic resonance imaging
MS	multiple sclerosis
MSTS	Musculoskeletal Tumor Society
MSU	monosodium urate
MT	metatarsal
MTC	Major Trauma Centre
MTP	metatarsophalangeal
MTPJ	metatarsophalangeal joint
MTP-PE	muramyl tripeptide phosphatidylethanolamine
MUA	manipulation under anaesthetic
MUPs	motor unit potentials
NAI	non-accidental injury
NAP	nerve action potential
NBM	nil by mouth
NCS	nerve conduction studies
NDI	Neck Disability Index
Nf	neurofibromatosis
Nf-1	neurofibromatosis type 1
Nf-2	neurofibromatosis type 2
NICE	National Institute for Health and Clinical Excellence
NIPE	Newborn Infant Physical Examination
NJR	National Joint Registry
NOF	Neck of femur
NOGG	National Osteoporosis Guideline Group
NPV	Negative Predictive Value
NSA	neck shaft angle
NSAIDs	non-steroidal anti-inflammatory drugs
OA	osteoarthritis
OBPI	obstetric brachial plexus injury
OCD	osteochondritis dissecans
OCL	osteochondral lesions
ODEP	Orthopaedic Data Evaluation Panel
ODF	osteoclast differentiation factor (aka RANK ligand)
OI	osteogenesis imperfecta
OITE	Orthopaedics In-Training Exam

Abbreviations

OK	Outerbridge and Kashiwagi
ON	osteonecrosis
OP	opponens pollicis
OP	out-patient
OPG	osteoprotegerin
OPLL	ossification of the posterior longitudinal ligament
ORIF	open reduction internal fixation
ORL	oblique retinacular ligament
OSCAR	Orthosonics System for Cemented Arthroplasty Revision
PA	posterior anterior
PAO	periacetabular osteotomy
PCL	posterior cruciate ligament
PCP	perimeniscal capillary plexus
PD	proximodistal
PDGF	platelet-derived growth factor
PDP	personal development plan
PE	polyethylene
PE	pulmonary embolism
PEEK	polyetheretherketone
PER	pronation–external rotation
PET	positron emission tomography
PF	patellofemoral
PFFD	proximal focal femoral deficiency
PFO	proximal femoral osteotomy
PGE_2	prostaglandin E_2
PICU	paediatric intensive care unit
PIN	posterior interosseous nerve
PIP	proximal interphalangeal
PIPJ	proximal interphalangeal joint
PIS	pinning-in-situ
PITFL	posterior-inferior tibiofibular ligament
PJI	periprosthetic joint infection
Pl	palmaris longus
PL	posterolateral
PLAD	posterior lip augmentation device
PLC	posterolateral corner
PLIF	posterior interbody lumbar fusion
PLL	posterior longitudinal ligament
PLRI	posterolateral rotatory instability
PMMA	polymethylmethacrylate
PNET	primitive neuroectodermal tumour
POP	plaster of Paris
PP	proximal phalanx
PPV	Positive Predictive Value
PQ	pronator quadratus
PR	per rectum
PRC	proximal row carpectomy
PROSTALAC	prosthesis of antibiotic-loaded acrylic cement
PRP	platelet-rich plasma
PRUJ	proximal radioulnar joint
PS	posterior stabilised
PSA	prostate specific antigen
PSIS	posterior superior iliac spine
PSO	pelvic support osteotomy
PT	pronator teres
PT	prothrombin time
PTFL	posterior talofibular ligament
PTH	parathyroid hormone
PTHrP	Parathyroid hormone-related peptide
PVD	pelvic disease
PVD	peripheral vascular disease
PVL	Panton–Valentine leukocidin
PVNS	pigmented villonodular synovitis
RA	rheumatoid arthritis
RANKL	receptor activator of nuclear factor kB ligand
RC	radial collateral
RCT	randomised controlled trial
RHK	rotating-hinge knee
RICE	rest, ice, compression and elevation
RL	radiolunate
RLL	radiolucent line
RLT	radiolunotriquetral
ROM	range of movement
ROTEM	rotational thromboelastometry
RR	relative risk
RRR	relative risk reduction
RS	radioscaphoid
RSA	radiostereometric analysis
RSA	reverse total shoulder arthroplasty
RSC	radioscaphocapitate
RSD	reflex sympathetic dystrophy
RSL	radioscapholunate ligament
RSWP	radial side wrist pain
RT	radiotriquetral
RTA	road traffic accident
RTSA	reverse shoulder arthroplasty
RU	radioulnar
RUL	radioulnar ligaments
RVAD	rib vertebral angle difference
SAC	space available for the cord
SAC	Specialty Advisory Committee
SACH	solid ankle cushion heel
SAS	specialty and associate specialist
SBA	single best answer
SC	sternoclavicular
SC	supracondylar
SC	synovial chondromatosis
SCA	single correct answer
SCC	squamous cell carcinoma
SCD	sickle cell disease
SCFE	slipped capital femoral epiphysis
SCIWORA	spinal cord injury without radiological abnormality
SD	standard deviation
SEMLS	single event multiple level surgery
SEPs	sensory-evoked potentials
SER	supination external rotation
SERMs	selective oestrogen receptor modulators
SHH	sonic hedgehog
SI	sacroiliac
SJ	sternoclavicular
SL	scapholunate
SLAC	scapholunate advanced collapsed
SLAP	superior labrum from anterior to posterior
SLE	systemic lupus erythematosus
SLL	scapholunate ligament
SLR	straight leg raise
SMAC	Standing Medical Advisory Committee
SNA	stem-neck angle
SNAC	scaphoid non-union advanced collapsed
SNAP	sensory nerve action potential
SOL	space occupying lesion
SPECT	single photon emission computed tomography
SPN	superficial peroneal nerve
SPORT	Spine Patient Outcomes Research Trial
SPR	superior peroneal retinaculum
SRS	Scoliosis Research Society
SSA	stem-shaft angle
SSEPs	somatosensory evoked potentials
SSSC	superior shoulder suspensory complex

ST	scaphotrapezial
ST3	surgical trainee year 3
ST6	surgical trainee year 6
STAR	Scandinavian total ankle replacement
STC	Specialist Training Committee
STIR	short tau inversion recovery
STR	soft-tissue realignment
STS	soft-tissue sarcoma
STT	scaphotrapeziotrapezoid
SUFE	slipped upper femoral epiphysis
TA	tibialis anterior
TAD	tip apex distance
TAL	transverse atlantal ligament
TAR	total ankle arthroplasty
TB	tuberculosis
TBW	tension band wiring
TCC	total contact casting
TEA	total elbow arthroplasty
TENS	transcutaneous electrical nerve stimulation
TER	total elbow replacement
TFA	thigh–foot angle
TFC	triangular fibrocartilage
TFCC	triangular fibrocartilage complex
TGF	transforming growth factor
TGF-β	transforming growth factor-beta
THA	total hip arthroplasty
TIMPs	tissue inhibitory metalloproteinases
TKA	total knee arthroplasty
TKR	total knee replacement
TLHKAFO	thoraco–lumbar–hip–knee–ankle–foot orthosis
TLICS	Thoraco-Lumbar Injury Classification and Severity Score
TLIF	transforaminal lumbar interbody fusion
TLSO	thoracolumbar spinal orthosis
TMA	transmalleolar thigh angle
TMJ	temporomandibular joint
TMT	tarsometatarsal
TNF	tumour necrosis factor
TORCH	toxoplasmosis, other, rubella, cytomegalovirus, herpes simplex
TP	tibialis posterior
TT	tibial tubercle
U&E	urea and electrocytes
UCL	ulnar collateral ligament
UFD	unilateral facet dislocation
UHMWPE	ultra-high-molecular-weight polyethylene
UKA	unicompartmental knee arthroplasty
UKITE	UK in Training Examination
UKR	unicompartmental knee replacement
UL	ulnolunate ligament
UMN	upper motor neuron
US	ultrasound
USMLE	United States Medical Licensing Examination
USS	ultrasound scan
USWP	ulnar side wrist pain
UT	ulnotriquetral ligament
UTI	urinary tract infection
UV	ultraviolet
VACTERL	vertebral, anorectal, cardiac, tracheal, oesophageal, renal and limb
VATER	vertebral, anorectal, tracheal, oesophageal, renal
VIP	vasoactive intestinal polypeptide
VISI	volar intercalated segment instability
VITO	valgus intertrochanteric osteotomy
VMO	vastus medialis obliquus
VP	ventriculoperitoneal
VTE	venous thromboembolism
VVC	varus–valgus constrained
WBC	white blood cell
WCC	white blood cell count
WHO	World Health Organization
WOMAC	Western Ontario and McMaster Universities Arthritis Index
Y-TZP	yttria-stabilised tetragonal zirconia particles
ZPA	zone of polarizing activity

Interactive website

The website to accompany the book

www.postgraduateorthopaedics.com

This website accompanies the textbook series: Postgraduate Orthopaedics.

It includes:

- *Postgraduate Orthopaedics: The Candidates Guide to the FRCS (Tr & Orth) Examination*, third edition
- *Postgraduate Orthopaedics: Viva Guide for the FRCS (Tr & Orth) Examination*
- *Postgraduate Paediatric Orthopaedics*

The aim is to provide additional information and resources in order to maximize the learning potential each book.

Additional areas of the website provide supplementary orthopaedic material, updates and web links. *Meet the editorial team* provides a profile of authors who were involved in writing the books. There is also a list of Postgraduate Orthopaedics courses available for candidates to fine-tune their examination skills. Details of the next diet of exams is also provided.

There is a link to additional orthopaedic websites that are particularly exam focused.

It is very important our readership gives us feedback. Please email us if you have found any errors in the text that we can correct. In addition, please let us know if we haven't included an area of orthopaedics that you feel we should cover. Likewise, any constructive suggestions for improvement would be most welcome.

Chapter 1

General guidance

Niall Breen, Benjamin W. T. Gooding and Jonathan R. A. Phillips

The FRCS (Tr & Orth) is the major obstacle in higher surgical training. It is regarded as a fair but very probing examination. Passing depends on knowledge, performance on the day and a bit of luck. However, as with all exams, preparation is the key to success. That preparation should encompass not only reading to accumulate facts, but should include clinical experience, history taking, clinical examination and, most of all, practice.

The exam constantly evolves and opinions and views continually move forward and change. We hope the chapter acts as an introduction to the current format of the FRCS (Tr & Orth) exam and serves to provide prospective candidates with some useful preparation tips and tricks.

Examination format

The current FRCS (Tr & Orth) encompasses two sections. Section 1 is the written test and section 2 is the clinical exam.

The Joint Committee on Intercollegiate Examinations (JCIE) published regulations in 2012 that govern the current FRCS (Tr & Orth) examination. Candidates have 7 years to complete the examination process. For section 1, candidates will have a 2-year period from their first attempt, with a maximum of four attempts with no re-entry. If successful, they can then proceed to section 2, where candidates have a maximum of four attempts and up to one further exceptional attempt.

For further details and to ensure no further changes have been made following this publication, we suggest all candidates carefully review the JCIE website[a].

Section 1: The written test

The written section of the exam covers the 'theory' of trauma and orthopaedics and is comprised of two separate computerized papers sat back to back on the same day. Paper 1 is a two-hour long Single Best Answer (SBA) paper whereas paper 2 is made up of Extended Matching Items (EMIs) over 2.5 hours.

The statistical analysis of an orthopaedic paper that was previously part of paper 1 is no longer a part of the exam. Candidates will still be expected to know about statistics and methodology and this will be tested in either the paper 1 SBA or the paper 2 EMI section. We are unsure why it was scrapped, perhaps it was more difficult to standardize from exam to exam and/or it did not prove to be a good differentiator of candidates. Possibly it may have been too time consuming to construct a separate statistical section for each diet of exams with the time better invested in building up a more substantial SBA/EMI bank.

This section is delivered via computer-based testing at Pearson VUE Test Centres throughout the UK and Ireland. This environment can be unsettling, with people taking their driving theory test either side of you, although once absorbed in the exam this shouldn't be a concern. It is possible to finish this exam before the time ends and you can leave once you are happy you have completed it.

These papers may probe any part of the vast T&O syllabus. A solid knowledge of the theory is required, but exam technique is also essential for this part, which can only be developed through practice questions. Preparation for section 2 is very different and requires a change in revision strategy, but the basic knowledge learned from section 1 is extremely important and should not be underestimated.

Section 2: Clinicals and orals (vivas)

This section comprises clinical cases and structured oral interviews (also known as 'vivas' – The terms being interchangeable for the purpose of this book but referred to officially as orals by the Intercollegiate Specialty Board). This section is held usually at a hospital for the clinical component on day 1 and a nearby hotel or conference venue for the oral component.

The clinical component is broken down into three upper and three lower limb short cases, each of 5 minutes' duration (30 minutes in total) and two intermediate cases of 15 minutes each (which can be upper limb, lower limb or spine).

The oral component, comprises four, 30-minute orals in:

- Adult elective orthopaedics, including spine
- Trauma, including spine
- Paediatric orthopaedics/hand and upper limb
- Applied basic sciences related to orthopaedics, including anatomy and surgical approaches, pathology, biomechanics, audit, methodology and outcome-based medicine

[a] https://www.JCIE.org.uk

The current exam format has now had time to bed in and provides a fairer and more structured assessment than previous iterations. Candidates should feel confident that the process is predictable and can focus on displaying their knowledge rather than being caught out.

In the past, the spot diagnosis was a significant part of the clinical component. Some candidates could move through numerous cases if they made quick diagnoses whilst others would only see a few cases. The current format of short cases ensures that 5 minutes is spent with each of the six cases. For example, in a case of Dupuytren's contracture you may state the diagnosis within seconds, but you will still be expected to examine the hand, discuss issues such as indications for surgery and consent, as you will not move on until the 5 minutes is up.

Marking

Many candidates waste valuable time fretting over the complexities of the marking system for the FRCS exam. It is important to note that the scoring systems used are devised by statisticians and educationalists and standardized by the examiners, with the intention of making the marking as reproducible and as fair as possible. Rather than worrying, your time is better spent reading, practicing your examination technique and your ability to deliver succinct answers in a viva situation.

There is no set percentage pass rate; the examiners meet the evening before to set the standard and establish a cut-point for passing or failing candidates. This is a standardized method for marking examinations and if you are interested in the theory behind this, please refer to references at the end of the chapter.

That said, nervous curiosity among candidates would naturally lead to speculation about how their performance is graded. We, therefore, offer the following advice.

Section 1

A combined pass mark between paper 1 and paper 2 is necessary to progress to the next stage of the exam. We understand that the examination board raised the pass mark for section 1 in 2013 to make it more likely that candidates progressing to section 2 will pass. The reason for this is that section 2 is difficult to organize and doubly difficult to organize well. The clinical cases need to be of a uniform high standard that will stretch candidates. There is no point in organizing these complex examinations and allowing candidates to sit them if they have very little chance of passing. There is a world of difference between passing an MCQ paper and examining a patient with a difficult knee condition.

We know from the JCIE that a process of 'standard setting' is performed, where a group of experienced and trained examiners sit the exact same examination, and subsequently set a pass mark for each paper. A question may be excluded if considered too ambiguous or unclear by the examiners following this process so try not to ponder too much over what you have submitted. Remember, a good second paper can compensate for a nervous performance on the first, thus, paving the way for section 2.

It has been suggested that up to 20% of questions are discarded along the journey from being developed by the Examination Board. Questions can still be rejected after being used in the real exam paper following item analysis and trainee feedback. The main point stressed by examiners involved in writing the MCQ paper is that a question will be discarded if it is deemed ambiguous.

Section 2

The scoring in this section is less straightforward, and little information is publicly available. The following is our own interpretation of the marking system (Figure 1.1). It makes a few assumptions, but we believe it to be fairly representative.

Each clinical case and viva question is marked from 4 to 8, equating to the following:

- 8 (exceptional pass)
- 7 (good pass)
- 6 (pass)
- 5 (fail)
- 4 (poor/complete fail)

In more detail:

- 8 – Gold medal standard. Difficult for the average standard candidate to achieve. At ease with higher order thinking.

Flawless knowledge

Excellent understanding/knowledge/management/prioritisation of complex issues. Demonstrates excellent command of the literature. Able to apply the literature to justify management decisions. Instils confidence. Patient rapport very good.

Well-rehearsed keeps talking without prompting but discussion still relevant and pertinent to topic. Not fazed by questions, able to deal with them consummately. Able to intuitively know where the questions are going. Well-trained all round performance.

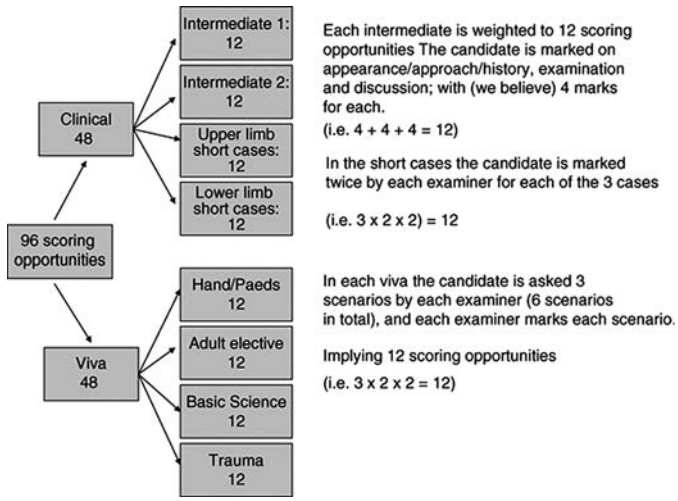

Figure 1.1 Marking system for the FRCS (Tr & Orth) examination

- 7 – Good pass. Very good answer, no hesitation or gaps in knowledge. Able to demonstrate good command of the literature to the examiners. Polished and articulate answers. Quotes from the literature

Able to prioritize. Goes beyond the competency questions. Gives patient confidence quickly. Good awareness of patient's reaction.

Not as thoroughly conversant with the literature as an 8 candidate, some gaps. Not able to fully grasp the viva opportunity presented requiring some prompting as to where the topic is going.

- 6 – Satisfactory pass. Good working knowledge of the subject. Covers the basics well. Copes with competence questions. Important points mentioned. No major errors. Treats all patients appropriately. Observes patient expression

Cannot get to the next level, draws blanks with the more difficult probing viva questions.

- 5 – Some hesitation, not answering the point of the question. Waffling a bit. Surface knowledge and not able to go beyond the basics. Has rote learnt rather than understood the topic. This mark gives a candidate a reasonable chance to recover. Demonstrates a lack of understanding. Confused and disorganized answers. Hesitant and indecisive answers. Lack of an organized structure to the answer. No introduction to patient. Does not listen to patient
- 4 – Unsafe. A miserable failure. Difficult to salvage. Poor knowledge with gaps. Gross basic mistakes. Not knowing a topic expected for this level of senior exam (calcium metabolism, anatomy). Not able to get past the basic viva question asked. Difficult to pull it back

Abrupt, brusque manner with patients. Arrogant and rude. Inappropriate attitude. Rough handling of patients. Poor basic knowledge and judgement. Unpersuadable – Prompts do not work. Did not get beyond default questions. Lacks insight.

'96 opportunities to score'

There are 96 scoring opportunities for each candidate in section 2 – 48 in the clinical and 48 in the viva, and the total mark attainable is 768, with a pass mark of 576. This is implied from the fact that a 6 at each scoring opportunity indicates a pass, and the pass mark is 576 ($6 \times 96 = 576$). Note that there is no deliberation in these marks. If you get 575 you will fail! This has happened to candidates in the past.

The reason for the high number of scoring opportunities is that if there is an issue with a particular examiner or section the effect on the candidates overall score will be diluted by the large number of other examination marks. There are equal marks available for the orals and clinicals.

Orals

There are two examiners marking at each of the four viva stations, although there can be lay observers, examiner assessors or trainee examiners also present. There are at least six questions in each viva; three from each examiner. Each examiner marks each scenario meaning there are 12 scoring opportunities ($6 \times 2 = 12$) at each station. In 2 hours (120 minutes) 8 examiners can independently assess each candidate on a total of 24 topics, with each topic represented by a clinical scenario, and generate 48 test scores, which should provide a valid and reliable measure of a candidate's ability in terms of professionalism, patient care, knowledge and judgement and quality of response.

Clinical

The scoring here is a little less clear; however, the short and intermediate cases are weighted equally, implying 24 scoring opportunities for each section.

The scoring system is open to considerable speculation and interpretation. Whatever way you look at the scoring system there is a concern amongst examiners that some candidates may be getting the wrong advice regarding examination tactics. It appears that candidates at various courses have been instructed to aim for a steady 6 where, in fact, they should be aiming for a 7. In our opinion, candidates should aim high so that even if their performance drops, the candidate should still achieve a safe pass. It is extremely easy for a candidate to drop down to a 5 at one viva question or clinical examination case so you need to score some 7s along the way to counter balance this. A steady 6 all along the way in the exam with a couple of 5s will mean you fail the exam.

The examiners also stress that the oral examination is about the principles of orthopaedic practice and management and not about stalling for time or evading the answer. For example, if a scenario of polytrauma is presented by the examiners of an open comminuted tibial fracture and coexisting pelvic fracture, the first comment should not be that you would send it to a trauma centre. This answer will just irritate the examiners – Far better to go through the principles of how you would actually manage this patient. The second comment should not be an attempt to stall and focus exclusively on ATLS® principles, especially if these have already been covered in an earlier question or the examiners mention the injury is a 'closed isolated fracture'.

Another point to make is that immediately after each intermediate case, shorts or viva, the marking sheet is collected. Hence, subsequent examiners do not know how you've performed previously. So if you think a case has gone badly, put it behind you and move on – You still have everything to play for!

Preparation advice

The aim of the exam is to assess whether you have the knowledge and understanding to practice safely as a Day 1 Consultant Orthopaedic Surgeon in a District General Hospital. This is the standard reference setting criteria to fall back on. However, the syllabus is vast that you can be asked almost anything! The following are some helpful tips in organizing your approach to the exam.

'Plan, prepare, practice, perform'

Plan

- It is important to start revising early. Although everyone has their own individual style of study, the general consensus is that a minimum of 6–12 months intense preparation is needed to talk with confidence to your examiners
- Consider the available exam dates and apply early for the one you need. The examination application process is structured and requires references. You don't want to add to the stress of the event by risking a late application!
- Avoid taking on time-consuming extra-curricular tasks (e.g. research/paper write-ups) in the 6 months prior to the exam in order to give yourself a clear run at it without extra unnecessary pressure
- Controversially, some candidates may be advised to take up a less busy registrar post in the 6 months or so prior to the exam to give themselves more time for study. This can work the opposite way in that a busy post may provide a lot of additional clinical experience that may prove useful in the exam. What probably isn't a good idea is to be travelling long distances to and from home each day in the 6 months before the exam. Even in this situation, previous candidates have still managed to use travelling time effectively by listening to orthopaedic discussion/tutorial type CDs in the car
- Plan your training if possible. Most training programs will be designed to expose you to the general breadth of trauma and orthopaedics; however, if you feel you have a weakness or deficit in a certain area, and have the opportunity to request a specific subspecialty, this is worthwhile considering
- Alternatively, if you are lacking in experience in a particular subspecialty, attend clinics in those areas and enrol on specific courses
- Book your study leave early and avoid the hassle and stress of late rota swaps to facilitate attending courses
- Plan your attack of the syllabus! It is so vast that you could literally spend years reading around it. Make realistic goals and set timetables
- Revision is a very personal issue. Most people have developed their own style of studying but it is important to pace yourself. You do not want to burn out. Make time for your family and maintain a social life (albeit a somewhat less busy one!) – At times you will need the support of your friends and family
- Maintain your momentum. It is important to sit section 2 of the exam as soon as possible after passing section 1. An anonymized questionnaire of 156 orthopaedic surgeons who passed the FRCS (Tr & Orth) exam showed a 90% first-time pass rate for those who took the second part at the earliest opportunity[b].

[b] http://postgraduateorthopaedics.com/books/pg-orthopaedics-second-edition/chapter-1/frcstrorth-risk-factors/. Additional material for exam preparation is contained on the Postgraduate Orthopaedic website.

Prepare

- Form a study group. If others are sitting the exam at the same time then team up (three is an ideal number). Choose like-minded individuals with whom you get on! A group will allow you to compare your progress and share your anxieties. It is also useful to focus your studying and bounce ideas off each other. When it comes to studying for section 2, your reading group can provide you with clinical and viva practice
- Don't worry, however, if you find yourself studying on your own. According to the questionnaire quoted above, candidates studying alone seem to do just as well as those who prepare in groups. (Group work does, however, definitely make the whole process less lonely!)
- The reading material you choose to utilize is down to individual preference. Some choose key textbooks while others prefer to use orthopaedic websites. Choose your 'poison' early and try to avoid swamping yourself with too many sources of information (more on this towards the end of this chapter)
- Source the latest versions of important national guidelines (in topics such as fractured neck of femur patients, open tibial fracture management and osteoporosis, for example) and KNOW these. This will easily convert a pass to a good pass and can act as a structure to help you build an answer if you are stuck
- Although it is not essential to quote specific papers from the literature, it is helpful to know a couple of key papers in each topic, especially in controversial areas
- It is probably fair to say that candidates focus the majority of their time preparing for the oral component (for example with this book). To ensure you pass and pass well, do not neglect the clinical component, which carries the same number of marks as the oral and in which a good performance on day 1 will set you well on track

Practice

- Make it known amongst your colleagues and the consultants at your unit that you are preparing for the exam. Try to avail of any interesting exam cases they know of and utilize any opportunity to be put in an 'exam scenario'
- Each time you see a patient try and deal with him/her as a short or intermediate case. Practise getting straight to the point in your history, as you only get five minutes for this in the intermediate cases, and become slick at doing a thorough examination. Have someone examine you, keeping to the allotted time; be it your consultant, educational supervisor or study group partner. Be confident in eliciting clinical signs without hurting the patient; this is a deadly sin and you will be failed. Always be courteous and respectful to the patient
- PRACTICE, PRACTICE, PRACTICE your clinical skills. On patients. On your study group. On your parents.

On your friends! It is essential to look slick in the clinical section. There is no time to pause to think of what comes next in a hip or knee examination. It needs to flow and look like you have done it a thousand times. Have a reproducible method for examining each system, but be aware that you may need to focus your examination or adapt it in the real thing according to the examiners' instructions

- If possible, arrange mock clinicals and vivas with consultants from each subspecialty. This is possibly one of the most useful things you can do to practice for the real thing. The exam is an expensive way to practice if you fail first time! There is no point in doing this too early, however, when your knowledge is still lacking. Best to leave it until the run up to section 2 when you are practicing 'polishing' your answers. If this is not possible, there are excellent clinical and viva courses for the FRCS (Tr & Orth) exam which candidates have found to be extremely beneficial
- Be confident at interpreting x-rays and scans. This will help improve your confidence in a viva situation
- Practice drawing pictures and diagrams to demonstrate your knowledge. It is not uncommon in the viva to be asked to illustrate certain concepts; for example, stress-strain curves/free body diagrams. A list of diagrams to consider familiarizing yourself with is provided in Table 1.1. This is by no means exhaustive but it has been compiled from suggestions by previous candidates and consultants alike
- The annual UK in Training Examination (UKITE) provides a 'mock' type experience in preparation for section 1. Although the questions are of a somewhat different style, it provides an opportunity to track your learning progress and allows practice with SBA format and exam timing

Perform

- Unlike section 1, where you could probably sit the written papers in your pyjamas, your appearance actually matters in the clinical and viva sections! This part of the exam is somewhat like an interview. You need to present a well-rounded, professional 'package' to your examiners, i.e. smartly dressed and polite with good communication skills – And this is even before you have answered any questions!
- Make the examiners job easier. It is like taking your driving test again. You need to make your assessment of the patient obvious to your examiners, such as checking for insoles in shoes or acknowledging the walking stick propped in the corner of the examination cubicle
- Never hurt the patient! Make it obvious that you are looking at the patient's face for a painful reaction as you examine, and ask them to say if you are causing discomfort
- Listen carefully to the examiner's instructions. For example, in the short cases, if an examiner says, 'I'd like

Table 1.1 Suggested potential diagrams for FRCS (Tr & Orth)

Basic science	• Stress/strain curves
	  . Including ligaments and tendons
	• S–n curve
	• Young's modulus curves for different materials
	• Scratch profiles
	• Viscoelasticity graphs
	  . Creep
	  . Stress relaxation
	  . Hysteresis loop
	• Screw anatomy
	• Collagen structure
	• Micro- and macro-structure
	  . Nerve
	  . Cartilage
	  . Ligament
	  . Tendon
	  . Bone
	  . Skeletal muscle
	• Proteoglycans
	• Cutting cone
	• Osteoclast
	• Menisci
	• Intervertebral disc
	• Action potential
	• Reflex arc
	• Gait cycle
	• Clotting cascade
	• Free body diagrams
	  . Hip ± stick
	  . Knee up/down stairs
	  . Elbow
	  . Ankle
	  . Spine
	• Prosthesis components
	• Statistics
	  . Sensitivity/specificity table
	  . Table of levels of evidence
	  . Survival curve

Anatomy	• Brachial plexus
	• Cross-sections
	  . Upper limb, inc. carpal tunnel and extensor compartments
	  . Lower limb, inc. compartments
	  . Spinal cord
	• Hands
	  . Flexor/extensor tendon zones
	  . Finger extensor apparatus
	  . Finger pulleys
	  . Incision for carpal tunnel decompression and associated landmarks
	• Blood supply
	  . Femoral head
	  . Talus
	  . Scaphoid
	  . Humeral head

Table 1.1 (*cont.*)

	• Attachments to the coracoid • Spaces posterior to shoulder and what passes through them • Relations to piriformis • Hip trabecular patterns • Femoral triangle
Paediatrics	• Physis and zones • Salter–Harris classification • Selenius graph
Genetics	• Pennett squares for inheritance patterns
Misc.	• Z-plasty • Tendon repair methods, e.g. Kessler • Traction types, e.g. Hamilton–Russell/ Thomas splint • External fixation • Tension-band principle

YOU to examine this patient's right great toe', don't start by taking a history and looking at the hands for signs of systemic disease. This will only waste vital time and irritate your examiner. If the examiner guides you by suggesting that you ignore your systemic assessment and concentrate on the big toe, listen to the advice!

- If the examiner asks 'Are you sure?' take the hint you may have answered wrongly – Why else would they say this?
- The short cases are exactly that – SHORT! The 5 minutes seem to last a blink of an eye, so don't delay in eliciting those important clinical signs and relay them to the examiner
- If permitted, narrate as you perform your examination. Most examiners don't seem to mind this technique and it makes it clear what you are trying to demonstrate. Don't waffle, however, try to keep what you say succinct
- In the intermediate cases you will have 5 minutes to take a history, 5 minutes to perform a focused examination and 5 minutes to discuss the case. Get off to a good start when presenting your findings for both the history and examination. For example: 'Mr Jones is a 45-year-old, right-hand dominant electrician who presents with a 6-month history of pain and weakness in his right shoulder which is now beginning to affect his work'. Already you will have delivered a succinct summary of vital points of information. It does not look slick if you have forgotten the patient's name or occupation when you come to relay the history you have just taken. Again, a polished delivery of your findings comes with practice
- Make sure that you answer the question that is asked of you
- Finally, it has been suggested that you should try to imagine the vivas and oral discussions as a conversation between consultant colleagues discussing a case. This can be extremely difficult under pressure, but remember that the examiners for the orals are no longer specialized within that subject

The event itself

Section 1

- Follow the instructions given to you by your designated examination centre
- Be sure you know how to get there on the day and allow time for traffic delays or unexpected problems
- Get a good night sleep the night before and try to get lunch between papers – The day is long and you will need all of your energy

Section 2

- Book yourself into a decent, comfortable hotel and ask for a quiet room. Day 1 of the exam is currently usually held on a Sunday
- Remember that you've already forked out near-enough £2K, so now isn't the time to start being cheap – You're worth it! Beware, however, that the nicest hotel is usually where the examiners stay. Get an early night and go easy on the coffee and alcohol, as you want to be at your best
- On the day give yourself plenty of time to get to the venue. Consider a 'trial run' the day before so that you know where you are going. The last thing you need is added stress if you get lost or stuck in traffic
- Dress conservatively and avoid outspoken ties or ostentatious suits. You want to look smart. Be aware that you may need to comply with local infection-control policies such as 'bare below the elbows'
- Watch what you eat before the exam – You don't want to stink of garlic or cigarettes – Nor do you want to reek of too much aftershave or perfume
- Once you get there you will soon realize that the exam is run with military precision. This in itself is confidence inspiring, as there can be a lot of candidates sitting the exam at any one time. Just listen to the instructions given and concentrate on delivering what you have practiced when it is your turn to show off your knowledge!
- Come prepared with any props you might need for examination purposes. You may not have the opportunity to use them, but if you have time, a quick demonstration of transillumination of a soft-tissue lump with a pocket torch, for example, can look pretty slick and add useful information to your examination. Other suggested props include a tape measure for leg length discrepancy, and coin and key for testing hand function. Again, however, be guided by the examiners and use your discretion. Don't waste time utilizing props just because you have them in your pocket, and it certainly doesn't look good if you cannot find your prop!

The short cases

Today's exam format means that you examine 6 short cases only (3 upper limb and 3 lower limb) and spend 5 minutes with each case. You may spot the diagnosis immediately and

can tell the examiners what it is but you will still spend 5 minutes examining and discussing the case regardless.

'Common conditions are common'. There are usually very few surprises in the clinical cases. Spend time talking to previous candidates about their experiences and obtain advice from senior consultants where possible.

The intermediate cases

With 15 minutes allocated for each of the two cases, these tend to feel a little less hectic. Take a slick but focused history and examination and deliver the relevant points succinctly to the examiner.

Again, there are unlikely to be any surprises in the intermediate cases. You may, however, get a patient with more than one orthopaedic complaint. If this occurs simply listen to the examiners question carefully and cater your approach as necessary. If you are completely stumped by a case from the start, simply go back to the beginning and rely on your methodological history and focussed examination according to the patient's complaint. You may not always be expected to get an accurate diagnosis in an uncommon syndrome but you will be expected to discuss the orthopaedic issues pertaining to the case in question.

Orals

Many candidates find the orals the most intimidating aspects of the FRCS exam. Again, lots of practice of viva technique with colleagues and consultants prior to the event is the key to passing this part.

The current oral exam format is comprised of 3 standardized questions per examiner (6 questions per viva, 5 minutes each), where you are marked on each question by both examiners. This happens for each of the four viva stations.

Since November 2014 examiners have had the viva questions pre-prepared for them by the Examination Board. This means that all the examiners will be asking the same questions at each viva station. In the afternoon the questions will get changed presumably to prevent candidates discussing the questions amongst themselves at lunch. This change is made to further improve exam consistency and ensure it is as fair as possible to all candidates.

In addition, the same clinical photographs and radiographs for a particular question are shown to each candidate. This again improves the standardization of the examination and indirectly ensures only good quality props are used. The examiners are very quick to point out any unclear or confusing clinical pictures or radiographs.

Questions are not deliberately set to catch you out, but some are designed to extract 'higher order thinking' from the candidates. This does not mean that you need to know the intricacies of every operation for reconstructing a dislocated knee following a motorcycle accident, for example. Whilst awareness of operative management options in such an injury is clearly desirable, a safe, methodical approach to a suspected polytrauma patient, with knowledge of the emergency management of a potentially limb-threatening associated vascular injury is what the examiners will hope to elicit from a candidate who has what it takes to be a safe, independently practicing consultant.

It is best to enter the oral examination with the intention of answering the questions as clearly as possible, in a structured, sensible manner, demonstrating a safe and methodical approach to the problems presented.

For the oral component, from experience of the current format, the following assumptions can be made:

- Questions chosen must contain enough material for the candidate and examiners to discuss for 5 minutes without running out of steam
- If you can't respond to a question because you don't know the answer and only 30 seconds have elapsed out of the 5 minutes, the examiners can ask you a reserve question. In practice this situation is very uncommon and the examiners may still stick with the first question, only asking you very basic perhaps even unrelated questions to get some sort of discussion going. With a reserve question you will generally only be scored a 4, at the very best with a superb answer you will only a score 5. It becomes difficult to make up these lost marks in the exam. Again to reiterate, this is a very uncommon situation and perhaps more theoretical than practical as reserve questions are very rarely asked with the examiners preferring to stick with the original question
- If you are doing really well you may reach the reserve fourth question and you will be picking up bonus marks. This is not tending to happen now as there is usually enough to talk about with each question for the full 5 minutes even with a score 8 candidate and, if needed, the examiners will move onto the next question slightly earlier than planned
- Examiners have a list of points that they need to cover with each question and a model answer for reference. Marks are scored when candidates answer correctly the points that are asked
- There may be a series of candidate prompts to which the examiners can refer on the model answer form. The examiners use them if the candidate is straying widely off the mark with their answer to bring the candidate back to the main thrust of the question
- Some examiners believe that it is more difficult to examine candidates using this new system than the old ad-hoc method. Spontaneity is lost and examiners may refer too much to the model answer for guidance rather than let the discussion take its course
- Most candidates prefer the new system as it is perceived to be more impartial and fairer than the older method
- Candidates will be compared to their peers. Ten or so candidates being asked the same set of questions by the examiners will invariably mean they will be ranked in order of performance

FRCS (Tr & Orth) dry run

The exam is an expensive way to practise, but there are other exams that can be used to practise for the FRCS (Tr & Orth), namely the SICOT diploma and the EBOT. Several candidates use these exams as preparation for the FRCS (Tr & Orth) exam and pass them. The advantage is more letters after your name as well as preparation for the FRCS (Tr & Orth) exam.

EBOT examination

The EBOT examination has developed into a prestigious orthopaedic qualification in recent years. Exam applicants have increased significantly since 2011. The exam consists of two sections. Section 1 is a written MCQ paper completed online of 100 MCQs in the format of a single correct answer (SCA). Section 2 is the viva component composed of five viva (oral) stations. The sections examined are:

- Adult orthopaedic and trauma surgery – Upper limb
- Adult orthopaedic and trauma surgery – Lower limb
- Adult orthopaedic and trauma surgery – Spine
- Children's orthopaedic and trauma surgery
- Basic sciences related to orthopaedics, including biomechanics, statistics, audit methodology and outcome-based medicine

Applicants need to be successful in section 1 in order to gain eligibility to proceed to section 2. Candidates regard the examination as professionally very well-run and organized. The viva stations roughly alternate between elective and trauma questions, but there is less rigidity in the number of viva questions asked, varying between 3 and 7–8 depending on the quality of a candidate's response. The spinal section can be quite difficult and candidates really should have spent some time in a spinal unit to do justice to this viva.

In some specialties the European examination is treated as equivalent to the UK specialty exam. In trauma and orthopaedics this is not the case, the EBOT exam has no clinical component. The whole question of a European-wide examination in orthopaedics has recently been explored by David Limb BOA secretary in an article for the *Journal of Orthopaedic Trauma*[1]. Recently the EBOT committee has been exploring the possibility of providing parts of the exam in different languages other than English. In addition they are keen to assess skills as well as knowledge in the final exam. Essentially they are eager to include an additional clinical component to the exam. High stakes clinical exams are difficult to organize. Practicalities include finding a suitable venue, sufficient number of patients with good clinical signs and examiners thoroughly trained in clinical assessment etc.

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Chapter 2

What to read

Jonathan R. A. Phillips and Benjamin W. T. Gooding

Gone are the days where a single 'classic', multi-volume textbook read cover to cover will provide all of the information you need. Modern communication techniques and in particular the Internet have transformed the way to revise for postgraduate examinations.

The following are a number of resources that you may find useful.

Textbooks and websites

Our predecessors sought to fill the shelves of their offices with grand orthopaedic textbooks with leather bound covers. However, big textbooks are too heavy to take anywhere! Modern textbooks, especially those from the large publishing groups now allow access online, enabling the purchaser to gain access whenever or wherever they are. Many also offer multimedia such as videos and extra illustrations to complement their text. Many excellent textbooks have been written aiming specifically for the FRCS (Tr & Orth) exam.

There is another problem with textbooks; as soon as the textbook is published, its content is out of date. Certain websites aim to avoid this problem through real-time updates. Many of these websites have a huge amount of content, with much of it being delivered in an easy to read, summarized format. Table 2.1 lists some of what we found the most useful revision websites.

However, such websites have significant limitations. There is little or no peer-review, the content is frequently unregulated and plagiarism may be a problem. Authorship is generally not reported, and one must be careful to not put too much faith into everything that is read. Many websites are commercial enterprises or sponsored by implant companies (or law firms!), and many other websites are self-promotion websites by enterprising orthopaedic surgeons seeking fame and fortune. They can, however, form a fantastic quick reference tool.

An exciting new development is the Wikipaedics website currently being developed by the BOA. This is a project to update the old orthoteers website material that had been used in the past by trainees preparing for the exam. The website material was passed on to the BOA around 2012 for further development. The material is being radically updated as an on line learning platform and media interaction. The platform's content will be in line with the Applied Clinical Knowledge Syllabus of the Specialist Training T+O Curriculum, in order to provide trainees with a sound and logical instructional aid.

Table 2.1 Useful websites

MCQs
Orthobullets: www.orthobullets.com
Reference
Wheeless: www.wheeless.com
Instant anatomy: www.instantanatomy.net
Exam revision
BJJ exam corner: www.boneandjoint.org.uk
Trauma
AO surgery reference: www.aofoundation.org
Foot and ankle
Hyperbook: www.blackburnfeet.org.uk/hyperbook
Video calls
Skype: www.skype.com
Google Hang Out: www.google.com
Meeting planner
Doodle: www.doodle.com
Shared online storage
Dropbox: www.dropbox.com
iCloud (Apple): www.icloud.com
OneDrive (Microsoft): www.onedrive.com

Internet search engines are also invaluable tools that can be used to answer specific questions. Once again, care must be taken to ensure that the answer is factually correct. Bookmark useful websites once found to build a library of online resources.

There are a number of review articles published in the major orthopaedic journals, and there is a review journal aimed specifically at the UK FRCS (Tr & Orth) exams[a].

[a] *Orthopaedics and Trauma* journal. Available from: www.orthopaedicsandtraumajournal.co.uk.

The content in these review articles is reliable, although unless you have access to the journal through a university or library, it may be expensive.

Multimedia

Textbooks, whether paper or online, form only part of the arsenal of the modern student. The Internet has developed a new format called 'Webinars'. These web-based seminars are normally put on by experts in their field, and enable interaction and questions both during and after the session. These are useful ways of gaining expert opinion on specific topics. Most of these are hosted in the USA, which unfortunately means they take place when we in the UK are at work or asleep!

Most of us honed our examination skills by learning from experienced consultants. Now you can learn even the most obscure clinical tests off video-streaming sites such as YouTube[b] or VuMedi[c]. Students must be warned though that there is no point being able to perform the test for Piriformis syndrome when they have not mastered the Thomas' test.

Audiobooks and podcasts are a different format of acquiring a breadth of knowledge. Lectures from both the American Academy of Orthopaedic Surgeons and the Miller textbook can be purchased online[d]. Most of us have to commute to work so why not combine commuting with revising? Many orthopaedic podcasts can also be downloaded for free or for small fees from iTunes. Once again, investigate the publishers of any information for potential bias.

Apps

A variety of apps can be bought or downloaded for free for many types of smartphone. One of the better ones available is that provided by the AO Foundation, providing useful information on trauma procedures and surgical approaches[e]. Unfortunately many of these apps are online-only and, therefore, do not work in big hospitals with thick walls where phone reception is poor (such as in theatre!).

Interactive case discussions and discussion forums

Online question banks are invaluable when studying for the written part of the exam. Doing well at MCQs comes from lots and lots of practice. One of the largest online 'free' resources is Orthobullets[f]. We found Orthobullets an extremely easy website to use, with detailed explanations presented with each of the answers. The ability to monitor your progress and compete with your peers helps to maintain motivation. Orthobullets also offers a study programme (for a fee); a feature of which sends out daily emails to cover a topic each day with relevant questions. The website is American in origin, however, and the questions may seem less relevant to British exams. Despite this, practice is practice so they are still recommended (and free!).

Revision groups and communication

Many people find gathering into small groups of like-minded individuals a great way of studying, especially for viva practice.

One of the difficulties with organizing revision sessions is trying to bring busy people together. Other commitments and on-calls frequently get in the way. Doodle is one of a number of free websites that allow people to organize dates for meetings[g].

Another difficulty was that such sessions were often incompatible with family life. We're sure that we weren't the only people revising for exams with newborn babies; in fact one of the people in my revision group had just had twins. For us, it was impractical to meet up in early evenings. We started to have revision sessions on our iPads over Skype or Facetime[h,i]. Having the face-to-face contact during revision sessions, while both still being able to access our own computers for notes was invaluable. When there are more than two of you, certain websites also offer video conference tools (often at a charge).

The ability to share content and revision notes online is now possible. Sites such as Dropbox, iCloud and Onedrive allow users to upload notes that can be shared between members of the revision group[j,k,l]. Having a shared 'useful papers' folder can be extremely helpful in keeping up to date.

Revising at work

Laptops, tablets and phones have now become the favoured way to work and gain access to the Internet. Many with young families find the demands of family life get in the way of study, meaning that working on the move whenever a spare moment arises becomes necessary. We found mobile phones too small to do any effective work on, but mobile broadband via 'Dongles', mean that laptops and tablets can be used on the move. Be careful with your usage though as large volume use can be expensive. Most phones can now be used as a "hotspot" which is another great way to get on line.

Important papers

It is helpful to know a couple of key papers in each topic, especially in controversial areas. It is important to know national guidelines (such as on hip fractures, open tibial fracture management and osteoporosis). This will easily convert a pass into a good pass.

[b] YouTube . Available from: www.youtube.co.uk.
[c] VuMedi. Available from: www.vumedi.com.
[d] AAOS. Available from: www.aaos.org.
[e] AO Foundation. Available from: www.aofoundation.org.
[f] Orthobullets. Available from: www.orthobullets.com.
[g] Doodle. Available from: www.doodle.com.
[h] Skype. Available from: www.skype.com.
[i] Facetime. Available from: www.apple.com/uk/ios/facetime.
[j] Dropbox. Available from: www.dropbox.com.
[k] iCloud. Available from: www.apple.com/uk/icloud.
[l] OneDrive. Available from: www.onedrive.live.com.

Recommended reading

The choice of reading material is very much a matter of personal preference. There is no official reading list and there is currently a plethora of orthopaedic exam textbooks on the market, of varying quality. Unfortunately, because of the diverse nature of the exam, no perfect book exists for the FRCS (Tr & Orth), and it will be necessary to glean information from a variety of different sources.

Orthopaedic textbooks are expensive, and it is worth taking time before choosing. Get advice from trainees who have recently sat the exam and, if possible, borrow books to look through and decide whether they suit your style of learning. Failing that, you can browse in a good bookshop or using the 'look inside' facility available on some online bookshops.

Probably your most important purchases will be a good general textbook and a surgical atlas. Make these choices early and get to know them. Most people need around 12 months of intensive revision before sitting the exam, so make these two major purchases 2–3 years before you plan to sit it. That way you can become familiar with your books in plenty of time, and still have time to change them if they don't suit you. You can then supplement them as required with smaller, more specialized books as time goes on. Of course, if you buy all your textbooks right at the beginning of your training, they may begin to become dated by the time you actually sit the exam.

There is a definite balance to be struck between using too many sources of information superficially and concentrating on too few. As a general rule, change books or add to them only if there are significant advantages to be gained. If the style or content of a book does not agree with you (it is sometimes difficult to tell until you start actually to use it), discard it quickly and move on to something more suitable. In the early stages of training, it is worth reading up on the specialties to which you are attached – What you are reading will make much more sense, and will be more likely to 'stick' if it correlates with what you are seeing during the day. As you approach the exam, however, most people find it helpful to work out a study schedule to avoid running out of time and missing important topics.

One major difference since the second edition of this book was published is the almost exponential increase in the amount of FRCS (Tr & Orth) material now available. Some of this material is extremely good whilst other bits are of dubious quality. Self-publishing a book used to be annoyingly difficult time-consuming process, but in recent years it has become much more streamlined. Some candidates are now self-publishing their revision notes. This muddies the water as some of this material can be of a high standard whilst other books are disappointing and poor quality.

Amazon reviews

Amazon orthopaedic book reviews can be helpful in guiding candidates in their choice of book. However, be cautious, as they can also be misleading. Certain books suit particular learning styles more than others, so this may bias the personal review of the book. Amazon also lists whether the reviewer has made a verified purchase of the book; these reviews are likely to be more reliable than from non-verified sources.

Included below is a list of suggestions for the various categories. Full details are given at the end of the chapter.

General textbooks

Miller's Review of Orthopaedics[1]

This is the standard text used by most trainees. It is very compact, but extremely terse, and not necessarily easy to read except in small doses. Because of its size, it does assume a fair bit of prior knowledge. Some topics are covered in more depth than others, but it is reasonably comprehensive, with chapters covering basic sciences, anatomy and statistics as well as the more 'clinical' topics. Most people find it more useful later in their reading, when they already have a bit of knowledge to build on.

Oxford Textbook of Trauma and Orthopaedics[2]

The second edition pulled off a masterstroke by reducing the three-volume set to one. However, the content seems to have been summarized and the text detail disappoints in places. We never felt there was quite enough detail in certain sections and generally end up looking elsewhere afterwards for the information. It's a large book and also expensive to buy. Reviews have been mixed.

AAOS Comprehensive Orthopaedic Review[3]

This book is at the other end of the spectrum. It comes in three volumes and, although more comprehensive, is much more expensive and less compact. It's probably worth looking at and considering as an addition to Miller's if you struggle with the note-like form of the latter. The American Academy of Orthopaedic Surgeons publishes a large number of textbooks and it is a well-oiled machine. They are generally of a high standard although can be quite expensive and have a slant towards the American audience.

At the beginning of training, Apley's *System of Orthopaedics and Fractures*[4] is a good introduction, but you will need something much more detailed for the exam. It has a great series of pictures that can be used for viva practice prop picture revision.

Current Orthopaedic Practice – A Concise Guide for Postgraduate Exams[5]

This book has very favourable trainee feedback and comes highly recommended. The author has managed to include many current literature references, which candidates find useful in their exam preparation.

Surgical atlases

Hoppenfeld's Surgical Exposures in Orthopaedics[6]

This has become the standard atlas used for the FRCS (Tr & Orth) exam, and it is good. Having said that, Tubiana's *Atlas of Surgical Exposures of the Upper and Lower Extremities*[7] was a personal favourite owing to the clarity of the illustrations, text and layout. *Orthopaedics Surgical Approaches* by Miller et al

has a slightly different style to Hoppenfield that some candidates may prefer. It is a more exam revision-friendly book with good illustrations and a relaxed style of text.

Briggs' Operative Orthopaedics[8]

This is a fairly basic textbook that some people have found useful in tying things together. If you need a little bit of anatomy revision, it's worth looking at a copy of Whitaker and Borley's *Instant Anatomy*[9]. It has very succinct summaries of the courses and branches of nerves and blood vessels, and other such reminders.

Clinical examination

The second edition of Harris and Ali's *Examination Techniques in Orthopaedics*[10] is a significant improvement on the first edition. Each chapter has been extensively revised. It has had excellent candidate feedback and is now firmly established as the clinical examination book of choice for candidates sitting the FRCS (Tr & Orth) exam. It has had excellent candidate reviews on Amazon.

Reider's *The Orthopaedic Physical Examination*[11] is useful as a reference if you've got it in your library, but it's probably a bit too expensive to recommend buying. It also is a bit overlong and exam unfocused in places.

Basic sciences

Ramachandran's *Basic Orthopaedic Sciences*[12] has become the standard book, and is well worth getting. It is reasonably clear and detailed, particularly if you supplement it with the basic sciences chapters from a general book such as Miller's. We eagerly await the second edition that promises to be fully updated and revised to cover the latest breadth of topics in orthopaedic basic science

Einhorn et al.'s *Orthopaedic Basic Science*[13] is a more detailed text. If you have access to a copy, it may be useful as a reference source where you need more explanation, but it probably doesn't need to be read cover to cover.

The basic science section of *Oncology and Basic Science. Orthopaedic Essentials Series*[14] is excellent. It is easily readable and manages to clarify complicated information in a way that simplifies revision.

AAOS Orthopaedic Basic Science, 4th edition provides an excellent overview of basic science with some great diagrams. However, it is quite heavily priced and geared towards the American market thereby missing somewhat the FRCS (Tr & Orth) exam focus.

Paediatrics

Staheli's Practice of Pediatric Orthopaedics[15]

This book is fairly easy to read and comprehensive. It has had good reviews and the illustrations are excellent.

Pediatric Orthopaedic Secrets[16]

We are not great fans of the Secrets series as some of the material is not well-matched to the FRCS (Tr & Orth) syllabus. That said, we have come across a number of candidates who swear by the Secrets series and found this book good for viva practice It has had good reviews.

Joseph et al.'s Paediatric Orthopaedics[17]

This book is disappointing as it promises a lot but doesn't quite deliver the goods. That's not to say it isn't a good book, it's just the hype that doesn't quite match the contents. The book is targeted at higher surgical trainees and younger consultants. It can be difficult to extract a viva answer out of some of the material. It is written by Pediatric Orthopedic surgeons from four different continents, which gives the book an international flavour. However, in the very focused world of FRCS (Tr & Orth) exams this isn't that relevant or important.

Hand Secrets

Hand Secrets[17] is an option, although again the format of this series appeals more to some people than to others. An alternative is to use relevant chapters from a reference book such as *Green's Operative Hand Surgery*,[19] but you will need to be selective.

Trauma

Egol et al.'s *Handbook of Fractures*[20] is recommended as a reasonably concise and up-to-date text. Most trainees find that trauma is one of their stronger areas, and many people simply supplement their experience by looking up specific topics in a reference text such as *Rockwood and Green's Fractures in Adults* and *Rockwood and Wilkins' Fractures in Children*[21,22] or Browner's *Skeletal Trauma*[23]. Be careful not to get lost in these massive tomes, however!

Orthopaedic Trauma: The Stanmore and Royal London Guide[24]

This book was published in November 2015 to generally very positive reviews. Although not as detailed as Egol et al.'s *Handbook of Fractures*[20], it has more of an FRCS (Tr & Orth) exam feel to it. It can be easily read in a week and provides a solid framework that candidates can use as a basis for trauma revision. It is a slightly awkward book in that although it is not completely comprehensive it is also not a weak flimsy throw-away excuse of copied material from elsewhere. The material is original and exam focused and is a welcomed addition to the FRCS (Tr & Orth) armament. It has had excellent candidate reviews on Amazon.

Trauma for the FRCS (Tr & Orth) Examination[25]

This book was published in December 2015 to cover the trauma viva component of the exam. It is extremely well-written and focused on trauma viva topics that regularly appear in the exam. This book is published by the Oxford Higher Speciality group and is a significant upgrade in content detail on the general viva equivalent book that was published by Oxford University Press in 2012.

Statistics

Many basic sciences or general orthopaedic books (including Ramachandran[24] and Miller[14], respectively) have useful chapters on statistics. We also found selected chapters from Greenhalgh's *How to Read a Paper*[26] useful.

MCQ books

The *AAOS Comprehensive Review*[16] comes with a useful MCQ practice book.

Orthopaedic Basic Science for the Postgraduate Examination: Practice MCQs and EMQs[27]

Basic science can be difficult with the main textbooks as they can be either too notebook-like without sufficient explanation or too long and drawn-out. This is an excellent book for revision purposes as the questions are representative of the level and scope required for part 1 of the exam. However, you will still need to use the larger textbooks, as you also need to understand the fundamental principles of basic science and not just rote learn facts. This book has had excellent feedback.

Succeeding in the FRCS T&O Part 1 Exam[28]

This book has had mixed candidate reviews. Explanations can be confusing, and often contradict the answers given. It's perhaps a book to borrow rather than buy for an evening to look through as light reading if you are bored, but no more than this and nothing to base any significant preparation for the exam on.

Practice Questions in Trauma and Orthopaedics for the FRCS[29]

This is for MCRS preparation not the FRCS (Tr & Orth) part 1 exam; hence, the consistently poor candidate reviews. It still sells but do not expect to be challenged as it is way off the exam standard

First Aid for the Orthopaedic Boards, Second Edition (First Aid Specialty Boards)[30]

This is an American book that has been primarily written for the in-service exam (Orthopaedics In-Training Exam (OITE)). It has had some good reviews from trainees, more as a last-minute exam crammer before the part 1 exam than as anything else. It is easy to read and may help you score a few extra points; however, it is expensive for what it is.

Review Questions in Orthopaedics[31]

This book has been written for orthopaedic residents preparing for the in-training examinations of the American Board of Orthopaedic Surgery. Despite the American bias, it is exceptionally highly recommended by the vast majority of candidates sitting the FRCS (Tr & Orth) exam. It is the quality of the explanations of the answers that seems to set the book apart. It is fairly dated now (2001) and can be expensive to buy, but this does not seem to put candidates off.

1000 EMQs in Trauma and Orthopaedic Surgery[32]

This book does not reliably recreate the questions found in the real exam and has been found to be of limited use by those who have used it. Some trainees have found the questions confusing and over complicated.

There is an increasing number of websites with banks of questions that can be useful practice. See, for example, Orthobullets[6] or some of the websites run by large implant companies for which your local rep will give you a password.

Viva books

FRCS (Tr & Orth): MCQs and Clinical Cases[33]

The content has been selected from the examination corner section of the JBJS British volume. There are a few MCQs, but it's really the viva section that is quite excellent – very thorough and comprehensive. A couple of minute criticisms are that some answers are just too detailed for the real exam, perhaps only achievable if you are a score 8 candidate, and a small number of mid viva questions go off the mark and lose their thread, as you are unlikely to be asked these questions in the real test unless perhaps you are a gold medal candidate.

FRCS Trauma and Orthopaedics Viva (Oxford Specialty Training Higher Revision)[34]

This is generally a very good viva book. A few of the questions would have benefited from a more thorough work out but overall it is very useful for trainees in their preparation.

Reference books

Campbell's Operative Orthopaedics[35]

This is a useful reference source when you can't find the answer elsewhere!

European Surgical Orthopaedics and Traumatology: The EFORT Textbook[36]

This book is seven volumes and is expensive. It is aimed at higher orthopaedic trainees preparing for exams within and beyond Europe. It doesn't really have an exam feel about it and, despite the publicity, is definitely a deflating experience.

Summary

There are now many tools to aid in the preparation for the orthopaedic FRCS exams. You have to find out what is right for you. However, it is extremely important that any non-peer-reviewed information is viewed with a heavy amount of cynicism, as it may potentially be incorrect or subject to bias.

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Chapter 3

MCQ and EMI paper guidance

Mark Dunbar, Andrew P. Sprowson and David Limb

Section 1 is the theory part of the Intercollegiate Examination in Trauma and Orthopaedics and consists of two papers. It is no longer a 'written' examination and instead is a computer-based assessment taken in a local Pearson VUE Test Centre. One major change is that there is no longer a published paper to read and the questions applicable to that section have been replaced by multiple choice questions (MCQs) and extended matching items (EMIs) on statistics and research methodology.

Section 1 examinations are delivered at Pearson VUE Test Centres throughout the UK and Ireland to avoid the need for candidates to travel and incur hotel costs. Candidates will be able to choose the one that is most convenient for themselves. Be aware that many different types of tests may be going on at the same time as your exam (e.g. driving theory, USMLE) and so be prepared to focus so as not to be distracted by the movements of others. Some candidates have chosen to travel further to a quieter testing centre or to take the test at the same place as their colleagues to minimize the likelihood of disturbance.

You will be required to bring photograph identification and the exam conditions are strict. Video surveillance of candidates is common and no mobile devices will be allowed in the examination room. It is also wise to bring along a packed lunch to keep yourself refreshed in between papers, as you won't be able to buy food from most test centres.

As this exam is now computer-based questions involving multimedia (radiographs, slides, pictures) can be expected. You will also not be able to read ahead, but you will have the facility to flag difficult or ambiguous questions for review at the end.

Overview

Paper 1

- Single best answer (SBA) paper (2 hours)
- 110 MCQs (SBA format; one from five)

Paper 2

- EMI paper (2 hours 30 mins)
- 135 MCQs (EMI format)

There is **NO** negative marking; therefore, all questions should be attempted. Sample questions can be viewed at www.jcie.org.uk, although candidate feedback suggests these are neither particularly helpful nor representative of the real test.

A candidate's final mark is determined by the mean combined (SBA/EMI) marks achieved in paper 1 and paper 2. Experienced examiners perform a formal process of 'setting the standard' and this sets the pass mark for each paper.

Eligibility to proceed to the clinical component of the examination (section 2) will be the mean of the two marks set by the standard setting process. The details of criterion referencing/standard setting is complicated and not made public. There needs to be a spread of difficulty to the questions to differentiate between candidates. An easier paper will require a higher mark to pass. A question can be graded on difficulty by what proportion of candidates just passing the exam would be expected to achieve the correct answer. During the last few examinations, the pass mark has been between 65% and 68%.

The SBAs/EMIs are subject to quality assurance procedures through both examiners comments and candidates feedback. Difficulty level, content coverage, discrimination index and internal consistency are analysed.

The Joint Committee on Intercollegiate Examinations (JCIE) site contains a link to advice on format and structure of test questions[1]. It is useful to have some idea of how MCQs are constructed, what they set out to test, avoiding ambiguity with stems, use of distracters placed in the stem to change the entire meaning of the question, etc, but the book is very detailed and complicated and is perhaps more relevant for the examiners constructing the questions than for candidates.

The part 1 examination is designed to test knowledge across the whole cirruculum and does so as far as possible by using questions that require higher order thinking. Rather than asking for a fact, it looks for the application of knowledge to solve problems usually clinical scenarios. There has been a progressive rewriting of the question bank to reflect this change in emphasis.

Paper 1

The first paper is 2 hours long and comprises MCQs (SBA, 1 from 5). The paper consists of 110 single-response questions.

Since the middle of 2014 the 12 questions on the published paper comprehension have been replaced by SBI/EMI questions on statistics and research methodology. We are unsure why this has occurred. This part of the paper was always unpredictable and it may have been too difficult to consistently standardize this section from one exam sitting to the next. The examine committee's time was probably better spent on developing and improving the SBA/EMI question bank.

This may have unsettled a few candidates who had obtained higher research degrees. In theory these candidates would be at a slight advantage in this section. However, specialist knowledge at the level of a higher degree is not expected. Instead candidates should be able to demonstrate they have sufficient knowledge to critically appraise evidence and to decide whether or not to allow it to influence their future practice as consultant leaders.

The range of potential questions is wide, but it would be sensible for candidates to expect at least some of the questions to cover:

Sensitivity/specificity
Screening tests
Contingency tables
P-values and confidence intervals
Data presentation methods
Central tendency and measures of dispersion
Sample-size calculation and power analysis
Types of data and appropriate tests for them
Correlation and regression
Outcome measures
Validity and reliability
Levels of evidence
Survival analysis
Sources of bias
Impact factors

Most of these topics are adequately addressed in the commonly used basic science revision texts, but to date there are no statistics books specifically targeted towards the FRCS Orth exam.

The remaining questions are from various aspects of orthopaedics and trauma. There will be questions based on clinical scenarios, basic sciences, anatomy and surgical approaches. A number of questions will be trauma-related, especially spine and pelvic trauma. Anatomy accounts for a large number of questions.

It is equally important to have a good knowledge of medico-legal and medical ethics aspects. In both parts of the FRCS (Tr & Orth) exam candidates have been asked to discuss topics such as confidentiality, consent, GMC good medical practice, Jehovah's witnesses (blood transfusion) and child protection.

Single best answer questions (SBA 1 from 5)

The SBA question consists of an introductory theme, a question stem followed by five possible responses (A–E), of which one is the most likely suited answer to the question. SBA questions are exactly what the name suggests. A question will be set and the candidate has to choose the best from five possible answers. It is important to note that this is not a 'Single Correct Answer' question but a 'Single Best Answer'. In fact all five possible answers could be 'correct', but candidates are asked which is the 'Best' answer given the information presented in the stem. As questions are designed to test higher order thinking, this may mean that not all of the information needed is in the stem – Some of it may need to be judged from your knowledge of the available evidence. Questions about which some candidates complain 'There was more than one correct answer', the question was ambiguous, etc, can often be the best performing questions on the paper.

A few examples are given below.

1. **A 30-year-old woman presents to you with a comminuted fracture of radial head. What is the best management option for this lady?**
   A. Conservative method
   B. Open reduction and internal fixation
   C. Radial head replacement
   D. Closed reduction and percutaneous K-wiring
   E. Early mobilization
2. **A 73-year-old woman is seen in the clinic with a pathological fracture of the first lumbar vertebra. She has previously been diagnosed with metastatic breast disease and has been given a life expectancy of 1 month. What is your treatment plan?**
   A. Pain relief and supportive care
   B. Radiotherapy
   C. Chemotherapy
   D. Vertebroplasty
   E. Posterior instrumentation
3. **A 23-year-old sustained a penetrating injury to the sole of the foot while playing a game of tennis. What are the commonest infecting organisms?**
   A. *Staphylococcus aureus*
   B. *Pseudomonas* spp.
   C. *Escherichia coli*
   D. *Proteus* spp.
   E. *Staphylococcus epidermidis*
4. **What is the root value of adductor longus?**
   A. L1
   B. L1 and L2
   C. L1, L2 and L3
   D. L4 and L5
   E. L4, L5 and S1
5. **A 43-year-old man has back pain associated with EHL weakness. Which intervertebral disc is likely to be prolapsed?**
   A. L2/L3
   B. L3/L4
   C. L4/L5
   D. L5/S1
   E. L1/L2

6. **The radial nerve passes between which two muscles:**
   A. Long and medial head of triceps
   B. Biceps and brachialis
   C. Pronator teres and FDS
   D. Brachialis and brachiradialis
   E. Teres major and teres minor
7. **Which has the highest Young's modulus?**
   A. PMMA
   B. Titanium
   C. Stainless steel
   D. Cortical bone
   E. Ceramic
8. **The most common cause of dropped little and ring fingers is:**
   A. Tendon rupture
   B. Radial subluxation of extensor tendon
   C. Ulnar subluxation of extensor tendon
   D. Neurological causes
   E. Trigger fingers
9. **Which of the materials below is used as a bone graft substitute?**
   A. Tantalum
   B. Titanium
   C. Co–Cr
   D. Aluminium
   E. Ceramic
10. **Which muscle has a dual nerve supply?**
    A. Brachioradialis
    B. Brachialis
    C. Abductor pollicis brevis
    D. Pronator teres
    E. FDS

The time management in this section is very important. You have only 1 minute to read the question, which in some cases will have a long stem, and to mark your answer. If you do not know the answer, flag the question, so that you can come back later if time permits or mark an answer that you guesstimate is the correct answer.

Candidate feedback suggests that approximately 20% of questions are straightforward. These questions test standard textbook knowledge and answers can be easily narrowed down to two choices. The remaining questions are less obvious, stems are tricky and the question needs thinking about, i.e. they are difficult questions especially if you are underprepared.

Advantages of SBA's include:

- SBAs can assess a wide sample of curriculum content within a relatively short time period. This leads to high reliability and improved validity
- They are a highly standardized form of assessment where all the trainees are assessed with the same questions. It is a fair assessment in that all the trainees sit the same exam
- SBA marking is automated, removing examiner subjectivity from the assessment process
- They are easy to administer and mark as a computer-based assessment

Disadvantages of SBAs include:

- The trainee's reasons for selecting a particular option/response cannot be assessed
- Although a wide sample of assessment material can be assessed, there is no opportunity for an in-depth assessment of the content
- Constructing good SBAs needs considerable examiner training
- SBAs developed to test trivial knowledge should be avoided as this may lead to rote learning and fragmentation of knowledge

Advice for paper 1

- There are only 120 minutes for 110 questions so don't spend too long on the easier questions to allow you more time on the ones you find more difficult
- There is no negative marking so you must finish the paper
- Read the question carefully and understand fully what the SBA question is asking of you. In an SBA all of the options available may not be ideal, but you still have to select the best of those available

Paper 2

Extended Matching Item (EMI) questions

The second paper is 'extended matching items' (EMIs). This section comprises 135 questions and the time given to answer these questions is 2 hours and 30 minutes. EMI questions lend themselves to clinical scenarios – For example, data is given on a patient's history and examination findings along with test results and a diagnosis. You will be given a theme or a stem and about 10 matching options. There will be three questions based on the theme or stem. Again the information provided may be incomplete and what is needed is the most likely correct response from the list when you combine the information provided with your knowledge of the evidence and clinical experience (just like the decision-making process that you will have to undertake as a consultant, and that has to be safe).

The typical evolution of an EMI question is that the first time it is used in an exam it is flagged up as 'too easy'. It is removed from the exam, comes back to the question-writing committee, and a debate takes place about what information is essential and what is provided but could differ in the real world without altering the correct response. Information is stripped out, the question returned to the exam and its performance reviewed. In general it is a better question but if the two were looked at side by side, the original would have looked superficially to be preferable

A few examples are given below.

Low back pain

A. Scoliosis
B. Spina bifida
C. Central canal stenosis
D. Degenerative spondylolisthesis
E. Isthmic spondylolisthesis
F. Spondylolysis
G. Spondylosis
H. Cauda equina syndrome
I. Spinal dysgraphism
J. Osteoid osteoma

1. A 60-year-old man presents with pain in the back that is relieved with sitting and leaning forwards and aggravated by walking uphill
2. A 20-year-old gymnast complains of sudden onset of pain in the back after a practice session
3. A 23-year-old woman has back pain with sciatica and her mother has noticed a step in her lower back

Shoulder pain

A. Rotator cuff tear
B. Subacromial bursitis
C. SLAP lesion
D. Glenohumeral OA
E. Secondary impingement
F. Internal impingement
G. Acromioclavicular OA
H. Instability
I. Frozen shoulder
J. Cervical spondylosis
K. Acute calcific tendinitis

1. A 55-year-old man presents complaining of right shoulder pain for 3 months with difficulty in abducting. On examination Hawkins' sign was positive
2. A 23-year-old man sustained a traction injury to this left shoulder. Movement of the shoulder was preserved
3. A 60-year-old man presents with sudden onset of severe shoulder pain. On examination all his shoulder movements are painfully restricted

With regard to fractures of the acetabulum and pelvis

A. Anteroposterior compression injuries with symphysis widening >2 cm and anterior/posterior SI ligament rupture
B. Both anterior and posterior columns
C. The acetabular dome
D. The anterior column
E. The anterior column and posterior acetabular wall
F. The posterior column
G. The posterior column and anterior acetabular wall
H. Unilateral ramus fracture with ipsilateral fracture of posterior iliac crest

1. The structures best visualized by the oblique obturator Judet view
2. Has the highest association with major haemorrhage
3. Fractures associated with the poorest functional outcome

With regard to elbow fracture/dislocations

A. Ulnar nerve palsy
B. Radial head excision
C. Posterolateral rotatory instability
D. Medial collateral ligament injury
E. Essex–Lopresti injury
F. Monteggia fracture dislocation
G. Medial epicondyle fracture
H. Posterolateral elbow dislocation
I. Galeazzi fracture
J. Radial head replacement
K. Intra-articular loose body

1. A 10-year-old boy fell from a trampoline. He was brought to the A&E department with an isolated left elbow injury. On examination the left elbow was swollen and deformed with some bruising medially. Neurological examination showed loss of finger extension
2. A 34-year-old woman presented to a wrist surgeon complaining of right wrist pain for 3 years. Examination of the right wrist showed tenderness of the distal radioulnar joint. There was a lateral longitudinal scar at the right elbow and some tenderness at the scar
3. A 28-year-old man presented to the elbow clinic with a history of painful clicking and intermittent locking of his left elbow. There was a history of a sprain of the same elbow 3 years previously. He doesn't feel confident with this elbow, especially when doing push ups at the gym

The time for the EMQ session is 2 hours and 30 minutes. Candidates should have enough time to answer all the questions without having to hurry through it. Most of the questions are based on clinical scenarios but candidates should have reasonable core knowledge of the subject to answer it. The content of both examination papers is mapped to the curriculum and the curriculum content.

Advantages of EMIs include:

- EMIs provide a convenient method for assessing application of knowledge
- They can assess a reasonable range of the curriculum content in a relatively short time
- They are high in reliability
- All trainees are assessed identically; hence, examiner subjectivity is removed
- EMIs are easy to administer, score, mark and store
- EMIs are designed to assess clinical diagnostic skills and thinking processes relevant to clinical practice

- Due to the large number of options the likelihood of randomly guessing the correct answer is reduced

Disadvantages of EMIs include:

- There is less opportunity for the examiner to assess the trainee in-depth on a given topic
- Writing EMIs is difficult and time consuming. Ambiguity needs to be avoided
- Examiner training is necessary to construct good quality EMIs
- Agreement on the correct/preferred answer may be difficult to achieve especially with 'choice of management' options

Advice for paper 2

- If you are not too sure of an answer then do not dwell for too long deliberating on the answer. Eliminate obviously wrong answers then either do some intelligent guessing or flag the question for later
- If you have flagged questions keep a close eye on the time remaining. As a safety mechanism 5 minutes before the end you should stop everything give an answer to all flagged questions
- There is no negative marking so you must answer all the questions
- Keep a record of how many questions you are answering in a given time. Don't get behind and have to rush your answers at the end. Read the instructions of the EMIs first before answering. Candidates need to watch the clock on a regular basis. Check every 30 minutes that you have answered 30 questions and, if not, speed up. Reading the instruction and stems (which can be as many as 12) is still very time consuming
- Just before the end quickly look through the exam paper to make sure every question has been answered and nothing has been left unanswered

It is a test of time management, examination technique and English language comprehension as much as a test of orthopaedic knowledge and clinical judgement.

Exam generation

An exam is compiled by 'random' selection of questions from a bank by a computer – Random in parentheses, as rules are followed. The proportion of questions from each coded section of the curriculum is the same for all exams and each exam has blocks of established well-performing questions, new questions and rewritten questions. Candidate feedback after every exam always contains self-cancelling comments, e.g. 'there were too many lower limb questions' and 'there were too many upper limb questions', etc.

The first draft, which always contains a few more questions than needed, is securely sent to the chairman of the Examination Quality Assessment (EQA) Group. Their job is to check that there are no duplication of questions and that SBA and EMI questions aren't covering exactly the same material. They will also check that, for example, knee questions do not include the same trauma component that a trauma question covers when it deals with the knee. Similar and overlapping questions are removed to bring the paper down to the correct number of questions while maintaining balance.

This second draft is then considered by a convened EQA group meeting who go through the paper with a fine tooth-comb and pick up potential problems that can be ironed out before the exam. Even at this stage questions can be removed and substituted. Even with several read-throughs spelling mistakes and typos can creep through.

MCQ writing

The Examination Board has a bank of about 1000 questions to choose from for each exam. Questions come into the bank from many sources, but always through the question writing committee. The MCQ writing committee takes proposed questions from all sources and identifies those that can be written into a format consistent with best educational practice before being placed into the question bank.

The MCQ examination committee meets every 3 months for a day to prepare new material. Constructing SBAs needs considerable examiner training. A question will be proposed – It may be brought to the question writing committee by a member who has been asked at the previous meeting to write an SBA on a specific curriculum topic where a question is needed. The question will be projected for the committee to review and about a half will, after 15 minutes or so of debate, be rejected. Otherwise the debate will continue with numerous edits being made, and over the course of typically an hour, the question will be rewritten until it satisfies the committee. The question is then coded and banked as a new question.

Some potential SBAs can be deliberated over for 2–3 hours finally (to the sheer frustration of the committee) to be rejected usually for being too ambiguous. The examination bank is continually being added to and refreshed. The Examination Board is pushing towards developing a much bigger bank of questions that can be utilized for the exams, but this process takes a considerable investment of time, effort and organisation. Some other specialties, in particular General Surgery, have a much larger bank. Questions in the bank are coded to the curriculum so that the bank can be more easily scrutinized and question writing can be focused to address areas of relative deficiency.

As part of the quality assurance process a set number of MCQ panel members are asked to act as volunteers and sit the proposed next sitting of the SBA/EMI paper[a]. From this sitting

[a] Although a useful learning experience, it can be stressful for them worrying that they may score a low mark with gaps in their knowledge exposed.

a number of questions are discarded[b]. Banked questions may need to be updated or ditched in the light of fresh new information available.

When a new question is used in the exam it is flagged up as new and its performance compared to established 'superbank' questions that have a track record of solid performance. A large amount of statistical data is produced on the performance of each and every question. Only if the new question performs adequately will it count towards the final mark of candidates in the exam and be available for use in subsequent exams. If its performance falls short it is removed from the exam and does not affect the final mark of any candidate and with this returned to the question writing committee for review.

The over-riding message is that at any stage if there is any ambiguity whatsoever with a question it has to be discarded.

Educational theory

To ensure adequate content coverage and, therefore, that the exam is a valid test of the breadth of knowledge of orthopaedics, the process of blueprinting occurs. A spreadsheet is created that maps each of the questions to a learning objective on the curriculum. Miller in 1990 introduced an important framework that can be presented as four tiers/levels of a pyramid to categorize the different levels at which trainees needed to be assessed[2]. Although SBAs and EMI can be used to test application of knowledge and higher order thinking, their construction is difficult and in general they assess the bottom two levels of 'knows' and 'knows how' in Miller's pyramid (Figure 3.1).

Bloom et al., in 1956[3], described six levels in the cognitive domain. These were: (1) knowledge recall; (2) comprehension; (3) application; (4) analysis; (5) evaluation; and (6) synthesis. This was revised in 2001 with the use of verbs rather than nouns for each of the categories and a rearrangement of the sequence within the taxonomy (Figure 3.2).

Remembering: Reproduces previously learned material by recalling facts, terms, basic concepts and answers. List, name, label, identify and match

Understanding: Understanding and making sense out of information. Compare, contrast, explain, discuss, demonstrate, describe, summarize, classify and illustrate

Applying: Use information in a new (but similar) situation. Construct, draw, demonstrate, apply, calcualte and illustrate

Analysing: Take information apart and explore relationships. Categorize, compare/contrast, examine and make distinctions

Evaluating: Critically examine information and make judgements. Defend, determine, justify, rate, recommend, appraise, prove, test, critique and assess

[b] Usually if too many committee members fail a particular question. Causes may include the question being too difficult, unclear, ambiguous or obscure, or new evidence challenging a previously credited correct answer.

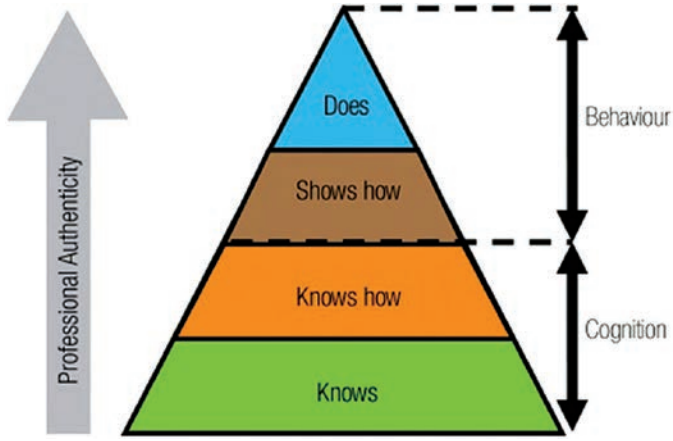

Figure 3.1 Miller's pyramid. Different levels of assessment

Creating: Use information to create something new. Construct, compile, develop, design, produce, improve, change, devise and propose

Bloom's taxonomy is a hierarchical classification, with the lowest cognitive level being 'remembering' and the highest being 'creating'. The lower three levels can be attained with superficial learning so called Lower Order Thinking Skills (LOTS) such as memorisation. The upper three levels involve Higher Order Thinking Skills (HOTS) and can only be attained by deep learning.

EMIs are used to assess clinical aspects at the level of 'knows how' in Miller's pyramid and Bloom's levels 3–6 (application, analysis, synthesis and evaluation). They can, however, also be used to test factual recall of knowledge and understanding (the first two levels of Bloom's taxonomy) and the 'knows' level of Miller's pyramid.

An ongoing development of the examination is the progressive rewriting of questions in the bank that are currently recorded as level I questions (factual knowledge) into higher order questions.

Standard setting

After the papers have been sat they are automatically marked and at this stage there is simply a raw mark indicating how many correct responses each candidate achieved. As mentioned previously, extensive data is kept on how each and every question is answered. As an example of the sort of data collected, the final scores of candidates are ranked and divided into quintiles. For each possible response to each question, data is generated on how each quintile of candidates responded. One measure of question reliability will be to look at how it predicts the final result of a candidate – A 'good' question will be answered correctly by almost all of the candidates who end up in the top 20% and incorrectly by most candidates who end up in the bottom 20%. All of this data is stored in the bank with the questions and is available when questions are reviewed.

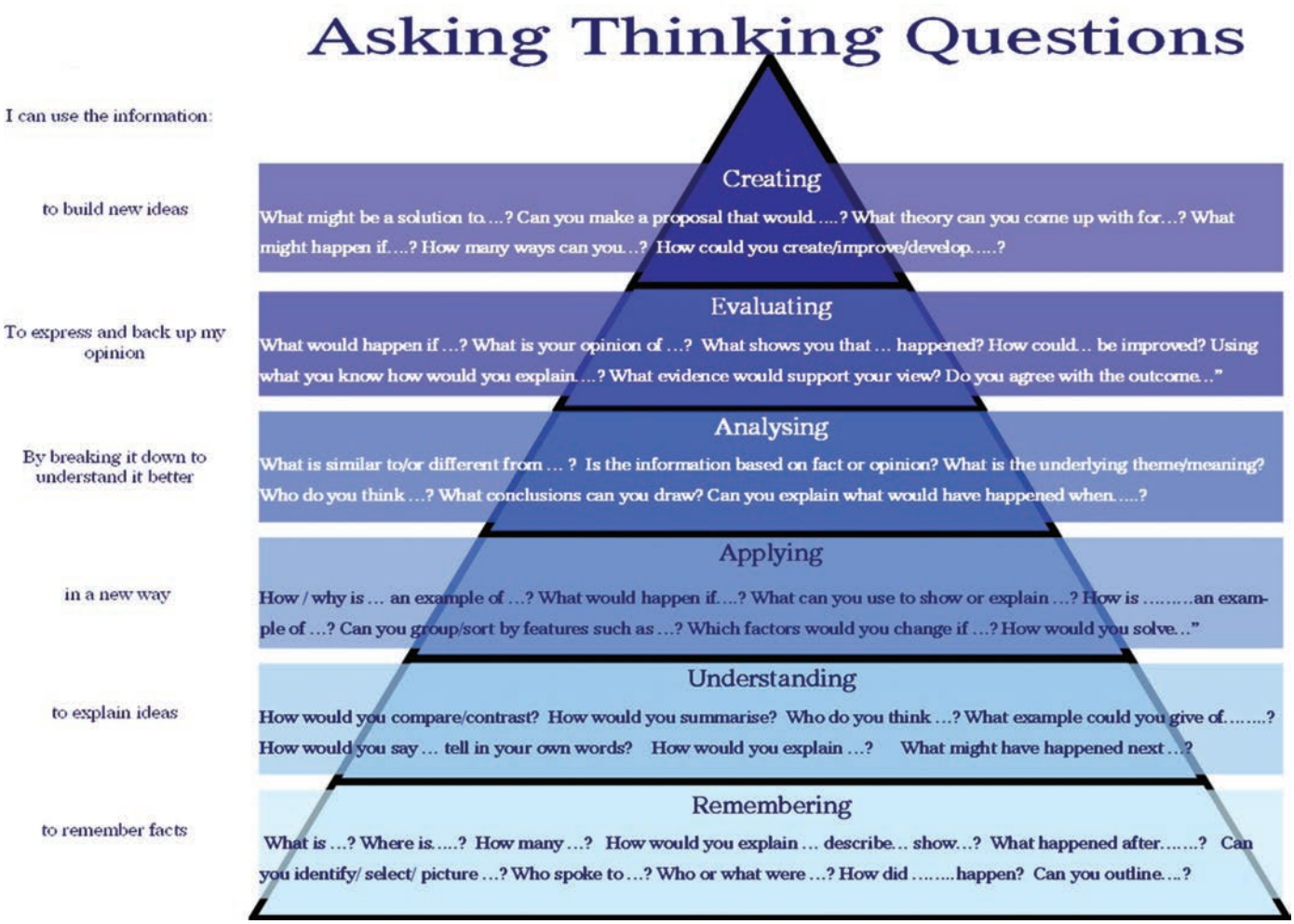

Figure 3.2 Bloom's revised taxonomy. Asking thinking questions

Facility refers to how easy a question is – If 90% of all candidates get a question right or wrong it is too easy or too hard and is a worthless question. Such questions are removed from the exam and do not count towards a final mark, but are sent back to the question-writing committee. If the purpose of the exam was to identify the best and worst candidates in the country reliably, giving a national rank, then these questions would be essential. However, the exam has to discriminate reliably around a pass mark based on specialty standards and by removing 'too easy' and 'too difficult' questions from the final consideration the middle ground becomes 'stretched out' and separates candidates better around the pass mark.

The process of standard setting is quite a time-intensive process involving around 20 or so experienced examiners. They will be first split into two groups to look at some of the SBA and EMI questions that have been flagged statistically as poor performers. Some questions will already have been removed automatically – For example all the questions that proved too difficult or too easy (usually new questions, as any question previously used would have passed through this hurdle already). The examiners will review each question and decide whether it is a fair question that should stay in the exam, or is flawed and should be removed and returned to the question writers. Reasons include unrecognised ambiguity, new evidence challenging previously held beliefs or simply the answer in the question bank is incorrect. Some very good questions end up being flagged up as having possibly the wrong answers yet are absolutely fine. For example, if a question is hard so that only 20% of candidates answer it correctly then 80% will choose the wrong response. If, say, 40% chose one of the incorrect stems – This flags as a possible wrong answer automatically, as more candidates have chosen a specific incorrect response that the correct one

The mark for eligibility to proceed is that which would be obtained by the candidate who just meets the standards required by the specialty and the GMC. This is often loosely defined as a Day 1 Consultant working in a District General Hospital who has spent an appropriate period of time revising for the specialty exam. This is somewhat ambiguous and contradictory.

A criterion-referenced method is used, which means that theoretically if everyone performed well there could be a 100% pass rate. However, recently the pass mark has been rising and

perhaps an understanding of the standard setting process will demonstrate how the exam attempts to be fair.

The most common method used is a modified Angoff method in which a panel of examiners reviews each question and is asked what proportion of borderline candidates would be expected to answer this question correctly. Each examiner works independently and considers each question in turn. The examiners are not told the answers – They do not need the answer paper to recognise how a borderline candidate will behave faced with a particular question, each having had considerable experience of borderline candiates both in their role as trainers and as examiners. Their judgements are combined and then reviewed in light of the results of previous examinations. A further round of discussion and altering of original estimates occurs before the final pass mark is decided.

To simplify matters, if we consider that the whole exam had only 10 questions and all of the examiners independently concluded that 6 of every 10 borderline candidates would get each question correct, then a pass mark of 6 out of 10 (60%) would mean that 50% of borderline candidates would pass and 50% would fail. The pass mark, therefore, divides the borderline candidates down the middle. If the pass mark has a lot of hard questions the pass mark will be lower. If there are a lot of easy questions the pass mark will be higher. The pass mark is unique to each diet.

In some high stakes examinations the pass mark is increased by one standard error of measurement (Standard deviation $\times$ $\sqrt{}$(1–reliability)) in order to reduce the chances that a truly borderline candidate will pass the exam. This occurs for the FRCS (Tr & Orth) exam as the exam is set for a standard of competence, not for a certain percentage of candidates to pass. For patient safety reasons the GMC would not want incompetent candidates being allowed to proceed, even if removing them means some potentially competent candidates are prevented from doing so. When this step was first introduced the historical performance of candidates scrapping through was reviewed and it was noted that they went on to fail section 2.

The FRCS (Tr & Orth) exam has data on its reliability including Kronchbach alpha values. For high stakes examinations the standard aspired to be is a Kronchbach Alpha around +0.8. Very few professional examinations, particularly in the medical specialties, achieve this. Part 1 of the FRCS (Tr & Orth) has never dropped below +0.9.

MCQ revision resources

Using MCQs for revision can impact positively or negatively on the final outcome. As a tool for summative assessment, appropriate questions will reliably demonstrate progression in understanding. When used as a revision guide having answers available during the perusal of the questions can lead to a false sense of security for the ill-disciplined student. Additionally, getting an SBA question right does not necessarily indicate that you know that topic particularly well.

It is critical to decide what type of revision you are going to use your MCQs for during each session and to only review the answers at the appropriate time. Incorrect answers should stimulate a review of the entire topic so that a deeper understanding can be approached.

UK in Training Examination (UKITE)

The UKITE is a national curriculum-based examination for a trainee that provides practice for the 'real' FRCS (Tr & Orth) exit examination.

It is a voluntary optional exam held during December every year. That said it is becoming increasingly more difficult to opt out of the exam. Trainees will appear on the radar of the TPD if they choose not to sit the exam. It is sat online and is free, but to be eligible to sit you have to be registered to elogbook and submit one MCQ and one EMI question in advance.

The exam was originally set up for specialty registrars but now CT2 trainees are encouraged to sit. Any SAS doctors wishing to take the test should contact their regional programme director. This should not be a big issue and the majority of SAS doctors should be allowed to sit the test if they wish to do so.

The test originated in the Northern Deanery in 2007 and each year the number of trainees taking the exam has grown. Nearly all deaneries now participate and most deaneries usually conduct the exam in the stipulated curriculum teaching time so that trainees do not have to arrange time off work. In 2014 the British Orthopaedic Association (BOA) integrated the UKITE examination into the BOA membership; thereby trainees sit this examination through its website.

The test has not been validated and there are a lot of disclaimers on the website. It cannot be used as a summative assessment tool during annual reviews, but it is excellent as a tool for monitoring your own progression of learning. Most trainees (80%) think the questions are equivalent to the actual standard of the FRCS (Tr & Orth) exam, while 10% think it is harder and the remaining 10% consider it easier. Scores are fairly consistent from ST3 to ST6 but jump up considerably just prior to the actual exam and dip down again in the final year of training.

Once you have made your EMI/MCQ choice and formally submitted it during the UKITE exam, you are given the correct answer with an explanation provided during the test. As time is tight most candidates just get on with the exam and don't read over the answers. You are allowed later to access the test and can go through the answers in a more leisurely fashion. At the end of the test you are given an immediate score.

Candidates can practice refining their MCQ tactics in a less nervous environment than the real exam. It should encourage trainees to read more formally and consistently through the various orthopaedic textbooks earlier than the usual 6–12 months before the actual exam.

The test contains 140 questions that are a combination of SBAs and EMIs. The maximum time for the exam is 2 hours and 45 minutes. The questions cover different topics from the whole orthopaedic curriculum.

The questions, answers and their explanations are available online for 3 months after the exam for further revision and this is a good opportunity for candidates to review the material of their incorrect answers.

UKITE and the FRCS (Tr & Orth) exam

Some of the questions in the UKITE bank have been sent to the FRCS (Tr & Orth) examination board for possible use. The actual FRCS (Tr & Orth) MCQ/EMI questions are difficult and time consuming to write. Questions are in short supply with a small bank of verified questions.

The FRCS (Tr & Orth) committee is unsure whether the submitted questions are worth the extra effort required to reach the FRCS (Tr & Orth) standard. They still spend a long time reworking and refining questions put forward by UKITE to avoid any ambiguity in their wording, and believe it may be easier and less hassle for the committee to simply construct them from scratch.

Online resources

Question banks and past questions can often be acquired from recent successful candidates, but two large online question banks currently stand out as potentially useful resources.

Over the last few years Orthobullets[c] has become a most useful resource for both revision and for questions. It is constantly being updated and reflects a lot of what has been written in Miller. The topics are related to MCQs and there are several cases presented that stimulate lively debate amongst the worldwide orthopaedic community.

Orthobullets is presented as a revision resource with information provided as bullet points. A deep understanding of all the topics is not possible without further reading. On its own it is probably not enough to confidently secure a pass in the exam. The anatomy in particular is not detailed enough to give confidence for the exam.

The American Academy of Orthopaedic Surgeons (AAOS) has a large data bank of MCQs. This question bank is geared towards the American exams and the structure and question style of the FRCS (Tr & Orth) MCQs are quite different. Despite this, they are a very popular resource for FRCS (Tr & Orth) candidates preparing for their part 1 exam.

One word of caution. Questions in the FRCS (Tr & Orth) bank evolve from exam to exam – Subtle changes make big differences to the correct answer. If you practice on a website and think you recognise the question in an exam be very careful indeed as there are a number of questions which, when used, generate very interesting responses. Clearly there is a correct answer that is agreed by all the examiners present, but when a whole cohort of otherwise sensible candidates plump for the same incorrect answer – Now why are they doing this? Unofficially these websites are consulted when a question is being proposed to make sure factual information out there on the website is correct.

MCQ terminology

It is important to understand the terminology used for MCQs:

- 'Always' means 100% of the time and is unlikely to be true
- 'Never' is another absolute term and may often be wrong
- 'Occasionally' can make many options potentially viable as correct answers and confuse you
- 'Commonly' means >75% or even more of the time
- 'Rarely' is equivalent to something that occurs <1% of the time
- 'Associated with' means that there is a definable link between the theme and this option
- 'Pathognomonic' means that if this particular item is not present in the stem it would cause the diagnosis to be in doubt

As 'always' means 100% and 'never' means 0%, they both are often wrong. Some advice given by ex-candidates is not too spend too long on very difficult questions as they will be rejected by the exam board. We are not entirely sure about this guidance, inherently it seems wrong. It is highly unlikely these difficult questions would get past the exam-setting committee in the first place if there were any concerns with them.

Questions are written to avoid cues being taken to allow guessing. For instance, the order of possible answer choices is simply alphanumeric. The possible answer choices are adjusted to be of similar length (in lesser exams the possible answer that is longer or shorter than the rest is the correct one!) and all possible answers will be of the same nature (e.g. if being asked about a diagnostic test the possible answers will all be radiological investigations rather than four radiological tests and one blood test). The bottom line is that candidates should not try to look for clues or patterns. If you have to guess you have to guess[d]. There are no negative marks.

Books

There are few decent MCQ books on the market for the FRCS (Tr & Orth) exam. It is very difficult and time consuming to construct good quality, relevant MCQ questions that meet the required standard for the exam. There have been concerns raised that some candidates may use these types of books as a major tool for learning the material for the examination. This will not get you through the exam, especially the orals, as at best these types of books are really only useful for quick revision near the end of your preparations, a sort of confidence boost.

A word of caution about MCQ practice books – The examiners are not allowed to write these books, so any published book is written by someone with no experience of the FRCS (Tr & Orth) writing group.

[c] www.orthobullets.com

[d] But just don't guess randomly- try at least to have an intuitive guess.

Courses

There are some MCQ practice courses available that give trainees the opportunity to assess their knowledge in an exam setting. Some candidates felt they were worth attending to improve technique and confidence. They are helpful for last minute quick revision but are not a substitute for adequate overall preparation. Regular practice of answering MCQs/EMQs using Orthobullets or, even better, UKITE experience would be more fruitful than attending these courses and spending unnecessary money.

As has been announced on the Joint Committee on Intercollegiate Examinations (JCIE) specialty board website[e], the allowed number of attempts for both parts will be restricted with no re-entry, so the best plan of action is proper preparation, starting very early before applying for the examination, asking as many colleagues who have previously done the exam about their experiences and setting up a study group. This is a demanding exam and the examiners expect a good standard of knowledge and experience to pass you. Remember, however, that the majority of candidates who work hard pass the examination in one or two attempts.

If you are one of a small number of candidates who has difficulty answering the SBA/EMI format then you will need to prepare more thoroughly for this part of the exam than the average candidate. Spend as much time as possible answering practice questions. Do not cut corners and learn the subject as comprehensively as you can. Some candidates may by personality over analyze SBAs/EMIs and fail this part of the exam despite excellent knowledge. Get professional guidance if necessary, as exam attempts are limited.

On the day of the exam most of us thought we had failed when in fact we had passed. Try not to get too disheartened, as there is nothing left to do for section 1 at this point. As it is computer based, the results can be released quite quickly and it usually takes around a week to find out if you passed or failed with the breakdown of your marks for each paper following in the post a few days later.

Final thoughts about the part 1 exam is that every question in every exam is statistically dissected and each exam is compared to all previous exams. Do not go into the exam thinking you may be treated unfairly – You are a number and enormous effort is put into making sure sound decisions are made on your eligibility to proceed.

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[e] www.jcie.org.uk

Chapter 4

Introduction to clinical examination techniques

Karen Robinson and Fazal Ali

Perfecting the art of clinical examination provides the orthopaedic surgeon the necessary skills to pass examinations but more importantly forms the basis on which accurate diagnosis and improved patient care will result. In this chapter we have laid out a general approach to clinical examination to use as a foundation to build upon as you learn.

General principles

It does not matter in which situation clinical examination is taking place. There are certain basic rules that should be mandatory on every occasion.

Respect the patient

Make the patient feel comfortable and at ease. Be polite and respect their dignity at all times.

Expose the patient

The area being examined must be adequately exposed whilst maintaining the patient's dignity. Expose the patient as much as possible from the joint above to the joint below, which you are examining. If you think scars may be hidden under clothing, ask the patient if they have any scars in that area and ask if they would show them to you.

Give clear instructions

Give clear and unambiguous instructions to the patient. It is often easier to demonstrate what you want them to do rather than try to explain. Patients are not exam trained. They may not understand exactly what it is you want them to do.

Observe all the time

Start your observations from initial contact with the patient and observe the whole patient before focusing on the area to be examined. Are they young or old? Do they have a walking aid? Do they have difficulty standing or undressing? Store this general information away to help you build your clinical picture.

Don't cause pain

Be careful not to get so engrossed in the examination that you forget to engage with the patient. Look at the face when examining so you know if what you're doing is uncomfortable or painful. Avoid hurting the patient.

Equipment

You will need some basic equipment for clinical examination and it is best to have this ready to use when needed.

- A tape measure used commonly for leg length measurements, but can also be used as a plumb line and to quantify muscle wasting
- A key, a 50 pence coin and a pen for a functional examination of the hand
- A goniometer to quantify range of motion
- Two pens to demonstrate range of forearm pronation and supination
- A tendon hammer for a neurological examination

Sequence of examination

The majority of orthopaedic examination is based on the sequence of look, feel and move. The elbow and wrist joints may flow better if performed as look, move then feel.

Stand the patient

Start every examination (except the hand and wrist) by asking the patient to stand. Ask them to stand up straight and put their feet as close together as possible. In the lower limb, standing the patient allows a deformity to be seen on weight-bearing that cannot be seen when sitting. By asking them to put their feet together, varus/valgus deformity is easier to see and limb length discrepancy is harder to hide. In the upper limb, it is easier to examine the shoulder and elbow with the patient standing as you can move around the patient and get a clearer view of the movements.

Look

Start with inspection, look carefully at the patient from all angles and comment on your positive and important negative findings as you go along. Point out what you see so the examiner knows what you are doing, e.g. pointing at supraspinatus 'I can see wasting of supraspinatus, I would like to go on to test the range of motion and the integrity of the rotator cuff'. If the diagnosis is clear from inspection, tell the examiner

what it is and go on to confirm your diagnosis with the rest of the examination, e.g. in the rheumatoid hand. Try to put your findings together, like a jigsaw in your mind, to settle on the diagnosis. Inspection will give you the clues you need to focus the rest of your examination on. Make sure you see what you are looking at, be confident and move on.

Feel

Palpate in an ordered fashion and make defined moves. Demonstrate clearly to the examiner that you know the surface anatomy and can elicit any signs, explain what you are palpating and why. Remember that some joints like the knee, hand, wrist, foot and elbow are superficial and a tender spot will be a clue to the underlying pathology. The shoulder, spine and hip joints are deeper; therefore, palpation is less rewarding in helping to make a diagnosis.

Move

Range of motion and special tests are performed as part of movement. Test all joint movements as active movements followed by passive movements. Assessing the joint in this way will tell you if it is stiffness or weakness that limits the range of motion and guides you to what the patient can do before you touch them. A stiff joint will be restricted in both active and passive testing, whilst weakness will allow you to perform a full passive range of motion.

Further examination

Although the majority of information is gained from the basic steps it is sometimes necessary to perform tests specific to a particular joint in order to confirm findings. In addition, there are certain generalized conditions that affect the musculoskeletal system that may result in pathology. These will need to be assessed.

Beighton's score

Instability or excessive passive movement may be a sign of generalized ligamentous laxity (Figure 4.1). This can be assessed by Beighton's scoring system (Table 4.1). A score of 4 or more indicates hypermobility. These patients present occultly with joint pain for example anterior knee pain. They may present with subluxing or dislocating joints, most commonly the shoulder and patella. A positive sulcus sign (shoulder) and a positive 'J' sign (patella) are also characteristically seen in these patients.

Grading muscle power

Neuromuscular disorders affect muscle strength and should be assessed and commented on using the MRC scale (Table 4.2). It is important to distinguish between grade 2 and grade 3 power. Make sure you know how to eliminate gravity for each of the muscle groups as this can get confusing in the heat of the moment (Figure 4.2). In the lower limb, this usually involves placing the patient on their side.

Table 4.1 Beighton's scoring

Points	Movement
1–2	1 point for each thumb that will bend backwards and touch the forearm
3–4	1 point for each little finger that bends beyond 90° at the MCP joint
5–6	1 point for each elbow that hyperextends
7–8	1 point for each knee that hyperextends
9	1 point for bending over and touching the floor with palms of hands and without bending the knees

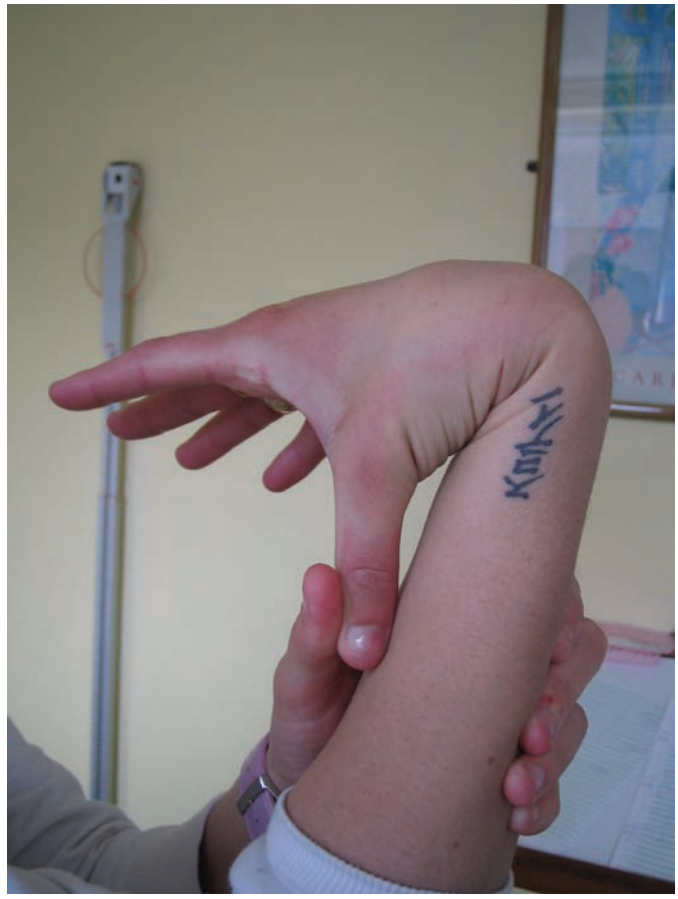

Figure 4.1 Excess passive movement suggestive of hypermobility

Tips on learning clinical examination

Phased learning

Under the pressure of an exam it is commonly clear to the examiner which candidate has not practiced the routine and is, therefore, thinking what the next step is in the sequence rather than trying to pick up the pathology. It is virtually impossible to pick up subtle pathology if one is thinking about the steps in examination. The authors, therefore, recommend that clinical examination is learnt in four phases of preparation:

Table 4.2 MRC grading

Grade	Observation
0	No muscle contraction
1	Flicker of muscle contraction
2	Movement with gravity eliminated
3	Movement against gravity
4	Movement against resisted gravity
5	Full power

Figure 4.2 Testing triceps whilst eliminating gravity

1. Be able to recite the steps

   Know the steps of each examination and be able to repeat them clearly, concisely and swiftly to yourself. You will need to be able to pick up the routine at different points to proceed with the examination
2. Practice on yourself

   Once you can vocalize the steps, practice them alone. Work out clear and concise instructions to confirm how you would ask a patient to do something. Get used to the routine and moving from one test to another
3. Practice on family and friends

   This is the opportunity to practice your routine on the 'normal patient'. Do your instructions make sense? Did they do what you asked them to do? Become confident in knowing what is normal
4. Practice on patients

   Without mastering the previous three stages it is extremely difficult to examine a patient and appreciate abnormalities that are not gross. In patients with multiple deformities most will be missed if the candidate has not repeatedly practiced their routine to the point where it becomes automatic

   Feedback is a vital way of evaluating your performance. Ask a colleague or your consultant to watch your examination and give you structured feedback that you can use to improve your performance. Are you demonstrating tests clearly? Are they confident that you look like you know what you're doing?

Treat it like a dance

A dance is a choreographed sequence of events. Treat the clinical examination the same. Learn the steps, repeat them the same way each time and practice the technique. This way, under the pressure of an examination situation, well-rehearsed routines will not suddenly be forgotten.

Treat it like a driving test

Like the 'mirror–look–manoeuvre' in a driving test it is sometimes beneficial to over exaggerate a technique in order to visibly demonstrate it to the examiner. Tell the examiner what you are doing and be seen to be doing it, e.g. look at the face for Horner's and say 'I am looking for signs of ptosis, myosis and enopthalmos', indicating the presence of Horner's syndrome or not. Other situations where this method can be used include squaring of the pelvis when laying the patient on the couch for hip examination and looking at the shoes and soles of the feet in a foot and ankle examination.

Special situations

There are many situations in an exam where an uncommon presentation faces the candidate. Dealing with this sometimes takes some thought. It is best, therefore, to think of how you would approach these cases beforehand. A few examples of these situations are presented:

1. A patient with an adduction deformity of the hip presents with apparent shortening

   This patient will compensate by using a shoe raise on the affected side, tilting the pelvis and trunk or by bending the other knee. For every 10° of adduction deformity there will be 2.5 cm of apparent shortening
2. A patient with an abduction deformity of the hip presents with apparent lengthening on that side

   This patient will compensate by using a shoe raise or stand on tiptoe on the contralateral side, tilting the pelvis and bending the ipsilateral knee. For every 10° of abduction deformity there is about 2.5 cm of apparent lengthening
3. A patient presenting with a fixed flexion deformity of the hip may be compensating by increasing the lumbar lordosis. Up to 30^0 of fixed flexion deformity of the hip can be compensated for by an increased lumbar lordosis

4. How do you do Thomas' test in a patient with a fixed spine such as ankylosing spondylitis?

   Proceed as normal and flex the contralateral hip as far as possible. This will remove any remaining compensatory movement there is in the lumbar spine. The lumbar spine does not need to be flat on the bed as long as any compensatory movement is obliterated
5. How do you do Thomas' test in a patient with an arthrodesis of the contralateral hip?

   Lift the arthrodesed contralateral hip until the lumbar curve flattens. At this point measure the fixed flexion deformity of the hip being tested
6. How do you do Thomas' test in a patient with a total hip replacement on the contralateral side?

   Proceed as normal but in a controlled manner. Place your hand underneath the lumbar spine of the patient and slowly flex the hip. Do not flex past 90^0 or risk dislocating the hip. Once the lumbar curve is obliterated, measure the fixed flexion deformity of the hip being tested
7. How do you measure limb length in a valgus deformity of the knee?

   This should be done in segments, measuring from the anterior superior iliac spine to the lateral condyle and from the condyle to the malleolus. It is impossible to recreate the deformity in the contralateral limb and, therefore, measuring in segments is the only way to comment if a limb length discrepancy is present
8. In what situation would you not want to use a tape measure as a means of assessing leg length?

   If the patient has any evidence of pathology in the foot then there may be shortening below the level of the malleoli such as with subtalar arthritis or previous calcaneal fracture. In this case it is more accurate to measure leg discrepancy using standing blocks
9. In what situation would you not want to use standing blocks as a means of measuring leg length discrepancy?

   If there is a fixed flexion deformity in any lower limb joint the use of standing blocks will not accurately measure the discrepancy. A tape measure should be used in this case as the contralateral limb needs to be placed in a similar position of deformity to comment on leg length discrepancy
10. How do you test Serratus anterior when there is no wall to push against?

    Stand at 90^0 to the patient and ask them to push against your hand with the affected arm. Apply a counter pressure and look at the scapula for winging as they do this (Figure 4.3)

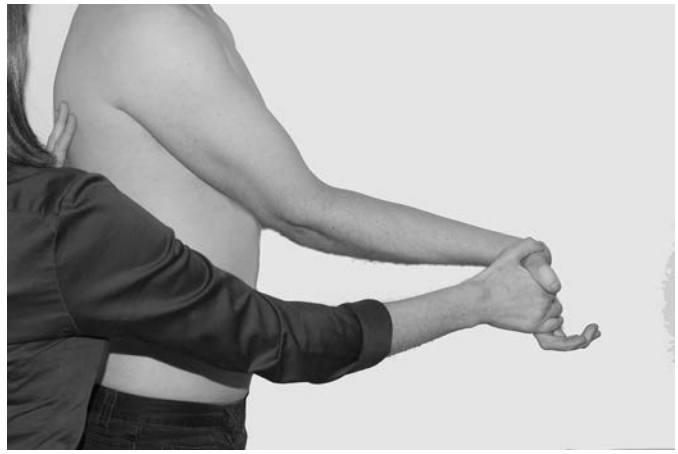

Figure 4.3 Testing serratus anterior without a wall

How special tests relate to anatomy and pathology

The basis of special tests is to utilize the damage to an anatomical structure in order to elicit a sign. It is important to know what you are testing and why. By relating the anatomy and pathology to the signs that you are eliciting you will be able to piece together the information and make a diagnosis. A good example of this is the brachial plexus examination. A clear understanding of the plexus anatomy and understanding how to examine each nerve will help you to pinpoint where along the brachial plexus the injury is likely to be.

Some other examples include:

a. Abdominal reflexes

   Abdominal reflexes are tested by stroking the abdomen in each of the quadrants with the sharper end of a tendon hammer. The umbilicus is T10 dermatome and is used as the centre point of the four quadrants. Light dermatomal touch is transmitted via the sensory afferent nerve to a short spinal arc. The efferent nerve is to the segmental myotome innervation of the abdominal muscles and, therefore, a visible muscle contraction underneath the point being stroked occurs. In the presence of a syringomyelia or other thoracic spine pathology there is a block of the electrical conduction and the contraction is not seen. For example, if there is no abdominal contraction above and to the right of the umbilicus then the pathological lesion is above T10 and on the right side

b. Allen's test

   Allen's test looks at the integrity of the ulna and radial arteries distal to the wrist. It is based on the fact that the ulna and radial arteries contribute to both the superficial and deep palmar arches. It is particularly important when assessing the patient prior to excision of a volar ganglion. Establishing the presence of a good collateral circulation from the ulna artery in case of damage to the radial artery at time of excision is important

c. O'Brien's test

   O'Brien's test can be used to detect superior labrum from anterior to posterior (SLAP) tears. When the arm is placed, at 90° forward flexion, adducted 15° and internally rotated the long head of biceps tendon is subluxed from the

Figure 4.4 Provocative test for Golfer's elbow

Figure 4.5 Provocative test for Tennis elbow

bicpital groove. As a consequence, resisted shoulder flexion is painful. When the arm in kept in the same position but externally rotated, the biceps tendon relocates into the groove and the pain is no longer present with resisted shoulder flexion

d. Dial test

The dial test tells you whether there is an isolated posterolateral corner (PLC) injury or whether this is combined with a posterior cruciate ligament (PCL) injury. It utilizes the fact that the PCL is active when the knee is in 90° of flexion but inactive at 30°. The PLC resists the external rotation forces; therefore, the foot will externally rotate more than the contralateral side with a PLC injury. Hence, if the PCL is also ruptured there will be no restriction at 90° and the foot will continue to externally rotate indicating a combined PCL and PLC injury. If the PCL is intact it will restrict movement and rotation will be much less at 90°

e. Coleman Block test

Table 4.3 Sensitivity of clinical tests

Name of test	Sensitivity
Hawkins–Kennedy test[5]	0.74
Neer's test[6]	0.79
Tinel's sign[3]	0.27
Phalen's test[7]	0.77
Trendelenburg's test[8]	0.94
Anterior draw[9]	0.55
Lachman's[9]	0.85
Pivot shift[9]	0.24

The Coleman Block test demonstrates if the varus deformity of the foot is forefoot driven (due to a plantar-flexed first ray) with a flexible hindfoot or a fixed hindfoot deformity. Ask the patient to stand with their foot on the edge of a block with the head of the first ray off the side (it may be easier to demonstrate to the patient what you want them to do). Observe the foot from the back, does the hindfoot swing into valgus? If so the hyperflexed first ray is driving the hindfoot into varus and the hindfoot is flexible. If the hindfoot remains in varus, then the deformity is fixed in the hindfoot. The Coleman Block test. Therefore. has a significant role in helping to decide surgical treatment

f. Testing for Golfer's elbow and Tennis elbow

Provocative tests for Golfer's elbow and Tennis elbow are based on the knowledge the forearm flexors attach onto the medial epicondyle and extend across the wrist joint and into the hand. Similarly the forearm extensors attach to the lateral epicondyle and extend across the wrist joint. Resisted flexion and extension are, therefore, used to test for Golfer's elbow and Tennis elbow respectively (Figures 4.4 and 4.5)

Reliability/sensitivity of tests

There are many special tests in orthopaedic clinical examination. Many of them have no routine place in everyday clinical practice and are never tested in examinations. Many of them have poor reliability and sensitivity. On the other hand there are some clinical tests that have great importance in diagnostic evaluation and are tested in clinical examinations.

In addition to the sensitivity of a single test in isolation, the use of tests in combination has shown to increase the likehood ratio (sensitivity/1–specficity) and, therefore, increase the probability of a pathology being present. For example, in a full thickness rotator cuff tear, the combination of a positive painful arc sign, Hornblower's sign and infraspinatus muscle test raises the post-test probability to 0.91 from 0.33 if only one of these is positive[2].

Some commonly used tests have a low sensitivity. Tinel's sign is an example of this. It has a high specificity and, therefore, good at identifying the true negatives, but a poor sensitivity[3]. In contrast, Phalen's test has a better sensitivity and, therefore, the two tests can be used together to help build a clinical picture.

Anterior tibial translation elicited with a positive Lachman's test has good sensitivity for a complete or partial (anteromedial bundle) ACL rupture, although it cannot distinguish between them. The posterolateral bundle (PLB) does not contribute to anterior tibial translation; therefore, the Lachman's test may be negative with a PLB tear[4]. Table 4.3 lists clinical tests that have great importance in diagnostic evaluation and are tested in clinical examinations.

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Chapter 5

The short cases

Mark Dunbar and Andrew P. Sprowson

Having spoken to many candidates about their experience in the short cases, almost all regard this as the most difficult part of the FRCS (Tr & Orth) examination. Simply, you have very little time to show the examiners your competence with each particular diagnosis. The skill is being able to demonstrate that knowledge in a succinct and effective way. Examiners often say that there are few surprises in the short cases as a 'rheumatoid' or 'Dupuytren's' hand is certain to be present in the examination hall. This is often true, but unfortunately you may not see any of these cases and, therefore, cannot simply learn these two diagnoses! There are also a number of more complex cases and these can catch you out if you are not familiar with them. First you must be able to confirm the diagnosis and secondly be able to talk around the topic. If you have nothing at all to say to the examiners, it makes it very difficult to achieve that pass. The examiners will ask you questions on a particular short case especially if there isn't much to find on clinical examination. For example, you may have finished your Dupuyten's exam after 2 minutes but you then have 3 minutes of grilling on the management, consenting issues and complications from surgery of this condition.

The new format of the short cases exam is divided into upper and lower limb sections. These last for 15 minutes each and you have a different pair of examiners. You will see only three cases each for upper and lower limb, having 5 minutes for each. At 5 minutes you move onto the next case, so if you know nothing about the diagnosis it's going to be a long 5 minutes. There is a random allocation of short cases, although in fairness there is usually a balanced mix of cases. You are extremely unlikely to see three hand cases, at the very least a shoulder or elbow case would be included to allow a more thorough assessment of your overall examination technique.

In the heat of the moment we can all say stupid things to the examiners when unsure. Easy cases can be ruined and failed if the candidate rushes into his/her answer. There is undoubtedly pressure for candidates to start talking as soon as possible, but try to pause for a few moments if uncertain about the best way to tackle the problem. Some candidates claim that the key to the short cases is a good start with the opening first case. A poor start can easily deteriorate into a fail if you are unable to turn it around. Avoid the downward spiral. Try not to become demoralized, take a deep breath and attack the next case anew. Allowances are usually made for first case nervousness but, if this continues into the second or third case, the examiners will quickly lose patience and fail you.

The usual advice applies –'Be smart and look the part'. As one of the examiners explained to me, if you stand out from the crowd it wakes the examiner from his semi-conscious slumber to concentrate his efforts on this poor nervous candidate. It goes without saying that you should dress appropriately and conservatively. The chance for you to be extravagant and stand out from the crowd is at a party afterwards to celebrate your successful performance. Have a clean handkerchief available to wipe away the sweat as it pours off your forehead down your face at the most inopportune time.

The examination is usually conducted in the style of a general outpatient clinic. The key to the short cases is being able to reproduce your examination technique under the immense pressure of the exam. The best way to do this is to practice your exam technique over and over and over again so that you perform exactly the same examination of the major joints each time. You should be able to perform these exam routines blindfold and without hesitation as if you have done them 1000 times. It is also unnecessary and unhelpful to learn all 100 different tests for ACL deficiency of the knee. Stick to a simple and reproducible exam regime with one test for each important part of the case. If you stumble through the case trying tests for the first time you will look unprofessional and rarely elicit the correct signs.

Listen to what the examiners ask – If they specifically ask you to perform a Trendelenburg test do so and do not start examining gait because this comes before the Trendelunburg test. This will annoy the examiners and lose you scoring points. The examiners are asking you to perform a targeted specific examination. You may still be asked to examine a knee or hip but this is more likely to be a focused exam based on the history or presentation information guided by the examiner For example, you may be asked to examine a knee, but the examination should differ significantly for an arthritic knee as opposed to a sports knee injury or patellofemoral instability. The good candidate will have prepared and be practiced for all of these examination situations and will effortlessly be able to pull out of his/her examination lexicon a slick technique that demonstrated the appropriate signs. For the safety conscious it may be appropriate to explain that based on the history given you are going to concentrate on the PF joint, for example.

You will usually have two examiners with perhaps a third person present if the examiners are also being assessed or an observer present. They are not actually trying to fail you, but are trying to give you the space to demonstrate what you know. Explain to them what you are doing as it gives them a chance to course correct you. For instance if you are given a patient with pes cavus, you may wish to do a full neuromuscular examination and if you believe this to be the case you should announce it, this then gives the examiner the chance to adjust your plan if in fact what they wanted you to concentrate on was the foot and to demonstrate your understanding of hind-foot mobility using the Coleman block test.

Useful tips and hints

The following tips and hints may seem a bit tired and cliché-ridden, but you may find some of them useful.

- Practise, practise and practise. It is easy to spot candidates who have not practised as they suffer from a lack of polish and fluidity, which affects the examiner's opinion of the overall clinical competence of a candidate
- Look the examiners in the eyes, both of them (and both eyes)!
- Imagine that you are seeing cases in the clinic and presenting your findings to your boss. Treat them like it's your first week in your next post and you want to impress – Don't miss any salient information out
- The most important part is to look slick, as though you have done it a hundred times, even if you haven't
- Take any opportunity you have to practise; ask more senior colleagues, fellows, keen consultants and volunteers at teaching. It's better to look a fool with any of those listed above than fail the exam
- Go to the wards and the day surgery unit to see as many short cases as possible in the 12 weeks before the exam
- Elicit the relevant clinical signs clearly and talk as you go
- The short cases should be fine as you should have seen them all before – There are no tricks, really there are none, the examiners want to pass everybody!
- I am sure they assess you very quickly as to whether they would let you fix their grandmother's fractured hip or not. Show the examiner that you have a logical and methodical manner
- Certain favourite topics always appear in the short cases. Make sure you know these extremely well. (See Chapter 3)
- Listen to the instructions: It's easy to go down the wrong track, which may take you into an area you didn't want to go into
- Look fascinated and grateful if the examiners make a point!
- Take note of the examiner's guiding comments; they are trying to help you
- Smile, be pleasant with patients
- Never make the patient wince. If you are examining a rheumatoid hand, ask the patient where it is sore today – Show your compassion
- Always stick to the look, feel, move, neurovascular then special tests routine
- Be friendly, courteous and polite to patients and professionals, but moreover relax and be yourself during the examination
- Stay calm and talk sensibly even when the diagnosis appears unclear
- The short cases are very artificial but you have to feel comfortable with your technique. If you feel embarrassed or lack confidence in your approach you will flounder
- Do not rush a case
- If you are unsure of the diagnosis then describe the findings and give them a differential diagnosis
- Try not to be obtuse in the short cases or to pick on unimportant details, as the examiners may then draw you into a frustrating and often irrelevant discussion as to what you mean and side-track you away from the main issue
- Get down to eye level with children and try to make them feel at ease
- This is where you need your wits about you. The questions are straightforward but it is an unnatural situation
- Always thank the patient afterwards, just before you move on
- No amount of bookwork can prepare you for the short cases. As much experience as possible beforehand under examination conditions is the best preparation. Volunteer at teaching, every week!
- Although candidates should be fully prepared for an Apley approach this may not be what the examiners want, so be flexible
- Do not argue with the examiners and be polite to patients
- Do not panic. This is greatly helped if you have practised a lot of short cases under pressure and have seen most things before
- Keep your head and think before you speak
- In my experience, the examiners seemed to vary in terms of their expectations from candidates for the short cases. In my examination all candidates saw just three cases, so you had the full 5 minutes with each patient
- In previous examinations, examiners wanted the candidate to see patients for a spot diagnosis and then discuss the case whilst walking to the next patient. As the examination becomes more standardized this approach is becoming much less common
- Do not take the exam unless you can prepare properly for it
- Make sure your answers conform to safe practice. If there are guidelines and you know them, let the examiners know
- The right tone to strike is friendly, efficient and business-like
- The short cases seem to fly by – Remember the obvious things such as be nice to your patients and introduce yourself. If you don't know something, say so and do not waffle – The examiners don't like people who waste time on things they know nothing about

- If you say something stupid, simply retract the statement immediately and explain that you made a mistake and apologize
- You need to work out how quickly your examiner wants you to go but if you are not sure go through the look, feel and move plan. The examiner will guide you firmly
- At the end of your short cases try not to dwell too much on your own performance even if it has fallen short of expectations. Refocus and move on to the next part of the examination – Don't get downbeat
- Keep positive; it's amazing how many candidates feel they have failed, yet have a good pass
- Do not assume that you have failed and not turn up for the remaining part of the examination. There are legendary tales of candidates not bothering to turn up to the orals thinking they had failed the clinical only to have subsequently found out they had comfortably passed the clinical
- Most importantly, believe in yourself and the skills you have gained over your training

Short case list

This is a list of common cases that are likely to come up in the exam and stir up uncomfortable feelings of hard work ahead. Try to imagine the typical scenario of each case, the likely positive clinical findings and possible questions the examiners will ask afterwards. This is not an exhaustive list. In the chapter we have commented on the likely frequency, so you can grade your efforts toward each topic. If you have left things late, then concentrate on the common topics first and finish with the rare. Think about the sort of patients that would be regularly available for an examination. Conditions that are painful will not be present, as 10 candidates cannot examine a painful condition. Make a list over the 6 months prior to the exam of conditions that candidates may encounter.

A number of short cases may have many positive clinical findings present especially if there is dual pathology. This makes them equally applicable to be used as intermediate cases and it is just the way the dice has rolled that they have been chosen as a short case. There is a lot of get through in these patients but the examiners will focus you on what they want you to examine.

Shoulder	
Common	You need to have an in-depth understanding of the topic, including surgical procedures. You are likely to get one of these topics in either the short or intermediate cases. Neglect this topic at your peril!
Frequent	You need a good grasp of the topic. Not a dead certainty as above, but not rare. All our short cases fell into this category, as we did not get any of the dead certain
Rare	You would normally see very few in clinic, but there may be one in the exam hall. You don't need to be a world expert, but have a few basics to hand

Elbow	
• Acromioclavicular (AC) joint dislocation	Common
• AC joint pain	Common
• Brachial plexus muscle power testing	Frequent
• Clavicle non-union	Frequent
• Erb's palsy	Rare
• Frozen shoulder	Rare
• Impingement tests	Frequent
• Instability of the shoulder post trauma	Frequent
• Instability testing – Unidirectional and multidirectional	Frequent
• Klippel–Feil syndrome	Rare
• Long head of biceps rupture	Frequent
• Osteoarthritis (OA) shoulder	Frequent
• Pseudoarthrosis clavicle	Rare
• Pseudoparalysis shoulder (septic arthritis) – Destruction of the humeral head as an infant	Rare
• Rotator cuff pathology and testing of muscle strength	Common
• Voluntary posterior dislocation of the shoulder	Frequent

Wrist and hand	
• Bilateral congenital radial head dislocation	Rare
• Congenital dislocation of the radial head	Rare
• Congenital absence of forearm	Rare
• Cubitus valgus	Common
• Cubitus varus	Frequent
• Madelung deformity plus osteochondromas	Frequent
• Osteoarthritis elbow post trauma	Frequent
• Radioulnar synostosis	Frequent
• Rheumatoid elbow	Frequent
• Rheumatoid nodules	Common
• Distal biceps rupture	Rare
• Tennis elbow – Demonstration of tests	Common

Hip	
• Congenital abnormality – Cleft hand, syndactyly, etc	Frequent
• Bilateral Dupuytren	Common
• Bilateral Dupuytren's plus peripheral neuropathy	Common
• Base of thumb OA	Common
• Carpometacarpal OA	Frequent
• Combined nerve lesions	Frequent
• Deformed hands due to Ollier's disease	Frequent
• Demonstration of Allen's test	Frequent

• Extensor pollicis longus (EPL) rupture	Common
• Ganglion	Frequent
• Kienböck's disease	Common
• Madelung's disease	Frequent
• Non-union of radius and ulna	Frequent
• Psoriatic arthropathy fingers with nail changes	Rare
• Quadriga effect	Rare
• Rheumatoid hand	Common
• Reflex sympathetic dystrophy (RSD) post-ulnar fracture	Frequent
• Severe carpal tunnel syndrome	Common
• Scapholunate advanced collapsed wrist (SLAC) and scaphoid non-union advanced collapsed (SNAC) wrist	Frequent
• Spaghetti wrists	Rare
• Ulnar claw hand	Common
• Wrist drop	Frequent

Knee

• Arthrodesed hip	Frequent
• OA secondary to avascular necrosis (AVN) post-open reduction internal fixation (ORIF) acetabular fracture	Frequent
• Perthes' with secondary OA	Common
• Untreated developmental dysplasia hip	Frequent
• OA hip	Common
• Post-traumatic slipped upper femoral epiphysis (SUFE) hip	Frequent
• Polio with limb length discrepancy (LLD) and Trendenlenburg gait	Rare
• Healing stress fracture hip	Rare

Ankle and foot

• Anterior cruciate ligament (ACL) plus posterolateral instability	Rare
• ACL rupture	Common
• Blount's disease	Frequent
• General examination including checking for effusion/synovial thickening	Frequent
• Lateral meniscal cyst	Common
• Medial collateral ligament (MCL)/ACL laxity post-knee dislocation	Frequent
• Open tibial fracture treated with external fixation (EF) and then circular frame with free flap. Stiff knee	Frequent
• Osteochondral defect of the knee	Frequent
• Patellectomy	Frequent
• Post surgery for high tibial osteotomy (HTO)	Common
• Post-compartment syndrome release of the leg	Frequent
• Pigmented villonodular synovitis (PVNS) knee	Rare
• Semi-membranosus bursa	Rare
• Testing for ACL and posterior cruciate ligament (PCL) injury	Common

Spine

• Ankle arthrodesis	Common
• Arthrogryposis with bilateral clubfoot	Rare
• Calcaneal fracture with Volkmann's ischaemic contracture	Frequent
• Drop foot	Frequent
• Gout ankle	Frequent
• Growth arrest after physeal injury	Frequent
• Haglund's deformity	Frequent
• Hallux rigidus	Common
• Hallux valgus	Common
• Hereditary motor and sensory neuropathy (HMSN)/bilateral foot drop	Rare
• OA ankle	Common
• Pes cavus: HMSN, spinal dysraphism	Frequent
• Polio	Frequent
• Rheumatoid foot	Common
• Synostosis of tibia/fibula and degenerative ankle	Frequent
• Tarsal coalition	Frequent
• Tibialis posterior tendon rupture	Common

Paediatrics

• Neurofibromatosis and scoliosis	Frequent
• Spinal stenosis	Common
• Cerebral palsy with foot and knee problems	Common
• Curly toes	Common
• Erb's palsy	Rare
• Arthrogryposis multiplex congenital (post-fusion)	Rare
• Genu varum/valgus	Common
• Osteogenesis imperfecta	Rare
• Overriding fifth toe	Frequent
• Proximal femoral focal deficiency	Frequent
• Lateral subluxing patella	Frequent
• Posteromedial bowing with LLD	Rare
• Surgically treated clubfoot	Frequent
• Femoral anteversion	Frequent

Chapter 6

The intermediate cases

Neil E. Jarvis, Puneet Monga and Stan Jones

Two intermediate cases replaced a single long case in 2009, each case lasting 15 minutes, and subdivided into history, examination and discussion. Usually a candidate will be examined on an upper and lower limb case, although they may get any combination of cases (UL/LL, LL/LL, paeds/LL, etc) depending upon the patient mix available. There are 96 individual scoring opportunities in part 2 of the FRCS (Tr & Orth) examination; 12 are in each intermediate case (24 marks in total). There will be two examiners for each intermediate case, and a candidate will never be left alone with the patient; all parts of the history and examination will be directly observed.

A well-rehearsed and slick technique is essential for the intermediate cases as this is a stressful experience. Don't worry if you finish the history and examination quickly as long as it is thoroughly done, there will be more time to pick up points in the discussion. You walk into the intermediate case with a 6, and your mark will go up if you are polished, conversely down if your technique is not up to pat. The first two parts of the intermediate case you are in control and it will be down to you to show what you know.

The examiners will not interrupt during the history unless it is rambling or the patient is garrulous, but it is easy to pick up points here and score a 7 by establishing a rapport, and pretending that you are in clinic[a]. Gel your hands. Be polite and make eye contact. Introduce yourself and shake the patient's hand, there are marks available for all of the above. Begin with their age, occupation or hobbies if retired, and hand dominance. Then ask about their chief complaint and how it all started, and its effect on their activities of daily living. Ask about previous operations and medical history – The patient may give you the diagnosis! Don't forget to complete your history as normal with allergies, social and family history. Listen to the patient, but keep in mind that you only have 5 minutes. If the patient is garrulous, ask direct questions but don't be rude. Practice in clinic is essential and repetition with consultants will bear fruit. If you are incoherent, erratic and unstructured and miss the basics it will be hard to pull back your mark, but remember each section is marked independently. A good history will arrive at a number of differential diagnoses, and will 'relax' the examiner who will likely listen to 20 candidates in a day, but a poor history will obviously put off the examiner.

Your examination needs to be slick and polished, too. Passing this exam means you are fit to practice as a day 1 Orthopaedic Consultant in a District General Hospital. If all else fails, remember Apley's – Look, Feel Move, Special tests. In the lower limb case always get the patient to stand, and comment from the shoulders, spine, pelvis, knees, ankles and feet both the positive and negative findings. Ask the patient to walk, and have a short speech ready about their gait. Practice this, from cadence, initial contact, the three rockers, and foot progression angle. If you are lucky enough to get a hip examination, perform a slick Trendelenberg's test (you get one go only to impress), don't forget to measure the limb lengths, and perform a Thomas' test. With knee examination start by rolling the leg in extension to rule out hip pain, check for a lack of hyperextension by comparing it to the normal side, look for an effusion, active then passive movements, and perform all the special tests (varus/valgus stressing, Lachman's, McMurray's, a gentle pivot test). With foot and ankle examination, check the shoes first for inlays, or comment if they are custom made, and abnormal wear. If they're new shoes state this, but that is what you are looking for. Finish with a quick distal neurovascular status, or at least mention you would perform it.

In an upper limb examination ensure adequate exposure, but bear in mind the patient's modesty! With the shoulder exam, stand behind and comment on the supra- and infrascapular fossae, deltoid wasting, regimental badge sensation, then gently palpate the SC and AC joints. Go through active then passive forward flexion, abduction, external and internal rotation. Check the power of the rotator cuff. In a hand examination, put the patient at ease by placing a pillow under their hands. Practise inspection and have a speech ready to go. Starting from the nails, comment on any positive and negative findings, through all the joints of the hand to the wrist, then ask the patient to turn their hand over and comment on the thenar eminences and any Dupytren's disease. Comment about the position of the fingers and thumb, before gently palpating all the joints in the hand. Then ask the patient to

[a] We always keep hearing from various sources that candidates should treat the clinicals as though you're in clinic with your consultant. The senior editor (PAB) doesn't buy this. The exam experience is nothing like clinic!

perform all the five grips, and functionally assess them with holding a pen, doing buttons up. Basically, do what you do every day in clinic.

You will probably be asked to perform a focused exam, dependent upon your history differential diagnoses. It is after all only 5 minutes. Clinical signs may be specifically asked for, but only demonstrate special tests if you are practised, and can describe how they work. You need to be aware of normal and abnormal findings in your examination. You should ensure that the examiner has noticed the important components of the clinical sign using verbal and expressive body language. If they haven't noticed you demonstrating these, you will not score the points! This is a bit like your driving test, you need to show that you can perform an accurate examination, but don't be put off if the examiner asks for a specific sign to be demonstrated.

The discussion usually begins with your differential diagnoses if not already given from the history. You will then either be asked for management – Don't forget to start with conservative, then surgical, or which investigations you would like. Start simple and work your way up, don't ask for an MRI first! Usually a radiograph is presented to you, and present it as you always have. The salient features will start a discussion about the surgical options and potential difficulties during surgery. As the discussion is only for 5 minutes, try and answer what is asked straightaway, i.e. listen to the question carefully. You will be scoring marks immediately if you do this, and failure to do so will be tedious for the examiner. When discussing treatment, do not offer or mention clever techniques which you have read recently but never seen or heard about. Instead stick to what is done in routine clinical practice. If you are going for an 8, mention recent randomised controlled trials or seminal papers but only if you are sure.

Don't be worried if you have a complex case, you will not be expected to demonstrate every single sign, but on the other hand if you are lucky enough to get something as simple as osteoarthritis, your history examination and discussion must be perfect. Ensure you are rested and get a good night's sleep. The most important thing is practise. Practise a concise, structured and focussed history. Practise your examination routines for all parts with colleagues, consultants and go on FRCS courses. They're worth the money as they replicate the examination, putting you under pressure, and making the real thing less of a shock. Offer treatments, as you would suggest to a patient in standard clinical practice. People who have fared badly in this section have had varying combinations of a shaky start, haphazard history, inability to reach a reasonable set of diagnosis at the end of history, poor examination technique or significant communication problems.

Top tips and tricks

Learn a technique so that if you get caught like a rabbit in headlights and your mind goes blank and you panic you will still have a system to get yourself out of jail.

If this even fails the default position is Apley's – 'Look, Feel, Move', and this can be used to get you started until you recover your composure and remember your examine sequence.

To the examiners it is obvious when observing a candidate how many times they have performed that test previously.

If you have written down an exam technique and practiced it a bit – It is probably not enough to get you through the exam. How you examine in clinic day in day out reflects more about how you will get on in the real exam. If you are skimpy, flimsy and inconsistent without a regular routine in the stress of the exam you may revert to this usual technique instead of your more polished practised approach that you have learnt in the previous 6 weeks.

A summary should be two, at maximum three, sentences, you don't want to keep repeating yourself again, and you will lose scoring opportunities. This is irritating to the examiners and they may pick you up on this if you start waffle on at the summary[b].

Clinical signs need to be demonstrated as asked. Listen to what the examiner asks and be guided by him/her.

Be able to differentiate and appreciate what is normal and what is abnormal.

You can predict what clinical patients will be present in the examination hall – You know there will be a rheumatoid hand or an old clubfoot. Define your topics by:

- Definitely
- Very likely
- Maybe
- Possibly
- Unlikely

You will not have too many painful patients in the examination hall or patients who will tire and fatigue easy. They may end up sitting out part of the day and this creates an organisation headache for the exam committee in finding replacement patients.

You need to bank/store up marks with your history and examination to compensate for any score 5s that you may pick up elsewhere.

You want to/need to score 6 on 96 occasions.

You enter the room with a 6 and if you do well your score increases whilst if you do badly your mark will go down. It's all up to your own performance how you will get on.

You need to maximize the marks you are in control of.

History

You are in control when taking the history, the examiner isn't going to interrupt and get involved if there are no uncertainties – you just need to keep practicing extracting a history from patients. You can easily score a 7 from this with some work. Multiply this for the four examiners and you have gone a long way forwards towards passing the FRCS (Tr & Orth) exam.

[b] Presenting the history all over again to the examiners!

Therefore, you need to get good at extracting a history from a patient[c]. Very occasionally it may be difficult to fully flow with the patient in getting that 7 with the history, but it really shouldn't fall below a 6.

Concentrate when taking the history. You may need to remember a complicated past history of orthopaedic operations from a patient. Do not forget these at the end when you are summarizing the history as this creates a very bad impression to the examiners and will lose you marks:

EXAMINER: The patient has told you what operations she has had performed so tell me them instead of saying she has had several operation around the elbow[d].

You are allowed to write down some notes as you go along but it is better if you can avoid this as it can also be a distraction. However, if you are going to forget something at the end when summing up such as drug history or previous operations then it is worth it (see above).

The examiners may ask you to take a focused history from the patient. What this means is that whilst the patient may have a complicated past medical history they want you to focus on the relevant details from this. They want candidates to succinctly get the information together[e].

Listen careful to the patient as they go through their history and appear empathic. Despite being very stressed, avoid at all costs being aggressive with the patient if they go off track with their story into irreverences. This will fail you this section and leave an overall bad impression with the examiners.

Clinical examination

If you keep practicing your examination technique you again can still become in control of this part of the exam and score highly. You will/should know what to do and, therefore, the examiners should not be able to catch you out by testing you on things you haven't come across. You need to think when examining a patient of what you are doing and why you are doing it. You can still be in control if you have done your homework[f]. Learn to examine well and completely. Take hints from the examiner:

EXAMINER: 'Are you sure that's what you really mean'? *(This is coded language to say that you are wrong.)* The examiner is checking that he really did hear what he thought he heard – He maybe didn't quite catch correctly what you said.

CANDIDATE: 'Absolutely'.

Make sure you take the hint from the examiner that you are wrong – 'I have been examining all day – it is sometimes not a nice day examining so listen carefully to my instructions and take my hints.'

Discussion

It is, therefore, only with the discussion that the examiners can start to ask you questions you can't anticipate or always predict so you are not in control of this situation. It is the only part of the examination you will/should be unsure of[g].

Intermediate list case

Here is a list of intermediate cases, which have been put up in the exams in the past. This is not an exhaustive list but the cases with a (*) next to them have been spotted frequently. Patients may have more than one region involvement and candidates have been asked to examine and present them simultaneously.

Hips

- Hip osteoarthritis (OA) (*)
- Avascular necrosis (AVN) of hip (*) (post-traumatic/post-steroid/alcohol-related)
- Sequel of developmental dysplasia of the hip (DDH)/ Perthes' in adult (*)
- Arthrodesed hip with no symptoms (*)
- Fracture neck of femur with failed internal fixation
- Total hip arthroplasty (THA), which was now becoming compromised by heterotopic ossification (HO), on a background of Paget's
- Mal-united slipped upper femoral epiphysis (SUFE)
- Mal-united femoral fracture
- Early painful total hip arthroplasty (<1 year)
- Paget's disease hip
- Loose painful total hip arthroplasty requiring revision
- Tuberculosis hip

Knees

- OA of knee (*)
- Medial OA knee in a younger patient (*)
- Valgus deformity in an arthritic knee (*)
- Rheumatoid arthritis (RA) affecting the knee (*)
- Post-traumatic valgus knee, multiple surgeries
- Previous traumatic knee dislocation with multiple surgical scars
- Painful total knee arthroplasty: Check hip, spine and vessels

[c] We think advice on history taking is OK and fairly reasonable.

[d] The candidate gave the impression he wasn't really listening to the patient. If you have difficulty remembering in the stress of the examination, write down some notes as you go along.

[e] The examiners may say that they want you to take a focused history from the patient accepting that you will not be able to get all the details from the history because of time constraints. This is coded language to tell you to get to the money fast!

[f] We disagree with advice on clinical examination, as no matter how much you practice it is still very difficult to be absolutely in control of everything that can happen.

[g] One could say if you learn everything for the discussion you will be in control as well, so making advice on discussion void, but we are becoming a bit too flippant.

Foot and ankle

- Clubfoot in adult (previous surgery)
- Ankle arthrodesis
- Kohlers' and congenital vertical talus

Spine

- Adolescent idiopathic scoliosis (*)
- Cervical myelopathy
- Spinal stenosis
- Lumbar disc prolapse

Upper limb

- Brachial plexus injury
- Rotator cuff arthropathy
- RA with shoulder, elbow (dislocated radial head), wrist involvement
- Instability (traumatic and atraumatic)
- Tumour prosthesis of the humerus
- Post-traumatic stiff elbow with compromised shoulder and hand function from coexisting rotator cuff disease and wrist fracture with EPL tendon rupture/transfer

Paediatric

- Neurofibromatoses with pseudoarthrosis of the tibia
- Congenital absence of forefoot and limb length discrepancy (LLD) in a 9-year–old
- Cerebral palsy
- Kippel–Feil and Sprengel shoulder

General

- Ankylosing spondylitis
- Diaphyseal aclasia
- Polyarticular RA
- Polio with LLD

Chapter 7

Shoulder clinical cases

Yusuf Michla and David Cloke

Introduction

Although there are always exceptions to the rule for the shoulder, think **age**:

- Young age – Instability, acromioclavicular (AC) joint dislocation, superior labrum from anterior to posterior (SLAP) tears, biceps tendinitis
- Middle age – Calcific tendonitis, adhesive capsulitis, impingement, rotator cuff tear
- Older age – Cuff tear arthropathy (combination of rotator cuff tear and arthritis), glenohumeral joint osteoarthritis, rotator cuff tear

The younger athletic person in their second decade of life more likely has instability, whereas the 60-year-old golfer with a painful shoulder more likely has rotator cuff disease.

Common cases include: long head of biceps (LHB) rupture with the resultant popeye sign, older patients with chronic cuff tears and a pseudoparalysed arm with lag signs.

Less common cases may include Sprengel's shoulder/Klippel–Feil syndrome, arthrogryposis, as well as brachial plexus lesions.

It is rare to need to carry out a comprehensive shoulder examination even for an intermediate case. Start with a general shoulder examination and then go on to test for rotator cuff strength. By that stage you may have picked up clues to suggest which area to concentrate on, but more likely the examiners are going to guide you into the part they want you to focus on. But, even if not instructed, as long as you follow a specific sequence as suggested below it is highly likely you will pick up the required signs.

With many of the short cases your examination technique will be diagnosis specific and you are more likely be instructed to examine the shoulder for instability or impingement.

- General examination. Look, feel, move. In palpation, feel the acromioclavicular (AC) joint, subacromial space, bicipital groove, trapezius, cervical spine
- Rotator cuff strength testing
- Impingement tests
- Instability tests
- Proximal biceps tests
- AC joint tests

There are a large number of specialized shoulder examination techniques described in the literature. This can be quite confusing as they are often described with slight variations in different textbooks! Stick to one or two tests for each specific condition and learn them well rather than try and remember the 16 different tests for a SLAP lesion! Concentrate on the common ones, rather than obscure ones that the examiners are unlikely to have heard of.

AC joint arthritis

The AC joint may be prominent due to osteophytes or a chronic AC joint dislocation. The patient will point with a finger to the joint if it is the cause of the pain. Pain is felt over the AC joint and may radiate into the trapezius area causing spasm.

History

Patients may complain of a sharp or catching pain, especially when working or lifting overhead as projecting spurs may cause typical rotator cuff symptoms. Patients may also complain of instability of the clavicle. Ask about a history of previous injury to the shoulder and the patient' s diagnosis, treatment and recovery.

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Clinical Examination

The most reliable signs that will guide you to AC joint pathology are: Point tenderness over AC joint, prominence of AC joint, and demonstrable instability of the clavicle on arm elevation.

Direct palpation will reveal well-localized tenderness and crepitus over the AC joint. Palpation may reveal asymmetry or irregularities within the joint.

Symptoms and clinical findings of significant AC joint arthritis are similar to rotator cuff pathology and often coexist. It is, therefore, important to test for active range of shoulder movement and rotator cuff muscle strength.

A number of provocative manoeuvres that load the AC joint have been described. However, these tests are not very specific for the joint and may overlap with other painful shoulder conditions such as rotator cuff tears or tendinitis, labral pathology or biceps pathology. Pain elicited by these manoeuvres should be localized to the AC joint rather than being non-specifically localized to the posterior or lateral shoulder. Suggest to the examiners a local diagnostic anaesthetic injection directly into the joint if any clinical uncertainty.

Terminal impingement pain: When full movement of the shoulder is possible a terminal impingement pain (pain above 120°) can be demonstrated on both active and passive movement.

Crossed arm adduction test or (Apley) Scarf test: The patient's arm is passively adducted across the body horizontally approximating the elbow to the contra-lateral shoulder. An augmented AC compression test can be performed if the examiner's thumb pushes the lateral end of the clavicle anteriorly.

Active compression test (O'Briens test): This test was developed for assessment of AC joint pathology but O'Brien noted in a series of pateints it was excellent for detecting labral pathology (sensitivity 100%, specificty 98.5%). The patient's affected arm is forward flexed to 90° while keeping the elbow fully extended. The arm is then adducted 10–15° across the body and maximally internally rotated so the thumb is pointing down. The patient should resist the examiners downward force to the arm. The test is repeated with the forearm supinated so that the thumb points upwards. A positive O'Brien's sign is present when pain is elicited with the first manoeuvre and reduced or eliminated by the second manoeuvre. Pain localized to the AC joint or 'on top' of the shoulder suggests an AC joint abnormality, whilst pain or painful clicking 'inside the joint' suggests a labral disorder. It is important to ask the patient if the pain is felt on the outside over AC joint or deep inside the shoulder; patients usually will reliably distinguish between the two.

Below are some additional small print pain exacerbating examination manoeuvres that imply AC joint pathology. It is unlikely you will be asked to demonstrate them, but sometimes a score 8 candidate will speed through a short case such that the examiners have to find additional material to test on so as to use the full 5 minutes allocated.

Dugas' test: The patient is seated or standing and touches the contra-lateral shoulder with the hand of the 90° flexed arm of the affected side. If painful the test suggests AC joint pathology.

Paxinos' test: Shear type test for the AC joint. The examiner's hand rests over the top of the shoulder with the thumb under the posterolateral aspect of the acromion and the index and middle fingers resting on top of the lateral third of the clavicle. The thumb applies an anterior and superior pressure to the acromion and the fingers push the clavicle inferiorally. A positive test occurs with an increase in AC joint pain.

Acromioclavicular resisted extension test: This starts with the upper limb elevated 90° in the sagittal plane, the elbow flexed to 90° and internal rotation to 90°. With the examiners hand fixed in space against the posterior elbow, the patient extends the shoulder in the transverse plane, meeting the examiners resistance. The test is positive if pain occurs in the AC joint

Buchberger's test: This combines an inferiorly directed force to the lateral clavicle with passive forward elevation of a slightly adducted and externally rotated upper limb. The test is positive if pain occurs in or near the AC joint

Investigations

AP radiograph shoulder (marginal osteophyte formation, subchondral bone loss, cystic resorption of the distal clavicle, generalized osteopenia distal clavicle)**:** Osteolysis of the lateral end of the clavicle is seen in weightlifters and will have clinical findings similar to AC joint arthritis.

MRI shoulder (look for rotator cuff and other subacromial pathology): MRI may also demonstrate impingement of the superior cuff by an inferior AC joint osteophyte, suggesting that removal of this should be included in decompression surgery.

Management

Conservative: Anti-inflammatory medication, modification of activities. Cortisone injection (usually x-ray or ultrasound guided) can be very effective in the short to medium term, and is also helpful in confirming the working diagnosis.

Surgery: For persistent and significant symptoms that have failed conservative management consider AC joint excision

Open (direct superior approach) or arthroscopic (isolated or combined with subacromial decompression): There is a split between surgeons using the open and arthroscopic techniques. With open techniques, excessive resection or failure to repair the capsule may lead to instability. With the arthroscopic technique inadequate resection (especially the superior and posterior part of the joint) is more common.

Subacromial impingement

The clinical picture can be confusing if coexistent shoulder pathology, such as a rotator cuff tear is present. In younger patients, think about possible underlying causes such as instability. Current aetiological theories include the classical anatomical impingement (Neer's), and more recent functional theories, i.e. cuff weakness causing dynamic impingement.

History

- Onset, duration, location and quality of pain
- Weakness, loss of motion (especially elevation), inability to sleep on the affected side, night pain, catching, crepitus
- Interference with activities of daily living (ADL)

Classic history is of shoulder pain felt over the deltoid, on the lateral part of the arm, exacerbated with overhead activities.

Clinical examination

In pure impingement there is minimal rotator cuff wasting; marked wasting should alert to the possibility of cuff tear. The acromion is palpated along its posterior, lateral and anterior margins. In impingement syndrome there is often tenderness at the anterolateral corner of the acromion. There may be mild tenderness over the greater tuberosity, which is best demonstrated by extending and internally rotating the arm. The greater tuberosity is brought anteriorly from under the acromion. It and the inserting supraspinatus tendon can be palpated. Tenderness and crepitus may be present with tears or tendonitis.

From behind check active abduction and forward flexion and assess scapula rhythm. Ideally there will be a mirror in front of the patient so any pain on active movement can be seen[a]. On elevation the initial 60° is glenohumeral, 60–120° is both glenohumeral joint and scapulothoracic, and above 120° is scapulothoracic. To check for capsular tightness, stabilise the scapula with a hand on the acromion and abduct then forward flex the arm

With impingement, there is classically a painful arc of active elevation between 70° and 120°, but this may be more or less in some patients. There may be an alteration of scapular rhythm. There may be soft crepitus when the arm passes through this arc. The pain is usually localized to the anterior region of the shoulder but may often radiate down to the deltoid insertion. There may also be pain at the anterior edge of the acromion on forced elevation

Formally test the power of the rotator cuff muscles: You will see either normal power or a minor weakness on resisted movements of the rotator cuff. This perceived loss of power may be secondary to pain inhibition, rather than a true cuff tendon tear. If a coexistent large cuff tear is present, significant weakness of abduction and external rotation can be demonstrated.

Do not forget to mention examination of the cervical spine at some point during your shoulder examination.

Discussion

- Definition of impingement
- Difference between Neer's sign and test
- Impingement tests

Definition of impingement

Impingement is pain emanating from the subacromial space and is caused by either narrowing owing to a subacromial bony spur and thickening of the coracoacromial ligament or by a thickened bursa and tendonitis. However, current thinking includes the phenomenon of impingement caused by poor cuff control in the absence of an anatomical lesion. Poor cuff control may lead to superior sublaxation of the humeral head, with resultant impingement. Consider poor cuff control, or os acromiale in a young patient presenting with impingement symptoms.

Difference between Neer's sign and Neer's test

Neer's sign: Reproduction of the patient's symptomatic pain with the arm in the scapular plane, internally rotated (thumb pointing down) and then taken through abduction to demonstrate painful arc. Repeating abduction but with arm in external rotation will improve pain.

Neer's test: Local anaesthetic injection into the subacromial space eliminates pain when testing for Neer's sign.

Impingement tests

There are three classic tests for impingement that require a confident, well-rehearsed slick technique and three obscure impingement tests that you probably will never be asked about unless you are gold medal candidate.

Neer's sign

- As above

Hawkins' impingement reinforcement test[b]

- Passive internal rotation in 90° flexion
- External rotation: Unlimited
- Internal rotation: Limited, exhibits painful endpoint
- Need to have full passive movement of the shoulder to be able to demonstrate impingement

A positive test occurs with shoulder pain and apprehension. The test jams the supraspinatous against the anterior portion of the coracoacromial ligament

Abduction test

- Classically painful between 70° and 120° – A painful arc as the rotator cuff is placed under maximum tension

Yocum's test

This test was described in 1983 to selectively test the function of supraspinatous tendon and is very similar to the Jobe supraspinatous test. It is performed with abduction of the patient's arm to 90°, forward flexion to 30° and maximal internal rotation. In this position, the examiner resists active shoulder abduction and reproduction of pain and/or weakness in this position suggests the supraspinatous tendon as the site of injury and implies impingement tendonitis.

[a] Whilst clinic may be OK, there is virtually nil chance of having a mirror available in the exam.

[b] Be careful with false positives. Analysis of this test in patients with AC joint OA reported that 90% had a positive Hawkin's result.

Internal rotation resistance stress test

This test differentiates between intra-articular (internal impingement) and classic outlet impingement. Internal impingement is commonly seen in overhead athletes with subtle glenohumeral instability. These patients have impingement of the posterior rotator cuff along the posterior superior labrum in the late cocking phase of the throwing motion and pain in the region of the infraspinatous insertion with abduction and maximal external rotation. If the patient has with a positive impingement sign and good strength in external rotation, this test is positive and predictive of internal impingement. A negative test (more weakness in external rotation) is suggestive of classic outlet impingement (external impingement).

Subcoracoid impingement test

The test produces compression of the rotator cuff between the humeral head and the coracoid. Although uncommon, the condition has been described in patients with a long or laterally placed coracoid process and should be considered in patients with negative classic impingement signs that describe anterior/lateral pain with overhead activities and have similar complaints to classic outlet impingement. These patients have impingement of the coracoid process on the proximal humerus with forward flexion and internal rotation of the arm.

Management

Conservative

Physiotherapy to improve cuff strength and improve shoulder mechanics

Steroid injection therapy (but be aware of current controversies over steroid effects on the cuff – There is some basic science evidence from rats of a detrimental effect of repeated steroids on cuff tendon healing[1])

Operative

Open or arthroscopic decompression – Surgery is generally felt to be indicated following failed conservative management. It is not uncommon for steroid injections to give a temporary relief; such temporary relief confirms the subacromial origin of symptoms, and if short-lived, points towards the need for more definite surgical treatment. Most surgeons use an arthroscopic approach to preserve the deltoid attachment. Controversy surrounds the proposed mechanism of action of subacromial decompression – physical removal of the coracoacromial ligament and acromial spur or removal of pain fibres in the bursa are sighted as potential mechanisms. The CSAW trial (Can Shoulder Arthroscopy Work[c]) is currently randomizing impingement patients in the UK to structured physiotherapy, conventional arthroscopic decompression, or sham arthroscopic surgery.

[c] www.situ.ox.ac.uk/surgical-trials/csaw

Examination corner

To be asked straight up by the examiners to 'examine this gentleman for impingement' can be off putting. Candidates normally expect to be greeted with the instruction to 'examine this patient's shoulder'. This upsets our normal routine as we anticipate starting with inspection looking for wasting, scars, deformity etc and not with specific tests for impingement such as the Hawkin's test.

It is important to note that the above tests can give similar findings in different subacromial pathologies (subacromial bursitis, isolated impingement, impingement along with a small rotator cuff tear, calcific tendionosis). Small-/medium-size rotator cuff tears may not produce any clinically demonstratable weakness, whereas substantial subacromial pain may give weakness like findings when testing the rotator cuff.

Rotator cuff tears

History

Chronic large full thickness tears usually occur in middle-aged or elderly patients. Symptoms include chronic aching and shoulder weakness that worsens with abduction and external rotation. Patients may complain of catching or locking. Symptoms are noticeably worse a night with significant sleep disturbance.

Clinical Examination

Look

Look at the shoulder posture, notably for the presence of any shoulder asymmetry, alterations in position and muscle wasting. Prominence of the AC joint indicates possible AC joint osteoarthritis. Look at shoulder movements whilst the patient is undressing. If the injury is acute there is unlikely to be significant muscle wasting unless there is a pre-existing shoulder problem. In the exam you are more likely to be given a chronic (possibly massive) tear in which there will be obvious muscle wasting.

Inspect the shoulder girdle, particularly the deltoid muscle, the supraspinatus and infraspinatus fossa. A combined supraspinatus/infraspinatus tear leads to prominence of the scapular spine and indicates a large tear. Look for anterosuperior escape, where the humeral head subluxes with attempted elevation, and 'pseudo-paralysis', when the attempted elevation results in a shrug because of isolated deltoid action.

Feel

Compress the lateral end of the clavicle to identify symptomatic arthritic change in the AC joint. There may be coexistent subacromial impingement caused in part by AC joint osteophytes.

In thin individuals defects in the cuff may be palpated. This is controversial as there are some shoulder surgeons who believe that the presence and extent of rotator cuff tears cannot reliably be elicited by palpation.

Move

There may be loss of active elevation and a disparity between active and passive ranges of movement. Significant loss of passive shoulder movement is uncommon even in the presence of a massive rotator cuff tear. You may, however, find abnormal scapular rhythm, especially on lowering the arm, owing to eccentric contracture. Motion will be segmented and irregular. A large chronic rotator cuff tear will lead to a severe limitation of active abduction mainly achieved by scapulothoracic rather than by glenohumeral motion

It is important to recognise shoulder stiffness in the presence of a rotator cuff tear. Strength is more difficult to evaluate when there is significant stiffness. In addition, the cause of stiffness (adhesive capsulitis, capsular contracture or glenohumeral arthritis) may be the cause of the patient's symptoms. Which tendon is torn? Assess rotator cuff strength and integrity.

Rotator cuff musculature testing

Patients with rotator cuff tear arthropathy often demonstrate severe pain and weakness with attempted strength testing of the rotator cuff musculature.

The supraspinatus can be assessed by applying a resisted downward pressure with the shoulder abducted 30° in the plane of the scapula, the elbow in extension, and the arm in maximal internal rotation.

The infraspinatus can be assessed by testing external rotation strength with the arm in 0° of abduction and the elbow flexed to 90°.

The lift-off test, can be used to assess subscapularis strength. With the arm in maximal internal rotation and the dorsum of the hand resting on the mid-lumbar region, resisted movement away from the body is assessed.

Teres minor can be assessed by testing resisted external rotation of the arm with the shoulder in 90° of abduction in the plane of the scapula and the elbow in 90° of flexion.

Supraspinatus

Two further tests of supraspinatous function can be performed:

1. **Empty can test**[d]**:** The shoulder is elevated to 90° in the scapula plane and taken into full internal rotation with the forearm in pronation so that the thumb is pointing to the floor. Downward pressure is applied to the arm while the patient maintains this position. Pain without weakness is suggestive of supraspinatous tendinopathy while painful weakness suggests a partial or complete supraspinatous tear
2. **Full can test**[e]**.** This involves repeating the test with the arm in 45° of external rotation so that the thumb points upwards which is less painful but has equal diagnostic accuracy

Subscapularis

Patients with subscapularis tears have internal rotation weakness, variable excessive passive external rotation (as compared to the opposite side) and a positive lift off test or belly-press maneuver. Three commonly used tests are described.

1. **Lift-off test**[f]**:** Place the patient's hand behind their back and ask them to push backwards against the examiner's hand. This tests the strength of further internal rotation and is the most reliable test for subscapularis. This test is difficult to perform when there is pain or limited shoulder motion that prevents positioning of the arm and hand behind the back (which is often encountered in this group of patients). If the patient cannot place their hand behind the back go on to the belly press test

   An internal rotation lag sign is also described whereby the examiner removes their hand and the patient is asked to maintain their hand away from the back. The lag sign is positive is the patient can not maintain the hand in this position and it falls back onto the lumbar spine
2. **Belly press test:** Both hands are placed on the abdomen (belly) with flat wrists. The elbows should remain anterior to the trunk while the patient pushes posteriorly against the belly (to ensure that belly push is not compensated by active wrist flexion giving a false negative result). Patients with subscapularis weakness demonstrate a dropped elbow because they use shoulder extension to compensate for weak internal rotation. This is probably the most reliable and reproducible in clinical practice
3. **Bear hug test:** This involves the patient placing their hand on the opposite shoulder while elevating their elbow. The examiner attempts to elevate the patients hand off the shoulder and a positive test occurs when this is easierly achieved

Infraspinatus/teres minor

There will be a loss of external rotation (ER), positive ER lag sign, drop sign and a positive Hornblower's sign.

Hornblower's sign: This demonstrates the difficulty in raising the hand to the mouth in the absence of external rotation of the shoulder. A positive test occurs if the patient is unable to do so without fully abducting the shoulder. This allows the weak arm to fall into internal rotation, so that the arm assumes a position like a hornblower

[d] This is also known as Jobe's test and was originally described by Jobe and Moynes.

[e] Kelly's modification of the Jobe and Moynes test

[f] This test is also known as Gerber's test and Gerber's lift off test

Lag signs

Lag signs represent a mismatch between active and passive ranges of motion, are indicative of substantial rotator cuff weakness (and, hence, massive rotator cuff tears) and may be more sensitive and specific than standard muscle strength testing. Several lag sign tests have been described to assess the integrity of the rotator cuff:

1. **External rotation lag sign (infraspinatus)**: With the shoulder held at 20° of elevation (in the scapula plane) and the elbow passively flexed 90°, the arm is placed in full external rotation, and then the patient is asked to actively maintain this position. If the arm starts to drift into internal rotation when the examiner releases the wrist, the test is positive
2. **Drop arm test (supraspinatous)**[g,h]**:** The arm is held in 90° of abduction, and the patient is asked to actively maintain this position. The test is positive when weakness or pain causes them to drop the arm to the side. Be careful to warn the patient, and don't suddenly let go with a resultant painful drop of the patient's arm. Consider letting go but moving your arm slightly down and catching the patient's arm if it was to give way
3. **Internal rotation lag sign (subscapularis):** Stand behind the patient. Flex the elbow to 90°, hold the shoulder at 20° elevation and 20° extension. The patient is unable to hold their hand away from their lumbar spine in near full internal rotation

Despite severe rotator cuff deficiency, some patients have good elevation strength owing to compensatory deltoid strength. Therefore, some patients with chronic massive rotator cuff tears have full active shoulder elevation. Park et al.[2] found that three positive tests made the diagnosis of full thickness rotator cuff tear likely: Positive painful arc sign, drop arm sign and weakness of external rotation.

Impingement and ACJ osteoarthritis often coexist with rotator cuff pathology, so suggest to the examiners that you would also like to test for these entities as part of your complete shoulder examination.

Differential diagnosis of a rotator cuff tear includes adhesive capsulitis, calcific tendinitis, superior labral tears, bicepital tedinopathy, glenohumeral and AC joint OA.

Investigations

- Radiographs – Three standard projections: True anteroposterior, supraspinatus outlet, axillary view
- Ultrasound – Cheap and non-invasive but operator-dependent
- MRI – Most sensitive and allows assessment of muscle quality (fatty infiltration) in large/massive full thickness tears

[g] There are a number of subtle variations of this test.
[h] Infraspinatous weakness can also result in a positive test.

Management

Conservative

- Small tears or irreparable tears in the elderly
- Physiotherapy (strengthening exercises for the rotator cuff, anterior deltoid and shoulder girdle). Steroid injections, but be aware of some current thinking on the effect of steroid on the cuff[1]

Surgery

- Open, mini open or arthroscopic repair, often combined with biceps tenotomy or tenodesis
  - There is no good evidence for one technique. Proponents of the arthroscopic technique cite the preservation of the deltoid as a major factor. Mini open surgery minimizes deltoid injury. The results of the UKUFF trial (United Kingdom rotator cuff study) that randomised rotator cuff tear patients into open or arthroscopic repair, are awaited
  - Surgery is generally indicated for failed conservative management, or truly acute cuff tears
  - Complications include postoperative adhesive capsulitis and re-tear, with a rate increasing with tear size, although even with re-tear outcomes appear improved compared to non-operative treatment
- Arthroplasty for rotator cuff arthropathy
  - Standard total shoulder arthroplasty contraindicated owing to abnormal glenoid loading and loosening
  - Hemiarthroplasty (stemmed or resurfacing), or reverse geometry prostheses – Be aware of the principles, i.e. medialisation and distalisation of the centre of rotation with reverse arthroplasty, along with a degree of constraint, allowing deltoid to act as the prime mover in abduction

Postoperative management includes physiotherapy.

Examination corner

Short case 1: Elderly man with cuff arthropathy

GP letter

'This 74-year-old man has been referred to the orthopaedic clinic with a 10-year history of shoulder pain and weakness.'

Muscle wasting within the supraspinatous and infraspinatous fossa is common. Supraspinatous atrophy is more difficult to detect as it is underneath trapezius. Sometimes a palpable defect at the supraspinatous insertion at the anterolateral aspect of the shoulder. Swelling due to subdeltoid synovial fluid may be present. Occasionally the humeral head can be palpated in the anterior-superior aspect of the shoulder in its subluxed position. Active forward flexion is limited to 50°. Passive forward flexion is 125° with external and internal rotation actively and passively limited to10°. He has 4/5 MRC supraspinatous and external rotation strength.'

- Describe the radiograph (superior head migration combined with severe degenerative changes of the glenohumeral joint) – Chronic subluxation humeral head often causes 'acetabularisation' as secondary erosions of the glenoid, acromial undersurface and clavicle occur to accommodate the humeral head
- Had MRI – Describe (note absent of cuff . . .)
- Discussion of management options – Treatment of patients with a massive irreparable rotator cuff tear associated with severe glenohumeral degeneration and humeral head collapse presents a difficult clinical challenge
- Management – Reverse vs limited goals hemi depending on patient expectations and objectives, and adequacy of glenoid for arthroplasty

Short case 2: Massive rotator cuff tear in an elderly man

- Ask a short history – Age, dominance, pain, stiffness and weakness
- Examination of cuff power, lag signs, pseudoparalysis (due to pain inhibition-make a dramatic improvement with cortisone injection)
- External rotation weakness is characteristic of chronic massive tears. Elevation weakness is a less consistent finding. Some patients have sufficient deltoid strength to mask the absence of supraspinatous strength
- Differential diagnosis for chronic massive rotator cuff tear – Cervical spondylosis, stenosis and radiculopathy can cause shoulder pain that mimics the pain of rotator cuff pathology. Shoulder girdle weakness can be the result of brachial plexus disorders (Parsonage–Turner syndrome, brachial neuritis) or suprascapular neuropathy. Septic arthritis
- Factors in surgical decision-making – Expectations, stiffness, cuff retraction and fatty atrophy on MRI or US

Short case 3: Chronic rotator cuff tear shoulder in a middle-aged woman (approx. 45 years) sitting in chair

EXAMINER: This lady is complaining of right shoulder pain. Just examine her shoulder and tell us what you're doing.

CANDIDATE: Is your shoulder painful?

PATIENT: Yes.

CANDIDATE: I'll try not to cause you any pain. Please let me know if it's sore and I will stop.

On inspection of the shoulder from the front there are no obvious scars, muscle wasting or asymmetry. The shoulders are of symmetrical height. There are no other obvious features of note. From behind there is some wasting of supraspinatus and infraspinatus (mild but definite wasting).

EXAMINER: Whereabouts is this wasting?

CANDIDATE: There is wasting over the right supraspinatus and infraspinatus muscles compared to the left side. *(Demonstrate the wasting. An important physical sign to pick up if present.)*

Where is your shoulder painful?

PATIENT: Over here (points to greater tuberosity).

CANDIDATE: I am palpating the shoulder from a medial to lateral direction feeling for any areas of tenderness or crepitus. There is no pain over the sternoclavicular joint, clavicle or AC joint. She is sore over the supraspinatus tendon and also the greater tuberosity, to which she pointed previously. The anterior and posterior joint line is non-tender. I would now like to test this lady's range of movement at the shoulder. Can you swing your arms out by your side and then upward into the air (abduction)? The patient only has 80° of active abduction and it is painful with loss of normal scapulothoracic rhythm. Passively there is very little extra movement and this provokes more pain. Can you swing your arms forward now (flexion)?

EXAMINER: You need to stabilise the scapula when there is a discrepancy between active and passive movements of the shoulder.

CANDIDATE: I usually test active and passive movements of the shoulder first and then go back and stabilise the scapula if there is limitation of movement in order to differentiate between scapulothoracic and glenohumeral movement. There is a restricted range of active forward flexion to 90° that I can't improve passively, and most of this movement is scapulothoracic. *(Hand stabilising the scapula when the movement was performed)* Can you swing your arms backwards?

Again, active extension is limited to 20° and painful.

Likewise, external rotation is painful and is only about 30° compared to the opposite side. I'd like to go on to test her rotator cuff muscles for power, testing supraspinatus, infraspinatus, teres minor and subscapularis in both the normal and the abnormal shoulder. She has a marked loss of power in her right rotator cuff muscles compared to the left side.

EXAMINER: What do you think the diagnosis is?

CANDIDATE: I think she may have an impingement syndrome.

(I have no idea why I said this, as she certainly did not demonstrate any impingement signs and I did not specifically test for them. I chose to ignore the fact that we had alluded to the diagnosis of a rotator cuff tear during the examination. I just felt that she was the wrong age for a rotator cuff tear and that I had not clinically demonstrated a significant difference between active and passive movements of the shoulder typical of a rotator cuff tear.)

EXAMINER: You demonstrated a loss of power in her rotator cuff muscles. These findings would suggest a rotator cuff tear. What investigations would you perform to confirm the diagnosis?

CANDIDATE: I would order some x-rays of her shoulder.

EXAMINER: What would you expect to find?

CANDIDATE: There would be degenerative changes in the acromioclavicular joint, a decreased coracohumoral interval, a break in Shenton's line with superior migration of the humeral head and possibly cystic changes in the greater tuberosity.

EXAMINER: What is the investigation of choice?

CANDIDATE: An MRI.

EXAMINER: OK. How are you going to operate on this lady's cuff tear?

CANDIDATE: We need much more information than this. I need to know how large the tear is, whether it is full thickness or partial thickness and the state of the surrounding muscles.

EXAMINER: It is 4 cm and full thickness.

CANDIDATE: Then I would refer her on to an experienced shoulder surgeon who repairs these cuff tears on a regular basis.

(*With this we moved onto the next case.*)

The examiner was trying to speed up the pace of the examination. It is important to take the hint and get on with it as swiftly as possible but not at the expense of appearing rushed or hurried. Try to remain professional and composed at all times during your clinicals. Defend yourself if you are being challenged but be sensible about it and try not to be too aggressive in your reply. To this day I am still unsure exactly what was wrong with this patient's shoulder. On clinical examination she did not present with the classic findings of a rotator cuff tear and we are still not entirely convinced that she had a rotator cuff tear. We went down the path of assuming it was a tear and discussed further management of it.

Short case: Examiner: "You can ask two questions only"

'Weak, painful shoulder with numbness, neck scar from lump excision. Asked to examine shoulder movements, then asked diagnosis. Patient had full abduction, restricted internal and external rotation. Said likely neurological lesion due to scar and numbness but difficult as no chance to do any further examination. Then quiz on rotator cuff. I found out later that patient had a C6 root problem and previous excision of neurilemmoma – Didn't feel I was given much chance to examine.'

Rotator cuff tear

Obvious massive cuff tear and biceps rupture. Test for rotator cuff power and lag signs. Discussion of investigation and management. Any new surgical techniques for massive cuff tears (Inspace Balloon procedure, Platelet rich plasma).

Frozen shoulder

GP's letter handed to the candidate to read before the start of the intermediate case: '*A 53-year-old woman who presents with a 6-month history progressive shoulder pain without any specific predisposing injury. She complains only of shoulder pain with some loss of motion. She has occasionally very painful episodes while trying to perform certain activities as at the limits of her effective shoulder motion.*'

History

Is the pain intermittent, constant, only with use or predominantly at night? The time course over which the stiffness developed should be explored. The magnitude of the functional disability related to the stiffness should be noted. Any history of previous shoulder trauma. Any previous treatments and outcome. Typical history is that of severe, acute onset pain that lasts for a few weeks (pain predominant phase) and then improves leaving substantial limitation of movement (stiffness predominant phase). With time movement begins to improve (thawing phase), with resolution of symptoms over 1–2 year period in many.

Clinical examination

On examination there is a normal shoulder contour, except perhaps some slight deltoid wasting. Tenderness may be present around the glenohumeral joint. There is no swelling or warmth around the shoulder.

'The patient demonstrates marked restriction of both active and passive movements of the shoulder when compared to the opposite normal side. In particular her active forward flexion is 100° and passive forward flexion is 110°. A firm endpoint to motion with pain was present. Her passive external rotation is 10° and active external rotation is 0°. Examine the true glenohumeral range with a hand stabilising the scapula. It can be difficult to test power because of the restriction in shoulder movement.'

Adhesive capsulitis must be thought of as a diagnosis of exclusion. Differential diagnosis includes large rotator cuff tears, subacromial impingement, posterior shoulder dislocation, and degenerative joint disease

There is an important association with diabetes. Frozen shoulder refers to idiopathic stiffness, but stiffness may also be post-surgical or post-traumatic.

Investigations

Radiographs are normal and the diagnosis is usually clinical. MRI arthrography can be used to assess capsular thickening and will show a reduction in capsular volume. Suggest a fasting blood glucose level in primary care to rule out frozen shoulder as first presentation of diabetes. The diagnosis is based on clinical demonstration of global loss of active and passive movements, with normal radiographs (i.e. absence of glenohumeral arthritis, proximal humeral fracture mal-union which may cause mechanical loss of motion)

Management

Conservative: Physiotherapy (in the form of stretching exercises), glenohumeral injections in early stages of disease. Some advocate hydrodilatation as a useful treatment modality.

Operative: Surgery aims to hasten recovery (as the natural history per se is in many that of spontaneous resolution). Surgery may be in the form of manipulation under anaesthetic (MUA), arthroscopic or open release. Most surgeons advocate intervention in failed conservative management and no range of movement (ROM) improvement over 3 months. No evidence for MUA vs release. Advocates of arthroscopic release cite the controlled nature of specifically addressing the rotator interval pathology. The UK FroST study (UK Frozen Shoulder

trial) is currently randomizing patients to structured physiotherapy, MUA or arthroscopic arthrolysis.

Glenohumoral osteoarthritis

History

- Pain, stiffness, loss of motion
- Objective assessment of the patient's functional impairment
  - Difficulty getting dressed
  - Brushing teeth
  - Reaching the top or the back of the head
  - Reaching the opposite axilla
  - Washing the perineum
  - Washing the face
  - Combing the hair
  - Writing or turning a key

'In summary, Mrs Smith is a 73-year-old retired right-handed headmistress who presents with several years history of intermittent shoulder pain. In the past year she has experienced increasing pain and stiffness of this shoulder. She denies any specific history of trauma. She has difficulties with activities of daily living particularly brushing her teeth, washing her face, etc. My provisional/working diagnosis is of glenohumeral osteoarthritis and I would like to examine the shoulder to confirm this.'

Examination

On inspection from the front there may be muscle wasting. There are no obvious scars but there is loss of the normal shoulder contour. The anterior shoulder may appear swollen due to an effusion.

Asymmetry of the shoulder may be present owing to gross muscle wasting and distortion of the bony anatomy. The displacement of the humeral head and severe erosion of the head or of the glenoid can markedly distort the contour of the shoulder.

'The posterior joint line is tender to palpation. There is gross painful restriction of shoulder movement with crepitus on glenohumeral joint movement.'

There will be a slight difference between active and passive movements of the shoulder because of pain, but this difference is never as distinct as in the case of a chronic rotator cuff tear.

With progression of the disease shoulder movement becomes restricted to scapulothoracic movement, which does not allow much rotation. Therefore, external and internal rotation are limited the most.

Power could be difficult to test smoothly because of the marked restriction of shoulder movement and pain (do not hurt the patient). The strength of the rotators and of the deltoid within the limited range may be surprisingly good. Pain rather than muscle wasting may cause some of the weakness.

Gross restriction of shoulder motion (active but also passive) in an elderly patient (>60 years) should be considered as glenohumeral arthritis until proven otherwise.

Management options

- Physiotherapy, intra-articular steroid injections
- Arthroscopic debridement, subacromial decompression, biceps tenotomy/tenodesis
- 'Biological resurfacing' (e.g. meniscal allograft) in young patients: Small series only in the literature
- Resurfacing or stemmed hemiarthroplasty: resurfacing supposedly restores anatomy by placing the prosthesis in the native anatomical location, and is bone preserving, but requires a reasonably well-preserved head
- Total shoulder arthroplasty: Concerns regarding glenoid longevity
- Reverse polarity arthroplasty for arthrosis in the presence of cuff insufficiency or as a salvage for cuff failure with a pre-existing total shoulder arthroplasty
- Resection arthroplasty: Salvage for failed arthroplasty
- Arthrodesis: Again, salvage procedure with better functional results than resection

In general, there is evidence for improved pain relief with total shoulder arthroplasty compared to hemiarthroplasty, but this is more technically challenging.

Shoulder instability

History

Did the shoulder come out because of a significant injury, or due to minor injury?
What position was the arm in when it came out?
Did the shoulder slide out of joint or did it pop out?
Did you have to go to hospital to have the shoulder put back in?
Did you feel any numbness or tingling in the arm or hand?
How frequent does the shoulder feel it wants to come out?
Is there a sense of looseness in the shoulder in between?
Non-descript level of discomfort and diffuse pain around the shoulder, discomfort is poorly localized and may be more scapular in location. This can be associated with paresthesias down the arm.
Does the sense of instability occur with the arm only in certain positions or is it present regardless of arm placement or position?
What activities and arm position provoke the symptoms?

Pain is a more common symptom with shoulder instability secondary to ligamentous laxity (AMBRI) and apprehension is more common with multidirectional instability

The classic patient with traumatic instability is a male athlete with an identifiable traumatic event in the course of violent sporting activity. Conversely, the typical patient with

multidirectional instability describes non-descript shoulder pain that also involves the scapula and provokes paresthesias down the arm occurring in the absence of a traumatic event.

Clinical examination

Be aware of the spectrum of instability, from traumatic lesions to overuse injuries and more generalized capsular and muscle patterning disorders.

The shoulder should be palpated for any areas of tenderness. Patients with anterior subluxation often have tenderness over the posterior capsule, whereas in multidirectional instability tenderness may be present along the medial angle of the scapula. Shoulder dislocation may have injured the axillary nerve, so it is important to evaluate for deltoid atrophy and weakness, and sensory deficit.

Both active and passive range of shoulder movement should be tested noting any dyskinesia and whether accessory muscles are activated with range of motion testing. Look particularly for scapular dysrhythmia and winging, as well as any evidence of overactivity of pectoralis major or lattisimus dorsi.

Look for signs of generalized hyperlaxity (Beighton score) and the inferior sulcus sign. In a patient with gross instability, a depression may be seen inferior to the anterior aspect of the acromium when the arms of a sitting patient are positioned along the side of the body. It is usually necessary to apply a traction force along the longitudinal axis of the humerus by pulling the humerus in an inferior direction. A measurement of >2 cm or an asymmetrical symptomatic sulcus sign is positive for inferior instability.

Anterior draw test – Grade and compare to other side. Perform apprehension and Jobe's relocation test, load and shift test. Test for posterior shoulder instability (posterior drawer test, posterior apprehension test, jerk test).

The patient is most likely to have normal musculature or, at worst, minimal shoulder wasting. Examine external rotation, internal rotation and abduction strength to rule out rotator cuff weakness. Chronic subscapularis, or supraspinatus tears may lead to anterior instability.

Investigations

- Plain x-rays – Bony Bankart , Hill–Sachs, previous anchors
- MRI arthrogram – 'gold standard' – Evidence of labral avulsion especially on abduction external rotation sequence (ABER)
- CT/CT arthrogram – Useful with previous metallic anchors on to look for glenoid bone loss (cause of failure of soft-tissue stabilisation, and indication for bony procedure)

Management

Conservative: Physiotherapy for scapular and glenohumeral control, especially in 'atraumatic' instability

Operative: Open and arthroscopic Bankart's repair, bony procedures for glenoid bone loss or revision. Presence of glenoid bone loss, especially with Hill–Sachs lesion, is indication for coracoid transfer (Latarjet procedure) or iliac crest bone block procedures.

Examination corner

Possibly a intermediate case, much more likely a short case. Young male, with normal musculature, and normal movement and power of the shoulder. Diagnosis rests on demonstrating instability tests. You may be asked simply to demonstrate instability tests rather than examine the whole shoulder.[i]

EXAMINER: What are the signs of instability and how do you elicit them? Do you know any instability tests?

Short case 1: A short case of voluntary posterior dislocation of the shoulder

The patient was able to spontaneously dislocate the humeral head and then reduce it painlessly. No evidence of generalized joint laxity. The dislocation occurred with abduction and extension associated with clicking.No history of shoulder trauma. No evidence of connective tissue disorder such as Ehlers–Danlos syndrome. The examiner asked whether I would want to perform surgery on this patient. I said no i would treat him conservatively as surgery would probably have a high failure rate. The examiners implied the correct answer with their question

Short case 2: Recurrent anterior dislocation of the shoulder

- Examination of the shoulder
- Apprehension test
- Arthroscopy portals and the role of arthroscopy
- Bankart and Hill–Sachs lesions
- Bankart repair

Rupture of the long head of biceps (proximal)

A spot diagnosis in the short cases[j]. Usually male, elderly. A bulbous mass is seen in the middle to distal anterior arm ('Popeye sign') that typically appears when the elbow is flexed.

'There is a large bulge 4 cm by 3 cm in the anterior flexor compartment of the arm suggestive of long head of biceps rupture'. A hollowness is evident in the anterior portion of the shoulder. Test for power of flexion/extension. During active flexion the biceps retracts, producing the classic

[i] We struggle to recruit the young male patient with shoulder instability for our own clinical course. In clinic the shoulder may be painful and we would worry about the joint being examined on 10 or so occasions during the day. Also (although generalising) young male patients can be unreliable at turning up for a course (or exam) (PAB comment).

[j] A spot diagnosis does still exist in the UL short cases only you do not move on to another case after 1 minute. The 5 minutes is spent on a more thorough shoulder examination and dicussion.

deformity of the biceps contour. It is important to look carefully for any atrophy of the rotator cuff musculature because often there is an associated attrition-type tear in the superior rotator cuff or an associated subscapularis tear. Such tears often preceed the biceps rupture and may/may not have been symptomatic before the biceps rupture. Mention to the examiners that you would like to formally test range of shoulder movement and rotator cuff strength. A palpable click or catch in the shoulder through a range of motion may occur due to secondary inflammation of the rotator cuff tendon or a retained proximal biceps tendon stump.

Management

Management is usually non-operative, but look for associated cuff tears. If young, healthy, active individual consider repair for acute rupture or if chronic suggest possible tenodesis in the subpectoral area.

CANDIDATE: I had a proximal long head of biceps rupture turned around om me in the short cases and was asked about the bony attachments, nerve supply and action of the short head of beceps tendon which I struggled through. To make matters worse I was then asked about distal biceps tendon repair about which I knew nothing. *(FAIL)*

Distal biceps rupture

An acute biceps rupture recently pitched up for the short cases that was going to be operated on the following week. More likely the case will be a chronic neglected rupture or a rupture in an elderly patient that has been treated conservatively.

One- or two-incision repair

One incision: The modified Henry approach. Make a curvilinear incision over the anterior aspect of the elbow. Locate the ruptured distal biceps tendon. Insert bone suture anchors into the radial tuberosity and reattach the tendon. Alternatively endobutton fixation coupled with unicorical interference screw.

Two-incision approach of Boyd and Anderson: Make a 3-cm transverse incision over the distal biceps tendon sheath. Insert a core tendon suture through the end of the tendon. Make a second incision on the posterolateral aspect of the elbow. Locate the tunnel between the radius and the ulna through which the tendon originally passed. Make drill holes through the radial tuberosity to allow anchoring of the tendon. Retrieve the biceps through the distal incision, then pass sutures through the tuberosity drill holes and tie them down

Long head of biceps (LHB) tendon pathology

Many different tests have been described to detect proximal biceps pathology. However, establishing an exact clinical diagnosis can be difficult. These tests can be falsely positive in the presence of rotator cuff disease. Tests described include Speed's test, Yergarson's test, Ludington's test, de Anquin's test and Lippman's test. Nobody can be expected to know or perform all these tests[k].

In the exam setting we suggest performing two clinical tests only to confirm a clinical diagnosis. The Speed and Yergarson tests are probably best known to most examiners.

Sources of LHB tendon pain

1. Attritional tendonitis from degenerative or post-traumatic osteophytes in the biceps groove
2. LHB instability (secondary to a pulley lesion, cuff tear or rupture of the transverse ligament)
3. Partial traumatic tear
4. Intrinsic LHB tendinopathy
5. Primary synovitis
6. Post-traumatic scarring (e.g. with proximal humeral fractures)

Investigations

- Ultrasound scan – This demonstrates extra-articular LHB pathology only. Fluid in the sheath can be seen, along with biceps hypertrophy and tendinosis. Irregularity of the biceps groove, associated cuff tears. Dynamis scanning may demonstrate subluxation/dislocation of the tendon with arm motion.
- MRI scan
- Arthroscopy – Not very suitable for extra-articuar LHB. Gold standard for intra-articular LHB pathologies

SLAP tears[l]

SLAP tears are traumatic injuries, most common in athletes, generally caused by overload trauma of the superior labrum. A common cause is a fall on an outstretched arm.

History

- Traumatic episode involving arm hyperextension, or axial loading
- Vague shoulder pain often with painful clunking, clicking, snapping and popping of the joint
- Pain exacerbated with overhead sports, lifting, throwing (late cocking phase) or with extremes of motion
- Pain is described as deep and may be associated with generalized shoulder weakness when a concomitant rotator cuff tear is present

[k] Attempting to learn all the known tests with all the different subtle variations between tests would complicate your revision to the point of being unmanageable. It suggests being unfocused about what the exam is setting out to test for.

[l] Unlikely as a clinical case although the exam keeps moving on and you can never discount it.

Clinical examination

There are numerous clinical tests described for SLAP lesions. These tests attempt to reproduce the force of the injury mechanism. They are divided into two categories:

1. Tests that reproduce a torsional traction force to the superior labrum (active tests): (O'Brien test, anterior slide test (Kibler))
2. Tests which reproduce a compressive force to the superior labrum (passive tests): (compression rotation test (crank test), Mayo Shear/O'Driscoll's SLAP test)

Similar to AC joint pathology, you are not expected to know all the tests for SLAP lesions for the exam. This will just unnecessarily complicate your revision. The O'Brien's test is a reliable test for SLAP tears.

Investigations

- Standard shoulder radiographs (AP, axillary, scapular-Y and Styker notch views)
- MRI arthrogram
- Shoulder arthroscopy

Management

Conservative: Cessation throwing activities , physiotherapy, NSAIDs, etc

The majority of patients with symptomatic SLAP lesions will fail conservative management

Operative: Arthroscopic labrum debridement or labrum repair. Biceps tenotomy or tenodesis are alternative options

Shoulder arthrodesis

Short case 1

CANDIDATE: On inspection there is a large scar extending from the spine of the scapula over the top of the shoulder down the arm. There is gross distortion of the normal shoulder contour with gross muscle wasting. Examination of shoulder movements revealed elevation to 100° although rotation was grossly restricted. In particular, when stabilising the scapula it was apparent that all movement was scapulothoracic; no glenohumeral movement was appreciated.

EXAMINER: What operation do you think he has had?

CANDIDATE: I haven't seen this before but the length of the scar would suggest a shoulder arthrodesis. I'd like to confirm this clinically. To do this I stabilise the scapula with one hand and move the shoulder with my other hand: I see gross restriction of abduction, forward flexion, etc.

Indications

- Infection unresponsive to conservative treatment
- Stabilisation of painful, paralytic disorders of the shoulder
- Post-traumatic brachial plexus injury
- Salvage for a failed shoulder arthroplasty
- Stabilisation after resection of a neoplasm
- Recurrent dislocations

Remember bilateral glenohumeral fusions are poorly tolerated functionally and relatively contraindicated

Position of fusion

- 25°–40° abduction
- 20°–30° flexion
- 25°–30° internal rotation
- Or 30°, 30°, 30° is the classic exam answer

References

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Chapter 8

Elbow clinical cases

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Introduction

EXAMINER: 'As the elbow is a superficial joint, everything of what you need to see is there.'

With a careful visual inspection because much of the joint is subcutaneous any appreciable alteration in skeletal anatomy is usually obvious. Gross soft-tissue swelling or muscle atrophy is easily observed.

Look particularly at the inside of the elbow and at the back for scars.

Avoid circumnavigating the patient during your examination.

As with other joints in the body, a candidate must be thoroughly familiar with elbow anatomy and with the abnormal conditions that may be encountered.

There are patterns of examination for each joint. Every joint examination sequence is a unique dance.

Introduce yourself to the patient; ask them if they have pain.

Don't talk too much – Talking too much with too many negative findings can be irritating to the examiners: 'I cannot see any muscle wasting; I cannot see any scars; there are no swellings seen.'

Do not say 'obviously'.

If you elicit pain – Stop – Empathize with the patient – You may be forgiven. Hurt the patient again and it's goodbye.

History

Most elbow conditions for the FRCS (Tr & Orth) exam will be seen as short cases. Candidates will usually be asked to just get on and examine the elbow without the opportunity to take a detailed history. With the intermediate cases the elbow is more likely to appear as part of an upper limb polytrauma or generalized inflammatory arthritis case rather than a stand-alone case. The examiners may ask candidates to mainly focus on the elbow joint and take a more detailed history

Introduction

- Age
- Sex
- Dominance
- Occupation

As per any upper limb case. Get this out of the way on autopilot.

Presenting compliant

Most patients with elbow disorders present with pain. This is often associated with reduced elbow movement. Patients may occasionally complain of recurrent instability, intermittent swelling or locking.

Pain

- Site (where?)
- Medial, lateral or posterior?

The site of the pain may provide valuable clues. Conditions involving the lateral compartment (radiocapitellar joint) provoke pain that typically extends over the lateral aspect of the elbow, with radiation proximally to the mid humerus and distally over the forearm. Distinguish pain at rest from pain on movement (former due to arthiritis). Also distinguish from pain in mid-range of elbow motion to that felt at extremes of motion (former due to arthritis, latter due to osteophyte impingement).

- Type
  - Aching – degenerative arthrosis
  - Sharp pain/catching-loose body
  - Pain after activity – Tendinosis
- Onset (How did it all start?)
- Duration
- Radiation
  - To hand and forearm (tendinous, nerve pain)
  - To shoulder
  - To neck
- Aggravating/relieving factors

Stiffness

- Early morning stiffness – Rheumatoid arthritis

Swelling

- Onset

- Duration
- Localized
  - Posterior
    - Rheumatoid nodules
    - Olecranon bursitis
    - Gouty tophi
- Generalized
- Spontaneous

Deformity

- Congenital vs acquired
- Associated with trauma

Instability

- Clicking, catching, locking like symptoms
- History of elbow dislocation

Neurological symptoms

- Altered sensation
- Weakness

Paraesthesiae of the hand may, in some cases, be related to ulnar nerve compromise at the level of the elbow

- Clunk/locking

Activities of daily living

- Hand to mouth
- Perineal hygiene, comb
- Lifting and carrying objects
- Dressing
  - Coat
  - Bra

History of injury

- Mechanism of injury
- Treatment
- Outcome
- Sporting activities

Examination

Preliminaries

Introduce yourself and ask permission to examine the elbow. Get the patient to stand up if possible.

CANDIDATE: I would like to start by examining the elbow in the standing position. Can you please stand up for me, sir.

Adequately expose the shoulders, elbows, wrist and hands2

CANDIDATE: I would like to more adequately expose the patient.

EXAMINER: That's alright we are happy with this.

If the elbow pain has a radicular pattern it is important to examine the patients cervical spine. Look at spinal alignment, check range of motion and perform neurological testing of the entire upper limb. Elbow pain may be referred from the shoulder; therefore, a visual inspection of the shoulder for muscle wasting and appearance should be done.

CANDIDATE: In everyday clinical practice I would start by examining the neck and shoulders to rule out referred pain or radiculopathy[a].

Do look at the patient as a whole paying particular attention to the contours of the neck and shoulders.

Look

Front

'Can you show me your elbows, please?'
'Can you put your arms out for me?'

It is much easier if you simultaneously demonstrate this to the patient to make it obvious what you want them to do. Arms are held straight alongside the body with palms facing forwards.

'Can you put your arms out in front, palm facing out, can you lift your arms up in front of you?"

CANDIDATE: The shoulders are demonstrating a smooth movement with no pain or apparent restriction of movement. I'm inspecting the elbows from the front, there is a normal carrying angle, no fixed flexion deformity of the elbow /normal full extension. (Look from the side as it is easy to miss subtle loss of full extension.)

When the elbow joint is extended in the normal anatomical position with the palms facing anteriorly, the longitudinal axis of the forearm is at a slight valgus (lateral) deviation to the longitudinal axis of the arm. This is known as the carrying angle of the elbow. The normal carrying angle is 11–14° in males and 13–16° in females[b]. The elbow moves from a valgus to varus alignment with flexion. In a post-traumatic condition abnormalities in the carrying angle cannot be accurately assessed in the presence of a significant flexion contracture

'I can't comment on the carrying angle of the elbow as the arm has a fixed flexion deformity. The carrying angle cannot be assessed.'

Angular deformities such as cubital varus or valgus should be easily identified. Rotational deformities following supracondylar fractures or other fractures of the humeral shaft are more difficult to distinguish

[a] Safer to mention although the examiner will probably tell you not to bother and to just concentrate on the elbow

[b] The carrying angle helps the upper limb to clear the pelvis while the arm swings during walking

'There is a valgus deformity of the elbow suggestive of possibly an old lateral condylar fracture non-union or mal-union. I would like to go on and check for any associated ulnar nerve palsy.'

'There is a varus or gunstock deformity suggestive of an old mal-united supracondylar fracture occurring when a child or early physeal closure medially.'

With cubital varus the angle is $<5°$ whilst with cubital valgus the angle is $>15°$.

Popeye deformity is a spot diagnosis. A biceps bulge in the anterior arm above the elbow. Distinguish a popeye sign due to long head of biceps rupture from that seen in distal biceps tendon avulsion.

CANDIDATE: Please bend your elbows.

CANDIDATE: Can you show me the back of your elbows please?

The above will allow you to inspect the back of the elbow. Again show the patient what you want them to do. Look for scars, swelling, skin discolouration. Muscle wasting of the radial muscles (mobile wad-brachioradialis, extensor carpi radialis longus and brevis), ulnar muscles of the forearm or small muscles of the hand.

Scars

'I can see no obvious scars over the medial or lateral epicondyles, no excessive fullness of the medial or lateral recess, the medial and lateral recesses being well preserved.'

'There is a linear well-healed surgical scar over the posterior aspect of the elbow.'

Posterior – This is the gateway to the elbow surgically – Therefore, look for a scar.

CANDIDATE: I am looking for any obvious scars on the posterior aspect of the elbow, the recesses are preserved there is no obvious effusion.

Swelling

Swelling around the elbow can be either localized or generalized. Causes include an effusion, synovial thickening, periarticular soft-tissue inflammation or osteophytes.

General swelling of the elbow is usually due to an effusion. The prominent subcutaneous olecranon bursa is easily observable posteriorly if it is inflamed or distended. Rheumatoid nodules are frequently seen on the subcutaneous border of the ulnar. Also look for psoriatic patches, seen in extensor surfaces of joints.

CANDIDATE: There are no obvious rheumatoid nodules (ulna border forearm) or an olecranon bursitis. There is a filling out of the normal hollow seen on the lateral side of the elbow.

Fullness about the infracondylar recess[c] just inferior to the lateral condyle of the humerus; the 'soft spot' suggests either an increase in synovial fluid, synovial tissue proliferation or radial head pathology such as fracture, subluxation or dislocation.

There is loss of the normal concavities (hollows) on either side of the olecranon process. The lateral recess, or soft spot, is the most sensitive area to detect a joint effusion and can be palpated between the olecranon tip, the radial head, and the lateral epicondyle. If you see a rheumatoid nodule mention this to the examiners, otherwise they will not know if you have noticed it. It may be barn-door obvious to everyone but still mention it.

'The skin condition is papery thin/normal.'

Thin, taut, adherent, pale discoloured skin over the lateral epicondyle may suggest lipodystrophy due to repeated cortisone injections in this area for resistant lateral epicondylitis.

Watch that you do not go through a checklist of negative clinical findings on inspection and come across as though you are examining a patient but that you are not inspecting the patient correctly and miss subtle positive clinical signs.

Anterior

The popeye deformity is a common clinical finding in patients with distal biceps tendon ruptures due to proximal retraction of the muscle belly. A popeye sign with distal retraction of the muscle belly is suggestive of long head of the biceps rupture at the elbow. A spot diagnosis but be prepared for what comes next[d].

Lateral aspect

A normal depression in the contour of the skin in the infracondylar recess becomes obliterated in the presence of synovitis or effusion. Look for dimpling consisting with lipoatrophy.

Posterior aspect

A prominent olecranon suggests a posterior subluxation or migration of the forearm on the ulnohumeral articulation. Look for a swollen olecranon bursa.

Medial aspect

Few landmarks are seen from the medial aspect of the joint. The prominent medial epicondyle is usually seen unless the patient has a large BMI.

Move

The look feel and move sequence is altered to look, move and feel, as this seems to flow better. It is important to compare both sides so as to detect any subtle differences in movement.

[c] Landmarks are lateral epicondyle, subcutaneous tip of the olecranon and radial head.

[d] Proximal and distal attachments of the biceps tendon. Techniques for biceps tendon repair both proximally and distally. Testing for rotator cuff pathology. Nerve supply and actions of the rotator cuff muscles.

'Can you raise your arms out to the side?' (Show the patient what you want them to do.)

'Can you just bring your elbows out to the side and can you now bend your elbows up?'

'I am checking his active range of movement and it is slightly reduced on the left side by about 5°'

'Can you touch your shoulders now?' (Excellent way to demonstrate passive range of elbow movement, any loss of flexion is easily seen.)

Flexion/extension

The principal flexors of the elbow are

- Brachialis (C5, C6 – Musculocutaneous nerve)
- Biceps brachii (musculocutaneous nerve)
- Brachioradialis (radial nerve)

The prime extensors of the elbow are

- Medial head of triceps
- Lateral and long heads of the triceps are considered accessory muscles

Begin with the forearm supinated and extended.

The normal range of active movement is from 0° to 145°. Passive flexion is approximately 160° limited by bony structures (head of the radius against radial fossa, coronoid process against the coronoid fossa), posterior capsule tension and passive tension in the triceps. A functional range is 30–130°. If there is a fixed flexion deformity (FFD) of 50°, movement is recorded as 50° FFD – 145° flexion. Up to 10° of hyperextension is acceptable. Anything more suggests either hyperlaxity or injury. Has the patient got a flexion or extension block? Is the loss of movement due to muscle weakness, capsular constriction, deformity of the joint? Test passive and active movements to distinguish between muscle weakness and mechanical block.

'The patient has a fixed flexion deformity of 45° with a further range of movement to 100° compared to 0–130° on the opposite normal side.'

Passive extension should have a normal bone to bone end feel due to restriction by the anterior capsule and the olecranon contracting the humerus. On the medial side with flexion of the elbow subluxation of the ulnar nerve anteriorly with a palpable snap can occur in 10% of the population. A subluxing ulnar nerve may give rise to medial elbow pain. Palpate the ulnar nerve whilst flexing/extending the elbow to feel for anterior subluxation/relocation.

Supination/pronation

Active supination and pronation should be assessed with the elbow flexed 90° tucked to the side of the body to prevent compensation for forearm rotation by shoulder motion. Normal supination is about 85° and pronation is around 80°; however, a minimum of 50° in both directions is enough for daily function.

'Can you tuck your elbows into your side, turn your hands palms down and then palms up?'

Prime supinators of the elbow are:

- Biceps
- Supinator

Prime pronators of the elbow are:

- Pronator quadratus
- Pronator teres

With pronation the end feel should be elastic due to the resistance of the interosseous membrane and flexor muscles. There is a limited hard end feel in advanced osteoarthritis and pain with limitation of movement of the proximal radioulnar articulation. The movement may compress the median nerve in the presence of pronator syndrome. Pain at the end of range may be due to tendinopathy at the insertion of the biceps tendon on the radial tuberosity.

With supination there should be a normal elastic end feel caused by the interosseous membrane and ligaments.

Painful reductions in range of movement (ROM) may be due to effusion, soft-tissue swelling, or bony impingement. Locking or mechanical symptoms may be due to loose bodies in the joint. Crepitus may occur throughout ROM in patients with osteoarthritis, in addition to decreased ROM in all directions. Soft blocks to motion may represent capsular contractures, effusions, or soft-tissue swelling.

'Active pronation from the mid prone position is 40° compared to 90° on the normal left side. Extension is passively full and painless. I am checking for subluxation and any flexion impingement signs, which occur in early osteoarthritis including osteophyes. With full flexion and extension of the elbow this would exclude early osteoarthritis.'

Rotational deformity

The examiner stands behind the patient with the elbow flexed to 90° and the forearm behind the back. With the shoulder bent forward and the shoulder in full extension the forearm is lifted maximally, resulting in maximal internal rotation. Differences between the two sides can be measured by the angle between the forearm and the horizontal of the back.

Feel (palpation)

'Does it hurt, is it painful?'

Develop a defined approach to palpation. Know the anatomical structures encountered and dry run your technique until it flows smoothly. Palpation can be divided into four zones.

Lateral

It is usual to start from the lateral side palpating the lateral supracondylar ridge, lateral epicondyle, common extensor origin, lateral collateral ligament, radiocapitellar joint and supinator. It is important to be able to distinguish between tenderness in these areas (which are in close proximity) to work out the source of pain. Showing that you are specifically palpating these also demonstrates you appreciate possible differential diagnoses of lateral elbow pain.

The lateral epicondyle is smaller and less well defined than the medial epicondyle. The radial head is best felt while pronating/supinating the forearm. Assess for tenderness or clicking which may suggest a fracture, arthrosis, symptomatic posterolateral synovial plica or radial bursa. Congenital or post-traumatic dislocation or subluxation of the radial head will be appreciated at this stage. The anconeus 'soft spot' should be palpated to evaluate for fullness, which could signify joint effusion, haemarthrosis, or even a subluxed or dislocated radial head. The radiocapitellar joint can be easily assessed for tenderness or clicking over the radial head with supination and pronation. Tenderness directly over the lateral epicondyle can be secondary to trauma or lateral collateral ligament injury.

The common extensors originate on the lateral epicondyle, but tenderness due to lateral epicondylitis (tennis elbow) is generally elicited just distal and anterior to the epicondyle at the origin of the extensor carpi radialis brevis[e], even though it maybe felt directly over the lateral epicondyle, or even distally over muscle bellies.

Palpate for tenderness over the supinator, which may signify a posterior interosseous nerve entrapment, a differential diagnosis of lateral epicondylitis.

With the elbow flexed to 90°, the medial epicondyle, tip of the olecranon and the lateral epicondyle form an isosceles triangle. These form a straight line when the elbow is extended.

There is a sieve you will need to go through in order to narrow the diagnosis down as to why it is painful on the lateral side of the elbow.

Anterior

Know the anatomical structures around the cubital fossa. Palpate the brachioradialis muscle, biceps tendon, brachial artery, median nerve, lymph nodes, anterior elbow joint (either side of the biceps tendon) passing from lateral to medial. Distal biceps tendinosis may be detected, and is a common enough condition to be encountered in the exam.

Medial

The medial epicondyle is easily palpable at the medial side of the distal end of humerus. It is subcutaneous throughout and may be tender (along with the origin of the common flexors) with medial epicondylitis. The ulnar collateral ligament can be palpated with the elbow flexed 50–70°. The ligament is palpated from its origin at the inferior medial epicondyle along the ligament to its insertion on the proximal medial ulna at a tubercle on the medial margin of the coronoid process. Pain may indicate anything from a partial intrasubstance injury to a complete tear.

Distal and slightly anterior to the medial is the origin of pronator teres and flexor carpi radialis (FCR) tendons.

The ulnar nerve is located in the sulcus behind the medial epicondyle and the olecranon. It should be palpated beginning above the medial epicondyle, through the cubital tunnel and distally as far as possible into the flexor carpi ulnaris muscle mass. Gentle palpation or percussion of the ulnar nerve should not cause any significant pain or discomfort. It is round, soft and tubular in nature. Perform Tinel's test on the nerve – Assess for pins and needles or sharp sensations along the ulnar nerve distribution. Remember to palpate the nerve whilst passively flexing/extending the elbow, to look for ulnar nerve subluxation.

Posterior

In the midline posteriorly the olecranon process and fossa on either side of the triceps tendon should be assessed for tenderness, swelling, thickening, crepitus, or bony fragments.

The triceps muscle can be palpated along with the olecranon bursa. The two epicondyles and the apex of the olecranon form an equilateral triangle when the elbow is flexed 90° and a straight line when the elbow is in extension. Feel for the ulnar nerve, test for mobility, perform Tinel's test.

> 'I'm palpating for any crepitus, nodules, lumps, thickenings, loose bodies.'

There are several important structures that must be palpated and assessed medially. Pain with palpation of the medial supracondylar ridge and medial epicondyle may be caused by medial epicondylitis, medial collateral ligament (MCL) strain or tear, or fracture.

Special tests

- Tennis elbow
- Golfer's elbow
- Instability

Stiff elbow

Most likely a short case. The majority of cases will be secondary to trauma. Morrey et al.[1] defined the concept of a functional range of arc motion between 30° and 130° extension/flexion and 50° pronation/supination necessarily for an individual to perform 90% of normal daily activities. There is significant disability when these 100° range of motion arcs are lost. A loss of 50° in the arc of motion causes up to an 80% loss of function. An average functional arc may, however, not be acceptable in some professions or activities that require full extension of the elbow; hence, enquire what specific disability the patient experiences.

Aetiology

Broadly divided into post-traumatic, atraumatic and congenital.

- Atraumatic causes include inflammatory arthritis, osteoarthritis (coronoid/olecranon/radial osteophytes),

[e] If you suspect medial epicondylitis drill down on to the provocative tests for the condition.

loose bodies, burn injury, triceps/biceps adhesions, heterotopic ossification, chronic infection, hemarthroses in hemophiliacs
- Post-traumatic stiff elbow causes include distal humerus fractures. complex elbow fracture dislocations and radial head fractures
- Congenital causes include arthrogryposis, cerebral palsy and congenital radial head dislocation

Post-traumatic elbow stiffness is challenging to treat and often involves young, active patients. Several classifications exist:

- Early or late from the time of the injury
- According to the structure impeding elbow range of motion (soft tissue, osseous or combined)
- Intrinsic, extrinsic, or mixed causes[2]. Intrinsic contractures are secondary to involvement of the articular surface (articular mal-alignment, loose bodies, osteophytes, intra-articular adhesions). Extrinsic are those not involving the articulation (skin, muscle, capsule, collateral ligaments, heterotopic ossification (HO)) whilst mixed involves extrinsic contractures developing secondary to intrinsic pathology

The severity of stiffness is graded according to the arc of flexion, with very severe stiffness defined by an arc $<30°$, severe stiffness defined by an arc of 31–60° and moderately severe stiffness defined by an arc of 61–90°, although these may not directly correlate with the severity of functional disability experienced by the patient.

History

Age, hand dominance, occupation. Some activities in labourers require full elbow extension, and, hence, even small loss of elbow extension can be disabling.

Onset, duration, character, and progression of symptoms.

If trauma establish the exact injury of mechanism, type of fractures or instability and subsequent treatments. The old hospital notes should be obtained to determine previous surgical exposures, nerve transposition, metalwork used and any complications. Ask about infection.

Risk factors for stiffness after trauma include length of immobilization, associated fracture with dislocation, intra-articular derangement, delayed surgical treatment, associated head injury, heterotopic ossification.

Functional limitations

Recreational interests

Examination

Inspection of the skin for scarring, open wounds, and previous surgical incisions. All bony prominences are palpated to detect areas of tenderness that may limit motion secondary to pain. Look for deformity and swelling.

The elbow should be put through extension–flexion and pronation–supination arcs of motion and these ranges quantified with a goniometer both actively and passively.

It is essential to examine both elbow flexion–extension and pronation–supination arc of motion and compare to the opposite side.

Types of stiffness include loss of elbow flexion, loss of elbow extension and loss of forearm rotation. The character of the endpoint at the extremes of motion should be noted. While a firm endpoint suggests a bony block to motion, a soft endpoint is indicative of a soft-tissue contracture. A soft endpoint may also signify that the contracture may be prone to stretching out with bracing. Crepitus appreciated during elbow range of motion may signify degenerative changes or synovitis.

The typical post-traumatic elbow stiffness is painless. Pain at mid-motion suggests an intrinsic component to stiffness. Pain at the extremes of motion suggests impingement between the olecranon or coronoid process and the distal end of the humerus, usually due to osteophyte formation. Rotational stability, motor strength and neurovascular status of the extremity. The ulnar nerve should be assessed for irritability, subluxation and sensory and motor function as it is commonly involved in elbow trauma. With previous open reduction with internal fixation (ORIF), the possibility of infection should be considered. Assess muscle strength and co-lateral stability.

Investigations

Standard workup of AP, lateral and oblique radiographs.

CT with three-dimensional reconstruction may accurately localize loose bodies and/or impinging osteophytes and assist in planning arthroscopic debridement.

MRI is generally unhelpful.

Rule out infection before any planned surgery with aspiration if needed.

Nerve conduction studies if any neuropathy identified.

Management

A multidisciplinary approach among patient, surgeon, physiotherapist and others for optimal treatment[f].

1. **Non-operative**

Serial bracing. Either dynamic or static. Dynamic splinting based on creep (an increase in length with the application of a constant load for prolonged time), static based on stress relaxation (a decrease in load required to maintain a certain length over time). Splinting usually has a role to play in early stiffness (<6 months), but not in chronic established stiffness. Stiffness with 'softer' endpoints may also be more amenable to splinting. Splinting can aim to improve flexion, extension or, by using alternating splints, both.

2. **Operative treatment**

Only after failure of non-operative treatment.

Examination under anesthesia along with gentle manipulation for early contractures, followed by splinting.

[f] Buzz sentence to keep the examiners happy.

Chronic contractures may require surgical release. Extrinsic contractures are usually managed with open or arthroscopic release. Those with a large intrinsic component are managed with release combined with arthroplasty.

Open release is considered by many as the gold standard. Several approaches may be utilized for open capsular release, depending on the location of the main capsular contracture.

- Lateral column procedure. This involves arthrotomy, capsular release, and osteophyte excision. It allows release of the anterior and posterior capsule. The incision is centered over the lateral humeral epicondyle, elevating the brachioradialis muscle from the humerus, the common extensor origin from the lateral collateral ligament (LCL), and the brachialis muscle off the anterior elbow capsule. The lateral capsule is excised, and the medial capsule is incised. Intra-articular adhesions as well as coronoid osteophytes are removed. Elevation of triceps and anconeus muscles from the distal end of the humerus and proximal part of the olecranon allows release of the posterior capsular and debridement of the olecranon fossa
- Medial approach. Pronator teres is elevated from the common flexor mass, to expose and release the anterior capsule. The triceps muscle is elevated off the humerus and olecranon, allowing release of the posterior band of MCL and posterior capsule and removal of any olecranon osteophytes. This approach does not give adequate access to the lateral part of the joint
- Anterior approach. This accesses the anterior capsule to better manage flexion contracture. Used for isolated anterior ectopic bone excision
- Posterior approach. Allows extensive medial and lateral releases. Midline posterior incision. The ulnar nerve is decompressed and the posterior part of the MCL is released

Complications include neurovascular injury, inadequate release, instability if excessive release (may need to protect elbow with an external fixator post release) and recurrent stiffness. Protect against heterotopic ossification (HO) formation with NSAIDs.

Arthroscopic surgical release

Arthroscopic osteocapsular release involves the removal of osseous components, such as osteophytes and ectopic bone, and capsular release. This is a challenging procedure because of the close proximity of the neurovascular structures. Steep learning curve, associated with serious complications.

Distraction arthroplasty

Distraction arthroplasty is used for instability following contracture release and reattachment of the collateral ligaments and following interposition arthroplasty to protect the graft.

Fascial interpositional arthroplasty

Various interpositional materials including autograft fascia lata or Achilles tendon allograft. Possible treatment option in young high demand patient, in whom replacement arthroplasty is not indicated due to its short longevity.

Replacement arthroplasty

This has a limited role in the stiff elbow. It is generally used in patients over the age of 60 with advanced arthritis or post-traumatic destruction of the joint.

Examination corner

History: A 23-year-old man who presented to A&E with a rugby injury 8 months previously. A dislocated elbow on the dominant side, manipulation under sedation in, given a back slab. He missed his fracture clinic appointment and delayed elbow mobilization by 7 weeks, Did not attend physiotherapy as went back to university.

EXAMINER: Examine this man's elbow.

CANDIDATE: The elbow in an attitude of 40° flexion, with some fullness around the olecranon and cubital fossa. There is some wasting of triceps and biceps when compared to the opposite elbow. There are no visible scars. There is no local tenderness to palpation around the elbow structures, the bony relationship between the epicondyles and olecranon is maintained with the elbow at 90° flexion and this is comparable to the normal opposite side. The range of movement is from 40° to 100°. Supination and pronation are full. His elbow is grossly stable for valgus and varus stresses, although I would prefer to exam for this under GA and image intensifier guidance. Sensation in the autonomous zones for median, ulnar and radial nerves are normal.

EXAMINER: These are the radiographs pre- and post-manipulation.

CANDIDATE: The AP and lateral radiographs show a posterolateral elbow dislocation in pre-reduction films, with no associated fractures noted. Post reduction through the back slab demonstrates a well-reduced elbow immobilized in about 100° of flexion.

EXAMINER: How would you manage a patient with a closed posterolateral elbow dislocation following a successful manipulation?

CANDIDATE: I would try and assess stability in about 30° of flexion after achieving reduction. If unstable, I would immobilize the arm in an above elbow back slab with the elbow in 90° flexion and the forearm in full pronation. This is followed by checking x-rays. I would try and limit the period of immobilization to <2 weeks and initiate early ROM exercises and physiotherapy. I would warn the patient about the possibility of stiffness and residual terminal restriction of extension and the importance of complying physiotherapy.

EXAMINER: This gentleman is struggling with elbow stiffness and all conservative measures have failed to improve his ROM. What would you do?

CANDIDATE: I would ask for some updated elbow radiographs and in addition request a CT scan to look for any bony blocks to

movement, such as post-traumatic ossification, loose intra-articular bony fragments or joint irregularity. An MRI may also be helpful in demonstrating intra-articular damage, cartilaginous loose bodies or impingement but generally less so than a CT scan.

EXAMINER: Ok, the x-rays and CT scan do not show any abnormality. What are you going to do?

CANDIDATE: This gentleman would be best managed by a Shoulder and Elbow surgeon with a plan to perform an EUA and gentle manipulation followed by physiotherapy, and stretching bracing. The stiffness in this case could be due to soft-tissue contracture including capsular and ligamentous involvement. If this failed to significantly improve his range of movement then an open or arthroscopic release followed by CPM or physiotherapy would be the next step.

EXAMINER: Yes, the evidence with CPM is not robust, but I suppose, nothing wrong in getting some passive motion early after surgery. Let's move on!

Stiff elbow mainly involving loss of supination and pronation

Think of rotation problems secondary to involvement of the superior or inferior RU joint such as congenital or acquired radial head dislocation, distal ulnar dislocation or a mal-united fractured radius and ulna with distorted interosseous anatomy. A congenital or traumatic synostosis especially in proximal third forearm injuries is a fairly common elbow short case.

Management of post-traumatic RU synostosis

A rare complication following fracture of the forearm and elbow. Risk factors for synostosis are related to the initial injury and surgical management of the fracture. Typically, patients present with complete loss of active and passive forearm pronation and supination. Evidence of bridging heterotopic bone between the radius and ulna can be seen on plain radiographs. Typically surgical excision is required. The timing of surgical intervention remains controversial. Early resection between 6 and 12 months after the initial injury can be safely performed in patients with radiographic evidence of bony maturation. Surgical management consists of complete resection of the synostosis with optional interposition of biologic or synthetic materials to restore forearm rotation. The rate of synostosis is higher in patients following a head injury, occurring in as many as 18%.

Risk factors: (1) Comminuted fractures of both the radius and ulna at the same level in the proximal third; (2) Head injury; (3) Iatrogenic trauma – Single incision technique, bone fragments or reamings, bone graft or hardware in the interosseous space; (4) Disruption of the interosseous membrane; (5) Severe soft-tissue injury; (6) Surgical delays in fracture fixation. Primary bone grafting should be used judiciously. When used the graft should be directed away from the interosseous membrane.

Clinical assessment

History to include original injury, any associated head injury, timing from initial injury to surgical management, postoperative complications, previous surgeries in the same bone. Hand dominance, occupation, recreational activities, patient's expectations and functional demands. Any residual deficit from a head injury may preclude the ability to comply with postoperative rehabilitation.

Patients with post-traumatic RU synostosis usually present with decreased ROM, specifically forearm rotation. It is important to assess the position of the forearm, because fixed pronation is associated with much less function than is fixed supination. Synostosis may interfere with elbow flexion and extension, as well, if the heterotopic ossification bridges to the humerus. A through neurovascular examination should be performed including looking for anterior and posterior interosseous nerve dysnfunction.

Forearm rotation is important in most tasks, and complete loss of pronation and supination can result in severe impairment of activities of daily living.

Most positional and functional tasks can be achieved within a rotation arc of 100° (50° pronation and 50° supination). More contemporary tasks such as using a keyboard, usually require an even greater arc of forearm rotation. Again enquire about disability experienced by the patient, as this will be influenced by activities the patient is involved in (different in a keyboard user, from a manual worker carrying weights).

Radiographs will confirm the diagnosis with bridging heterotopic bone between the radius and ulna. Serial radiographs may be helpful in assessing the progression of lamellar bone formation and eventual maturation. CT should help with the anatomy, size and location of the synostosis and three-dimensional CT may aid surgical planning in terms of surgical approach. Serial bone scans and alkaline phosphatase levels are no longer routinely requested for monitoring, serial x-rays are sufficient.

Non-surgical management: A thorough assessment of functional loss is required for each patient. Mild functional disturbance may be managed non-operatively. Also consider conservative management in low demand patients, recurrence following previous surgery, frail and unfit patients and those patients with the forearm in a fixed but functional position.

Surgical management: It is important to warn the patient about the risks of neurovascular complications and that the results from surgery can often be disappointing. Complications of surgery include neurovascular injury, infection, fracture, incomplete restoration of the forearm pronation-supination arc, recurrence of synostosis, risk of instability at either the proximal or distal RU joint.

Timing of surgery: This remains controversial. Surgical intervention is safe within 1 year of injury in a patient with radiographic evidence of bony maturation (a well-defined margin and bridging trabeculi). Early resection (6–12 months) has been performed safely without an increase in recurrence in patients with radiographic evidence of bony maturation.

Examination corner

Short case 1

EXAMINER: Would you examine this man's forearm?

CANDIDATE: This was the first short case and I just wasn't expecting this. I was completely unnerved and froze. Examine this man's elbow – Yes, examine the wrist – Yes, examine the forearm – No never ever been asked to do this ever! There was reduced forearm rotation and I was asked what I thought the diagnosis was. I said traumatic radioulnar synostosis but was then asked to examine the opposite forearm, which also had markedly restricted rotation. It was a congenital radioulnar synostosis. Part of the examiner's amusement when candidates say traumatic is to then make them examine the opposite forearm. I was then shown radiographs which demonstrated the synostosis and then asked about management. *(Fail – 5)*

Painful elbow with instability

Patients with a history of trauma and multiple postoperative scars around the elbow following trauma may have had their radial head replaced, medial epicondyle fixed, soft tissues repaired by suture anchors, etc. Remember the terrible triad (radial head and coronoid fracture with a posterolateral elbow dislocation) that often requires surgery and usually has some sequelae or residual problem.

Posterolateral elbow rotatory instability following previous open elbow dislocation

EXAMINER: Take a brief history and examine this gentleman's right elbow.

CANDIDATE: (*After 2 minutes*) In summary, this 22-year-old right-hand dominant man, supermarket worker, fell down from his bike 11 months ago sustaining an open injury to his elbow. This was operated on the same day with wash out, and primary closure. He had plaster immobilization for 3 weeks followed by physiotherapy. His main complaint is of clicking and pain especially when stacking shelves at work and also with gym activities such as bench pressing or doing parallel bars, etc. He is otherwise fit and well, a keen sports person.

EXAMINER: Ok, go on and examine the elbow.

CANDIDATE: The right elbow is in an attitude of flexion and slight pronation, with a healed irregular scar over the posterolateral aspect of elbow suggestive of the open wound with no signs of infection or inflammation. There is slight wasting of the triceps muscle compared to left side. Looking from back with the elbow flexed to 90°, the normal bony relations are maintained. There is tenderness over the lateral aspect of elbow . . . *(not very comfortable for the patient).*

EXAMINER: . . . Ok, there is nothing much to palpate any more, would you like to comment on his ROM?

CANDIDATE: The elbow lacks about 20° of terminal extension but further flexion to about 130° is possible and painless. Almost full supination and pronation is noted with the elbow in 90° flexion compared to opposite elbow.

EXAMINER: What do you think his symptoms are due to?

CANDIDATE: It appears that he is getting symptoms when he takes weight on the elbow, I suspect there is an element of injury to the lateral ulnar collateral ligament with instability?

EXAMINER: Is there a simple clinical test to find out the instability?

CANDIDATE: I would like to do varus stress test, pivot shift test . . .

EXAMINER: A simple test the patient can do it actively himself?

CANDIDATE: Oh, yes, rising from chair actively pushing his weight on both elbows the patient would be reluctant to extend their elbow fully *(elbow chair rise test)* or the floor push-up test?

EXAMINER: Yes, it is easy to do if you have a chair with arm rests! Go on, demonstrate that!

. . . .. so, does this help with your clinical diagnosis?

COMMENT: The elbow chair test is easy and quick to perform in a clinical setting (Figure 8.1). A patient is asked to push off the arm rest to stand up with both forearms in supination and the arms abducted. In this situation the patient is reluctant to fully extend the elbow since the maneuver applies an axial load, valgus and supination force which causes posterolateral instability. Apprehension, subluxation or frank dislocation may occur as the elbow is extended, depending on the severity of the instability.

With the (floor) push-up test the patient pushes off the floor with the forearms maximally supinated, elbow flexed 90° and arms abducted (Figure 8.2). The test is positive for posterolateral rotary instability if apprehension, subluxation or frank dislocation occurs with terminal elbow extension.

CANDIDATE: Yes, I can see the posterolateral subluxation of radial head over the end of distal of humerus.

EXAMINER: How would you manage this?

CANDIDATE: Confirmation of diagnosis by means of review of all radiographs performed from day 1 and further testing for posterolateral instability with examination under GA and image intensifier. MRI with contrast may show a torn LUCL (lateral ulnar collateral ligament). Some form of reconstruction of LUCL using a autologous/synthetic graft by an experienced surgeon.

EXAMINER: OK, let's move on to the next case . . .

A more recent test is called the table-top relocation test (Figure 8.3). The patient performs a press-up on the edge of a table using one arm, with the forearm in supination. In the presence of instability, apprehension or pain occurs at about 40° flexion.

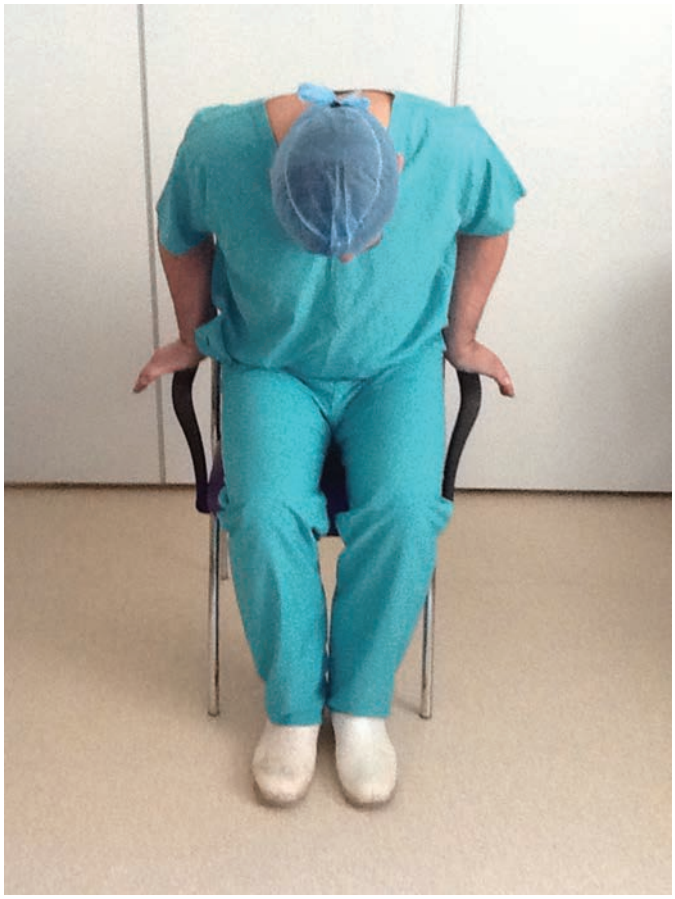

Figure 8.1 Photograph showing the chair sign. In the presence of instability, on attempting to rise from a chair pushing down only with the arms, and with the forearms supinated apprehension or radial head dislocation occurs as the elbow extends

Elbow instability

Valgus instability (ulnar collateral ligament injury)

Valgus stress testing is performed with the forearm fully externally rotated. Opening up of the elbow, local pain and tenderness suggest ulnar collateral ligament injury.

Varus instability

A varus stress is applied across the elbow joint with the shoulder fully internally rotated to lock the shoulder. If instability is present a gap between the capitellum and radial head increases.

Both valgus and varus stress testing are performed with the elbow in full extension and in 30° of flexion that unlocks the olecranon from the olecranon fossa.

Rotatory instability

Posterolateral rotatory instability (PLRI) results from insufficiency of the lateral ulnar collateral ligament (LUCL). It is the commonest instability pattern encountered in clinical practice. Patients complain of lateral elbow pain with recurrent clicking,

Figure 8.2 Photograph showing the active floor push-up sign

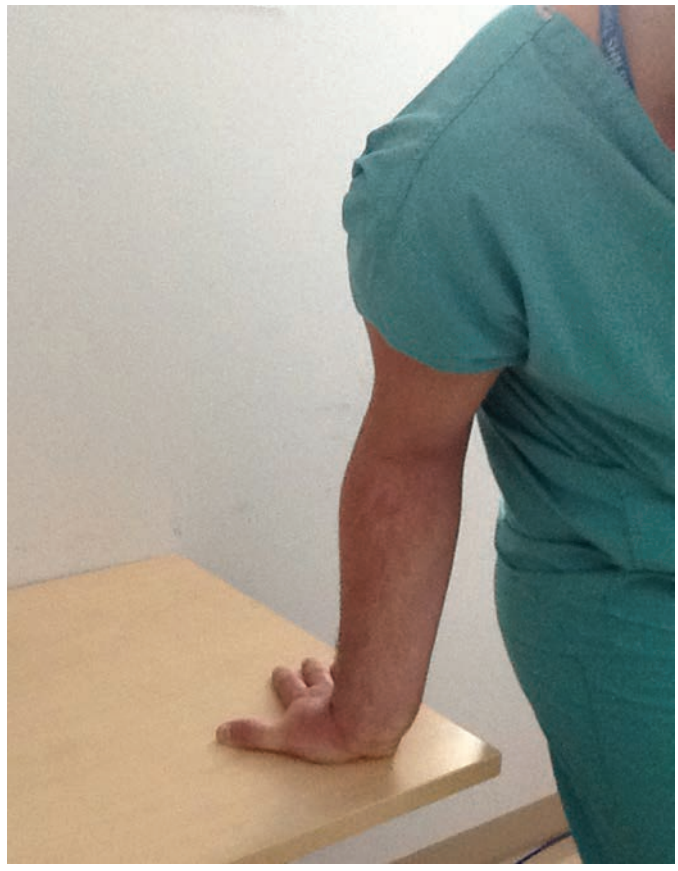

Figure 8.3 Photograph showing the table-top relocation test. A press-up on the edge of a table with the forearm in supination causes apprehension at about 40° flexion if instability is present

popping, snapping or locking of the elbow accompanied by a sense of elbow instability. Pushing down with the upper extremity to rise from a seated position, performing press-up/push-up exercises, and pushing heavy objects with an extended arm are common activities that reproduce symptoms. These activities place the elbow in an unstable position of external rotation of the forearm with valgus and axial loading of the elbow. Examination of a patient with PLRI is often unremarkable. Often range of motion is within normal limits, and varus and valgus stress tests are usually not provocative. Tests to confirm the diagnosis include:

1. **Lateral pivot shift (O'Driscoll)**

   Patient is supine, affected limb overhead. With forearm supinated, valgus and axial loading applied, the elbow is flexed from full extension. In posterolateral rotatory instability as the elbow is flexed the radial head subluxes or dislocates and is seen as a prominence posterolaterally. With flexion beyond 40° the radial head suddenly reduces with a palpable and visible clunk. The test is best performed under GA for radial head dislocation and relocation to be seen. When this maneuver is performed with the patient awake, the test is positive in presence of apprehension
2. **Drawer test**

   With the elbow flexed to 40°, anteroposterior force is applied to the radius and ulna with the forearm in external rotation. This aims to sublux the forearm away from the humerus on the lateral side, pivoting on the intact medial ligaments. Under GA the radial head is seen dislocating, whereas with patient awake apprehension occurs. This is often the easiest test to perform
3. **Arthroscopic examination**

   This reveals a widening of lateral joint space and/or posterior subluxation of the radial head, but not as accurate as EUA

Elbow instability following previous trauma (terrible triad)

This is also a common presentation, with either failure to repair LCL at the time of fracture fixation or failed reconstruction with persistent instability. Complications of a terrible triad injury include chronic elbow instability as well as stiffness, infection, pain, ulnar neuropathy, mal-union, non-union, heterotopic ossification arthrosis, osteoarthritis, and contracture.

In most cases, the examination will, therefore, be limited to LOOK, FEEL AND MOVE within the limits of pain and comfort. Special tests such as stress and instability tests are rarely tolerated, but a knowledge of underlying principles behind these tests is important.

These are cases where a candidate may be asked to take part of a history from the patient. A good summary of the history, a methodical description of what you see, a gentle feel of structures around the elbow including the bony relations along with careful demonstration of ROM with as little distress to patient as possible would earn candidates a good safe 6 pass. There will be time for review of radiographs and also discussion and plenty of opportunity to score extra marks with this cases.

Terrible triad injury of elbow

A fall on outstretched arm resulting in valgus, axial and posterolateral rotatory loads across the extended elbow joint. Most often follows a fall from motorbikes, injuries during contact sports, etc. Besides radial head and ulnar coronoid process fractures (which are often multifragmentary), the LCL is often torn. The anterior bundle of MCL may also be torn in the most severe injuries.

Fixation or replacement of radial head and ORIF of the coronoid process adds to static stability. However, it is the repair of lateral collateral ligament complex that is the key element of the whole reconstruction of the terrible triad. An assessment for stability under image intensifier should be made at this point of surgery and if needed, a repair of MCL should be performed.

The results from these injuries are often poor because of associated stiffness or instability. Careful reconstruction of all the bony and ligamentous injuries is, therefore, important to allow early mobilization using a hinged brace with a block to terminal extension.

Elbow instability following previous elbow surgery (tennis elbow release, surgical approaches to the lateral side elbow and radial head)

Over aggressive release of the LUCL, the release extending beyond equator of radial head can potentially make the elbow unstable in a varus or posterolateral direction. The scar may be similar to a typical tennis elbow scar. Sometimes, instability may follow an arthroscopic soft-tissue release (inadvertent lateral ligament complex release). It is important to describe the type and location of a scar to help differentiate between either a traumatic or elective procedure.

Cubital valgus/ulnar nerve

The carrying angle cannot be assessed fully when there a fixed flexion deformity Causes include: Non-union of lateral mass in children; non-union of intercondylar fractures in adults; injuries or infection in parts of distal humeral epiphysis in childhood leading to differential growth on either side of elbow; epiphyseal dysplasias such as Ollier's or multiple exostosis.

A thorough examination of the ulnar nerve is required keeping in mind the ulnar paradox, high or low ulnar nerve features.

'Can you show me your elbows, please?

(Demonstrate what you want the patient to do.)

'A spot diagnosis: This patient demonstrates a cubital valgus deformity of the elbow. There is also a suggestion of loss of full extension. There are no scars present over the medial or lateral epicondyle.'

Go straight to the hand to look for ulnar nerve signs (wasting of the interossei, hypothenar muscle wastage, sensory changes, etc).

'Is there any weakness of your hand or numbness and tingling of your fingers?'

'The most common cause is a lateral condylar mass non-union from childhood leading to a valgus deformity of the elbow (Milch type II fracture). There is usually marked prominence of the medial epicondyle.'

Examination corner

Short case: Cubitus valgus elbow in a 65-year-old male

Milch type II fracture of the lateral condyle missed at the age of 6 years.

Features of tardy ulnar nerve palsy.

EXAMINER: Would you examine this gentleman's elbows and describe what you are doing as you go along?

CANDIDATE: Can you show me your elbows, please, sir? On examination there is an obvious left cubitus valgus of the elbow present with a suggestion of loss of full extension.

EXAMINER: Come on, where else do you want to look? You look at his hands. Does he have evidence of ulnar nerve dysfunction?

CANDIDATE: Looking at his hands there appears to be hypothenar muscle wasting.

EXAMINER: Come on, where else do you have wasting? What about the back of his hands? You should be jumping to examine it. What about his interosseous muscles?

CANDIDATE: There is wasting of his dorsal interossei, particularly the first dorsal interosseous. Can you feel me touching your little finger? Does it feel normal? He has reduced sensation over his ulnar 1½ digits and also over the ulnar, dorsal aspect of his wrist.

EXAMINER: Do you need nerve conduction studies to confirm your clinical findings?

CANDIDATE: No.

EXAMINER: Good. What do you think of his x-rays?

CANDIDATE: These are AP and lateral radiographs of the left elbow. They demonstrate an old lateral condylar fracture, which has gone onto a non-union. There are severe secondary arthritic changes present in the radiocapitellar and humeroulnar joints.

In view of his sensory and motor ulnar nerve symptoms and non-union present with secondary degenerative changes, I would offer him ulnar nerve decompression with medial condylectomy and anterior transposition of the nerve. I do not think a simple decompression is adequate management for this gentleman.

Intermediate case

History

'A 50-year-old woman with long-standing, well-controlled (methotrexate) rheumatoid arthritis in hands and wrists. Fell onto right elbow 6/12 ago – Told it may be fractured, not sure. Managed in sling for 6/52. Since then has had an increasingly stiff, aching elbow with pins and needles along ulnar border of hand.'

Examination

'Very stiff, but not so painful, elbow. Explained I would start with neck and shoulder, etc, but told to concentrate on the elbow. Tinel's test very sensitive over cubital tunnel. Neuro examination distally. Motor fine, altered ulnar nerve sensation. Talked through functional assessment of hands (grips, etc).'

Discussion

'Investigations – x-rays (very poor quality). They were pushing me towards nerve conduction studies but I said it was obvious where the pathology was (? brave?!). Diagnosis of tardy ulnar nerve palsy 2-years post injury. I said there was nothing to suggest it was due to cubitus valgus but, apparently, you can't comment on this if the elbow won't extend! Discussed surgical approach for decompression, pros/cons of transposition (increased risk of late complications owing to nerve ischaemia).'

Cubitus varus with hyperextension deformity

EXAMINER: Examine this girl's right elbow.

CANDIDATE: *(Introduces himself)* . . . Could I ask you how old are you?

This 11-year-old girl has a varus deformity involving her right elbow with some hyperextension in right elbow when compared to left elbow. There are two tiny mature scars on either side of elbow, the one on medial side slightly larger (about 2–3 cm), no swelling noted, the normal bony relations maintained both in 90° flexion and extension when compared to left elbow. There is slight bony irregularity in supracondylar ridge over lateral aspect with no tenderness around the elbow. She can demonstrate good range of movements from –10° of extension to about 140°; flexion is slightly limited when compared to left elbow. Full and painless range of rotations of forearm with elbow in 90° flexion. Elbow appears to be stable and painless in both full extension and 30° flexion.

EXAMINER: What would you do?

CANDIDATE: Can I ask some history?

EXAMINER: She has been telling a similar story to all the previous candidates. She had a fall when she was 7 years old and had some surgery. Deformity noted about 1 year after injury, no pain and quite a keen gymnast!

CANDIDATE: She is most likely to have a malunited supracondylar fracture with no neurovascular deficit. This is an isolated deformity to right elbow, no evidence of hypermobile joints, functionally she seems to be doing well and I would like to know what bothers her and the family.

EXAMINER: Being a girl, it is obviously the cosmoses that she is bothered about! What are the causes of this deformity in general?

CANDIDATE: In general terms, the most common cause especially following injury in childhood is a mal-united SC fracture, but injuries to lateral condyle with consequent overgrowth, any injury or infection to medial side with growth arrest can lead to a varus deformity. Commonly,

it failure to correct the internal rotation and varus displacement during manipulation of supracondylar fractures which leads to a varus deformity with some degree of hyperextension.

EXAMINER: How do you manage?

CANDIDATE: I would like to look at all the available radiographs and determine the degree of deformity. However, since it functionally not causing any problems and also she is actively growing, I would explain that all surgery should be postponed until she is skeletally mature.

EXAMINER: What are the surgical options then?

CANDIDATE: Either a closed- or open-wedge osteotomy; A closed-wedge osteotomy with intact medial hinge, even though inherently stable and simple to perform, has been reported to have a lateral prominence in the longer term. Alternatives such as step-cut osteotomy or reverse step-cut, dome osteotomies have been described that reduced such the lateral prominence with good stability, but are technically demanding. The surgery be best performed by a specialist with expertise in such deformity correction.

Rheumatoid elbow

Short case 1

'On examination the patient has features of a generalized polyarthropathy, probably rheumatoid arthritis. There is an old well-healed scar over the lateral aspect of the elbow suggestive of previous surgery to the radial head. There are large rheumatoid nodules overlying the olecranon bursa. There are no features of either PIN (posterior interosseous nerve) or ulnar nerve neuropathy.'

Mention coexistent assessment of shoulder and hand function.

Short case 2

EXAMINER: Examine this lady's right elbow please. *(Woman in her late 40s.)*

CANDIDATE: *(After greetings and introduction.)* This lady's right elbow is in an attitude of flexion with subcutaneous nodules over olecranon, generalized swelling obliterating all the normal bony landmarks around the elbow. The skin is very thin and shows areas of ecchymosis and vasculitic skin lesions. The forearm is in an attitude of pronation. Generally, I can see a polyarticular arthritis of both hands and wrists with some wasting of the forearm and upper arm muscles. There is a slight local increase in temperature around her elbow, she seems to be in a degree of pain; therefore, I am going to feel for bony land marks as gently as possible. I can feel a bony rounded mass over the posterolateral aspect that appears to be the radial head. Gentle rotations of forearm confirm this. She is generally tender around the elbow joint with an effusion and some soft-tissue swelling over the ante-cubital fossa. She maintains a good range of flexion and extension in her elbow from about 40° to 130°[g], but the forearm is in almost full pronation with painful rotatory movements. Despite the involvement of other joints in the right arm, she seems to maintain the global movements of the arm and her hand-grip strength appears to be good.

EXAMINER: OK, let's concentrate on the elbow. What is her most disabling problem now? What is she going to struggle with?

CANDIDATE: *(The candidate asks the patient whether she is struggling to get her face washed.)* Looking at her both forearms that are in fixed pronation, her main problem seems to be getting her hand to her face.

EXAMINER: Yes, a supination deformity can easily be compensated by internal rotation of shoulder, but a pronation deformity like she has can be very troublesome. OK, these are the x-rays of this lady's right elbow.

CANDIDATE: AP and lateral radiographs of the elbow show marked soft-tissue swelling and some periarticular osteopenia and erosions, mild reduction in the joint space mainly radiocapitellar and no architectural distortion.

EXAMINER: Good, what is your diagnosis?

CANDIDATE: Poly-articular rheumatoid arthritis (RA) with possibly stage 2–3 Larsen grade, symptomatic in the form of pain and restricted supination and functional impairment with ADLs. Because it is at an early stage and she maintains a good range of flexion and extension, synovectomy either arthroscopic or open may help to control pain and improve her ability to supinate, which is the major disabling restriction for her.

EXAMINER: OK, we will go the next case.

Nearly 50% of RA patients have elbow involvement and in the majority of cases, it is bilateral. In the early stages, synovitis causes pain and tenderness, especially over the radiohumeral joint line, with associated loss of elbow extension. Later, the whole elbow may become swollen and stiff. Finally, when bone destruction is severe, instability and capsular rupture result in a flail elbow. Ulnar collateral ligament incompetence may cause valgus ulnar humeral instability and ulnar nerve dysfunction. Annual ligament incompetence can lead to radial head subluxation.

Symptoms from the rheumatoid elbow include pain, stiffness, swelling, instability and ulnar nerve dysfunction.

Examination of RA elbow: Look for scars, deformity, muscle wasting, rheumatoid nodules, swelling, composite movement of the whole upper limb into positions of function. Feel for rheumatoid nodules, any swelling, tenderness, ulnar nerve irritation. Movements: Flexion/extension, pronation/supination, crepitus and joint instability.

Larsen grading for RA elbow:

Stage 1: Involves the soft tissues and has near-normal radiographs

Stage 2: Presents with periarticular erosions and mild cartilage loss, there may be evidence of soft-tissue swelling and osteopenia on radiographs

Stage 3: Radiographs show marked joint space narrowing

Stage 4: Progresses to advanced erosions penetrating the subchondral bone plate

Stage 5: Radiographs show advanced joint damage and loss of articular contour

[g] Get the goniometer out for a more precise measurement.

Stage 1 and 2 may respond to synovectomy

Limited flexion–extension is an independent risk factor for a poor result. Therefore, it is important to demonstrate a good flexion and extension arc before surgery.

In the early stages of RA elbow, synovectomy is expected to improve pronation and supination better than flexion and extension.

Surgery is indicated when appropriate non-surgical management has failed, giving rise to functional limitations due to pain or loss of motion. The primary aim of surgery on the elbow is to relieve pain and/or restore joint function. Pain is the most common primary indication for elbow surgery. The pain relief is most predictable and complete after total elbow arthroplasty (TEA). A range of movement of $<100°$ that does not allow the patient to reach their mouth or perineum and is an indication for surgery. TEA is generally effective at restoring a functional arc of motion. A linked TEA may be effective in patients with instability and pain as major symptoms.

Larsen stages 3–5 may require TEA. There are two basic types of elbow (TEA) implants:

1. **Linked implants** are joined together by a 'sloppy hinge' to allow for some varus and valgus laxity during range of motion of the elbow; early loosening is a concern with these implants. However, they provide stability and do not rely on intact collateral ligaments which may be attenuated in rheumatoid elbows, or compromised at surgery. In patients with inflammatory arthritis, the soft tissues are often attenuated and there is a lower threshold for using a linked prosthesis
2. **Unlinked implants**, the humeral and ulnar components are not joined together and stability is provided by the surrounding soft tissues. Instability is the main concern with this implant construct

Complication rates can be as high as 40–45% and include infection, instability, loosening, wound healing, ulnar neuropathy, triceps insufficiency, periprosthetic fractures. In RA, 10-year survival rates of TER of between 80% and 92% have been reported.

Medial-sided elbow pain with ulnar neuritis

Causes of medial elbow pain including osteochondritis dissecans, loose bodies, ulnohumeral osteoarthritis, Golfer's elbow, snapping elbow (painful ulnar nerve subluxation).

Tennis elbow (lateral epicondylitis)

Typically a short case in which a candidate would be asked to demonstrate provocative tests for tennis elbow.

This is a syndrome/symptom complex characterized by the following:

1. Pain over the lateral epicondyle and proximal forearm exacerbated by movements involving a combination of a gripping hand and a forearm rotation
2. Tenderness on palpation of the extensor muscle origin at the lateral epicondyle
3. Reproduction of pain when the forearm and wrist extensor muscles are actively extended or passively flexed

Pathology

Uncertain – There are several theories:

- Avascular degenerative process
- Humeroradial bursitis
- Inflammation of the annular ligament of the radius
- Secondary trauma

Or, it may present as part of a 'generalized mesenchymal syndrome'.

Clinical features

Gradual onset of pain over the lateral epicondyle with radiation down the proximal forearm in line with the extensor muscles.

Differential diagnosis (may coexist with tennis elbow)

- Radiocapitellar arthritis/synovitis/plica
- Radial tunnel syndrome

Provocative tests

There are several provocative tests for lateral epicondylitis, but of the ones described below, the first two are the ones recommended for the exam.

Pain with the resisted wrist extension test

With their elbow extended, ask the patient, 'Could you make a fist, please; can you cock your wrist backwards' (getting the patient to extend their wrist) 'and resist me now? (Try to flex the wrist against resistance, feeling the lateral epicondyle at the same time.) This should reproduce the patient's symptoms.

Middle finger extension test (Maudsley's test)

Extending the middle finger against resistance reproduces pain by stressing extensor carpi radialis brevis (ECRB).

Chair lift test

This involves picking up a chair with an adducted shoulder, extended elbow and pronated wrist.

Bowden's test

The patient is requested to squeeze together a blood pressure measuring cuff inflated to around 30 mmHg held in their hand. Pain over the lateral epicondyle is suggestive of lateral epicondylitis.

Mill's test

The patient is asked to pronate the forearm and flex the wrist. The patient is then asked to supinate their arm against resistance.

Grip strength can be tested and compared with the contralateral side as patients often report weakness when gripping items.

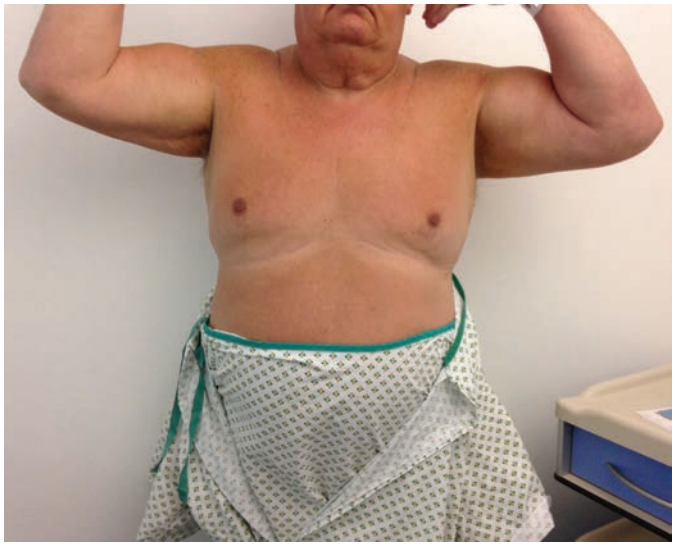

Figure 8.4 Truck driver, 60 years old, struggling with shaving and carrying shopping bags on the right side. Active elbow flexion of 90° (normal value is 145°)

Radiographs

Radiographs of the elbow are usually normal.

Management

Conservative

- Initially conservative as 90% of cases settle by 12 months
- Rest, modification of activities, non-steroidal anti-inflammatory drugs (NSAIDs), physiotherapy, epicondylitis (EPI) clasp, steroid injection

Surgery

- Release of ECRB either open or arthroscopic (it is often difficult to solely release ECRB)
- Extensor origin may or may not be repaired (usually not repaired)
- A short period of elbow immobilization in a plaster or splint postoperatively is used by some surgeons
- On average, 85% of patients will attain complete relief of symptoms with surgery, 5% will see no benefit and 10% will have residual symptoms
- Complications include iatrogenic LUCL injury, radial nerve injury, missed radial nerve entrapment (5%)

Examination corner

Short case

EXAMINER: Would you like to examine this lady's elbow?

CANDIDATE: On inspection the elbow looks normal. Flexion is full from 0° to 140° and painless. Full extension at its extreme point is, however, painful. There is definite point tenderness over the lateral epicondyle of the humerus and possibly weakness of grip strength.

EXAMINER: Can you demonstrate some provocative tests for tennis elbow?

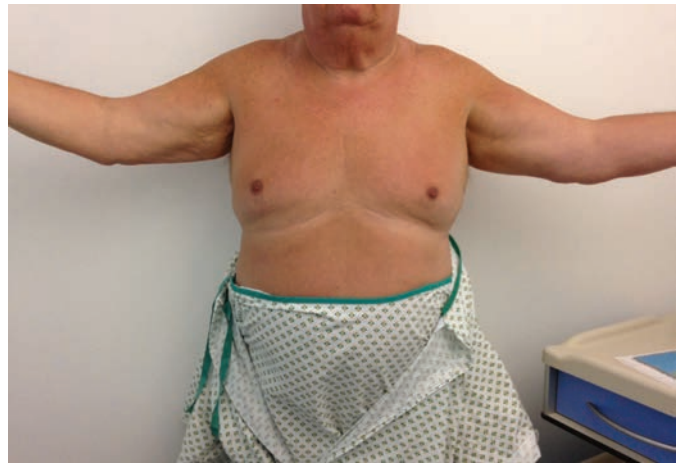

Figure 8.5 Fixed flexed deformity of 40°

Osteoarthritis elbow

EXAMINER: This is Mr Simpson, he is a 60-year-old truck driver who is having problems with his dominant (right) elbow. Could you please examine his elbow?

CANDIDATE: Mr Simpson holds his right elbow in an attitude of 45° flexion. There is a suggestion of soft-tissue swelling around the elbow, especially around the lateral and posterior aspects. The forearm held in a mid pronation. I can see altogether five small well-healed scars, two on the posterior aspect, two on the medial aspect and one laterally suggestive of previous arthroscopic portal scars. Palpation reveals some tenderness especially over the radiocapitellar joint with crepitus and also a soft-tissue effusion as revealed by a fluctuant swelling in the 'soft spot' laterally. The passive range of elbow movement is between 40° and 90° (Figures 8.4 and 8.5). Active movement was only marginally increased and this was associated with some pain in the terminal range of movement. Any attempt at further passive movement causes elbow discomfort. Supination and pronation was 60° bilaterally. There was no obvious instability associated with this range of movements and no distal neurovascular deficit in particular he has normal motor and sensory ulnar nerve function.

EXAMINER: What is the crepitus due to? *(The examiner places his hand on the back of elbow and asks the patient to move.)*

CANDIDATE: It could be due to a loose body or generalized osteoarthritis within the joint.

EXAMINER: Yes, you can actually feel a loose body mobile apart from the joint! OK, what is your diagnosis, given this patient has no history of trauma to the elbow, is fit and well, a hard-working plumber all his life?

CANDIDATE: Osteroarthritis elbow.

EXAMINER: Take a look his x-rays.

CANDIDATE: AP and lateral radiographs of the elbow show multiple osteophytes along medial aspect of olecranon and olecranon fossa, with a loose body just above the olecranon posteriorly.

EXAMINER: This gentleman had some loose bodies removed from his elbow 5 years ago with a successful outcome. However, he has been struggling with his work for the last 1year and recently had difficulty with his personal care, such shaving, washing his face, etc. He is keen to go back to work as soon as possible. What do you advise?

CANDIDATE: Given his symptoms and high demands of his job, I would recommend arthroscopy and removal of loose bodies with additional osteocapsular release, ideally performed by an experienced elbow arthroscopist. Open procedures such as the Outerbridge and Kashiwagi (OK) procedure allow removal of loose bodies and excision of impinging osteophytes at the extremes of motion. I would explain about the possibility of recurrence as well as risks such as neurovascular damage especially with arthroscopic interventions.

This is fairly short common case, either primary or secondary osteroarthritis.

Primary osteroarthritis (OA)

This is often in the dominant elbow of middle-aged men involved in heavy manual labour. *Loss of motion* is the commonest presenting feature, generally patients maintain a functional range of motion in flexion and extension, forearm rotation is less frequently involved. Pain is mainly aching in nature, unless occurring with episodes of locking when a more acute sharp localized pain is described. Pain is more common in terminal extension than terminal flexion because of impingement. In advanced cases, pain can be constant and even at rest and through the whole range of motion. Locking of the joint can be episodic and associated with acutely painful flare ups.

Always look for ulnar neuritis, as osteophytes tend to impinge into the cubital tunnel.

Secondary OA

This can occur in both sexes. Causes include trauma, infection, bleeding disorders, neuropathic diseases, osteochondritis dissecans.

Radiographs show osteophytes at the tip of olecranon and coronoid processes. The olecranon and coronoid fossae also demonstrate ossification and osteophytes. Reduction in the joint space and loose bodies may also be present(Figure 8.6 a and b).

A CT scan, especially three-dimensional, is useful to identify all potential osteophytes needing debridement. Nerve conduction studies may help to confirm an ulnar neuritis.

Treatment

Non-operative management

This includes analgesics, life-style modification, splinting, physiotherapy and viscosupplementation.

Operative interventions

1. **Arthroscopic surgery**

   Arthroscopic removal of loose bodies, excision of osteophytes and release of capsular contractures to improve motion, relieve pain and reduce locking episodes. Arthroscopic radial head excision in selected cases with lateral elbow pain secondary to radiocapitellar arthritis may be indicated
2. **Open procedures**

   Outerbridge–Kashiwagi procedure. A posterior triceps splitting approach, the capsule is incised. Loose bodies removed and ostephytes around olecranon excised. A fenestration made in the floor of the olecranon fossa providing an opening into the anterior compartment of the elbow. Loose bodies can then be removed from anterior compartment. Osteophytes around the coronoid process and coronoid fossa can be excised using a Kerrison rongeur. A partial release of the anterior capsule can be performed through this bony window

   Column procedure. Some patients with OA of the elbow present predominantly with symptoms of loss of motion. They develop progressive loss of extension with a reasonably pain-free mid arc of motion. This is due to a predominantly extrinsic contracture involving periarticular capsule ligamentous structures. The column procedure is useful in such patients in order to gain a functional range of motion especially of the extension deficit. The procedure involves a lateral Kocher incision with elevation of the brachiradialis and ECRB in order to gain exposure to the anterior aspect of the joint. The anterior capsule is then excised with removal of loose bodies and osteophytes. The triceps is then elevated to gain access to the posterior aspect of the joint and a similar procedure is repeated posteriorly

   Interposition arthroplasty. This procedure involves reshaping the distal humerus and proximal ulna, interposition of a membrane between the elbow joint surfaces, and suturing it to the humeral side. Skin, fascia, and Achilles tendon allograft are some of the materials used to interpose between the re-shaped joint surfaces. The collateral ligaments are either preserved or reconstructed and a unilateral hinged fixator may be used to keep the joint slightly distracted and to allow early ROM. This a good salvage procedure for young active patients with severe inflammatory or post-traumatic arthritis, especially with limited elbow motion. The procedure may lead to problems with instability and, therefore, is not be suitable for heavy manual workers

Total elbow replacement (TER). Ideally suited for inflammatory arthritis. It is recommended that after TER patients do not

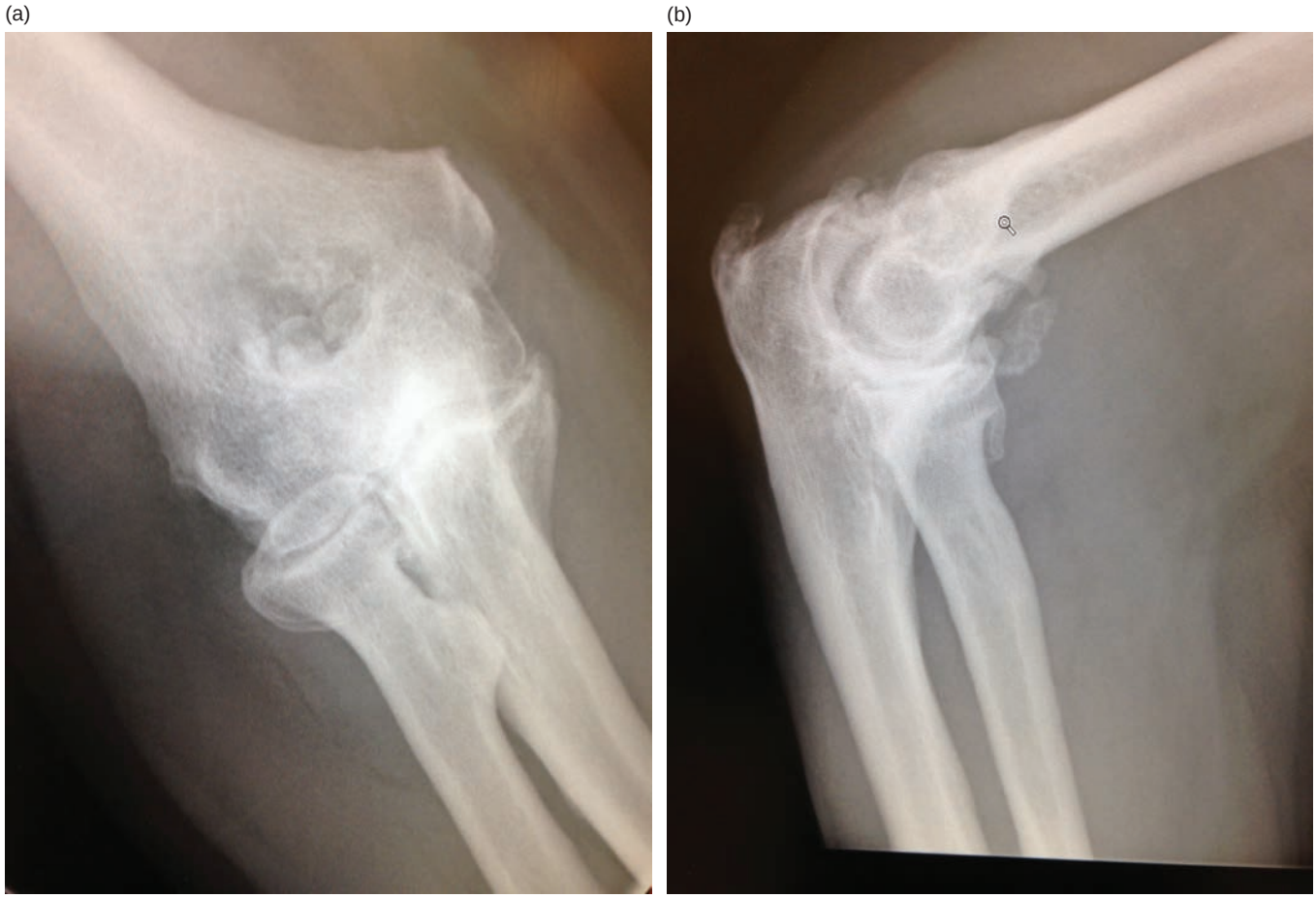

Figure 8.6 AP (a) and lateral (b) radiographs demonstrating advanced OA elbow

lift >4–5 kg with the operated arm as a single event or >1 kg repeatedly. Currently, TER is only indicated in patients with primary OA of the elbow who are older than 65 years of age (this is not absolute), have low activity levels, experience pain throughout range of motion or who have substantial deficits in motion in whom all other interventions have failed. Compliance with postoperative restrictions and life style is essential.

The OK procedure and arthroscopic osteophyte excision and capsular release for osteoarthrosis are now common procedures(Figure 8.7 a and b).

Candidates must be familiar with x-ray appearances of an elbow following such procedures (especially the OK procedure with a fenestration in the olecranon fossa in both AP and lateral views). Candidates should also be familiar with the x-ray appearances of a lateral elbow replacement and total elbow replacements.

Examination corner

Short case

1. Cubitus varus (adult)
   a. Describe deformity and old lateral scars
   b. Neuro exam (nothing)
   c. Function problems (nil)
   d. What other problems would he have – Wanted instability, got there eventually
   e. X-ray of recent posterior dislocation, hence, instability!
   f. Clinical testing for varus/valgus instability including posterolateral pivot shift

Painful elbow with previous surgery for cubital tunnel syndrome

Often in the exam, candidates may come across a case of ulnar nerve dysfunction with or without an old surgical scar over medial aspect of elbow. Apart from possible previous surgery for cubital tunnel release, the scar may be secondary to a previous fracture fixation (i.e. medial epicondyle), a MCL repair with ulnar nerve injury, a delayed ulnar nerve dysfunction secondary to callus formation or an angular deformity such as cubital valgus especially in children.

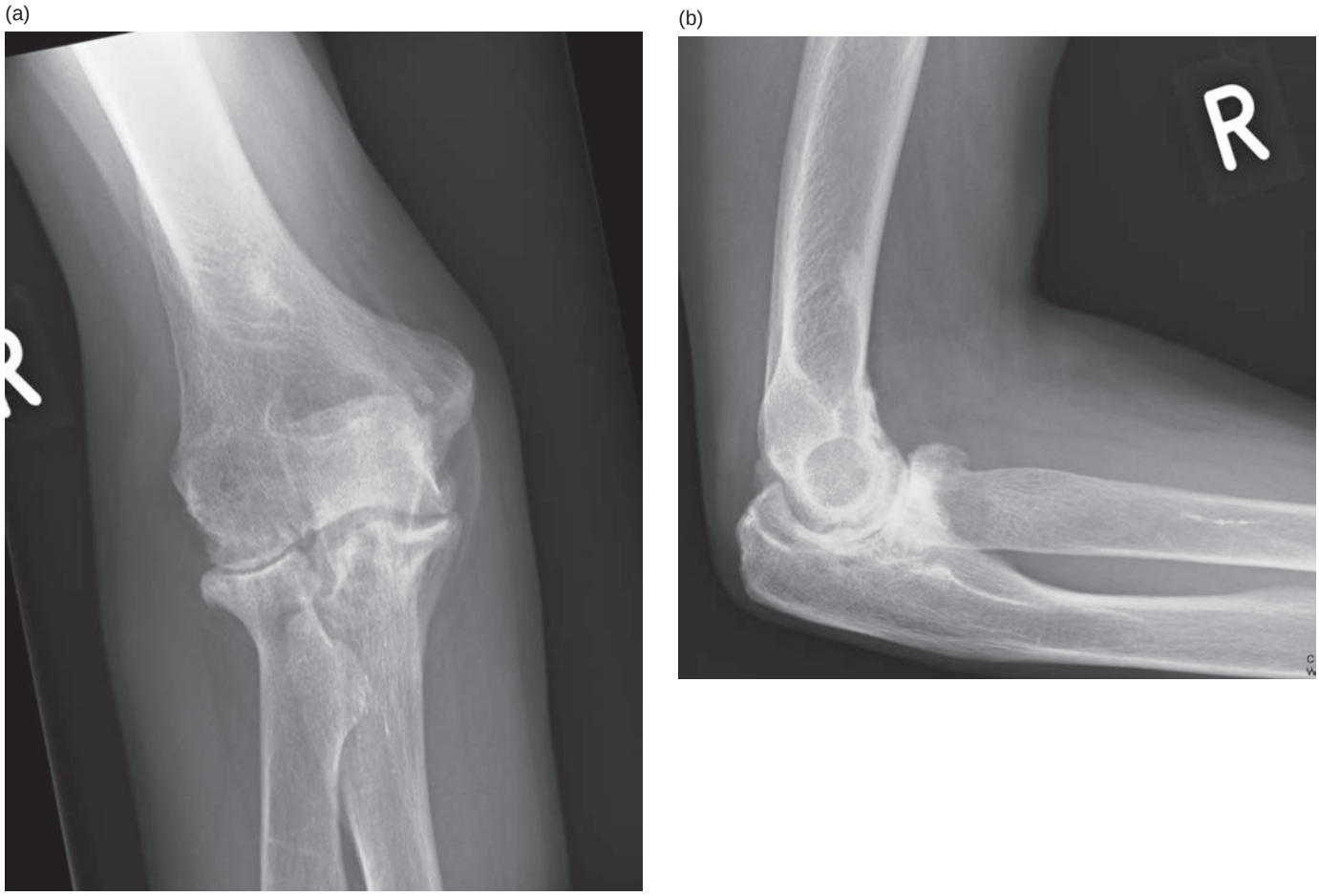

Figure 8.7 AP (a) and lateral (b) radiographs elbow after arthroscopic osteocapsular release for advanced OA elbow

Distal biceps rupture

This is usually a short case and may be an acute or chronic rupture. An acute week-old rupture has been known to be brought up the examination hall 2 days or so before planned surgery.

The examination should also include hook test, lag test and squeeze test as part of look, feel and move. Also important to determine the loss of strength in supination and elbow flexion when compared to opposite arm (very accurate in clinical practice –rotate the distal forearm rather than the hand to avoid compensating by wrist motion). Distinguish between long head of biceps rupture and distal biceps tendon avulsion, based on direction of retraction of muscle belly.

Discussion would be on the timing of operation, approach, one and two incision techniques, postoperative rehabilitation, delayed presentation and grafts that can used, etc.

Non-union, pseudoarthrosis of distal humeral fracture

EXAMINER: Please examine this woman's left elbow.

CANDIDATE: *(After introduction)* Mrs Parkinson's left elbow appears to be straight with some swelling/fullness of the elbow obliterating the normal fossae around the elbow. There is a 5-cm well-healed longitudinal surgical scar over the lateral aspect of elbow. The carrying angle is less when compared to opposite elbow. There is no tenderness around the elbow with palpation, the relationship between the three bony points around the elbow appears well maintained. She can freely flex to 90° and there is some degree of hyperextension by about 10–15° when compared to the opposite side. There is some bony crepitus when testing for ROM and instability that was not associated with any major discomfort. The composite movement of the shoulder, elbow and hand appears to be intact and functional. No distal neurovascular problems were noted with ulnar, radial and median nerve functioning well. Varus and valgus stressing of the elbow in 30° of flexion suggested a degree of elbow instability but with no obvious associated pain . . .

EXAMINER: What do you think Mrs Parkinson's elbow range of movements are due to?

CANDIDATE: Given the clinical findings, with an associated scar, slightly obliquely placed, I suspect the condition is post-traumatic with resulting instability either due to a combination of fractures and ligaments injuries such as terrible trial or non-union of a distal humeral fracture. In reality, I would like to take a thorough history and perform complete neurological assessment to rule out Charcot joint as well given the free and painless ROM, swelling around the elbow and some bony crepitus

EXAMINER: Have a look at the x-rays.

CANDIDATE: AP and lateral elbow radiographs demonstrate a transverse fracture pattern in the distal metaphyseal portion of humerus with signs of non-union and exuberant callus. The distal fragment is in slight recurvatum. The elbow joint itself shows gross OA changes with reduced joint space with multiple osteophytes.

EXAMINER: Yes, a typical fracture in an OA elbow will predictably go onto a non-union with or without operation intervention. What would you like to do?

CANDIDATE: I would like a full assessment including the main symptoms from the elbow and any functional disability. It appears from examination, that Mrs Parkinson has a good and painless functional ROM and if her activities involve low demands on a non-dominant side, I would advise no intervention. If on the other hand any functional problems were associated with significant pain, I would then consider TEA but once again this would only be suitable for low-demand activities. Mrs Parkinson's radiographs reveal a particularly low fracture in the distal humerus with doubtful collateral ligament functional integrity; therefore, a semi-constrained hinge arthroplasty would be needed in her case.

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Chapter 9

Hand and wrist clinical cases

John E. D. Wright and John W. K. Harrison

Introduction

To confidently approach the hand and wrist clinical cases you need two main examination approaches. The first is to deal with the case that on first inspection looks normal; this is in contrast to the case where the pathology is glaringly obvious – For example, advanced Dupuytren's disease. A structured general screening when asked to examine a hand or wrist should lead you swiftly to the underlying condition. You can then focus your examination. Classic cases where the diagnosis is immediately obvious, such as Dupuytren's and rheumatoid arthritis, should be ones you relish and can score highly on.

A crucial part of hand examination is an assessment of function. A brief confident assessment of hand function can be very impressive and can put you at ease at the start of a complex case. You should have a key, coin and pen in your pocket to complete this.

Common cases

The following upper limb cases are likely to appear (not in order of frequency):

- Arthritis – Rheumatoid hand, psoriatic arthritis, osteoarthritis
- Nerve lesions – Ulnar nerve, radial nerve, median nerve
- Dupuytren's disease
- Swelling – Ganglion, giant cell tumour, lipoma, carpal boss
- Tendon rupture – Extensor pollicis longus (EPL), Vaughan–Jackson syndrome, Mannerfelt lesion
- Intrinsic minus hands (Charcot–Marie–Tooth)
- Stiff finger – Trigger finger, volar plate contracture
- Kienböck's
- Congenital – Brachydactly, cleft hand, Madelung's deformity

Examination of the hand

When asked to examine a hand or wrist it is important to expose to above the elbows. An initial screening test allows for identification of most pathology and looks impressive. The skill is to know when to leave the general screening and focus in on a specific pathology. In complex cases and advanced rheumatoid arthritis an early functional assessment is a good way to get started.

Screening examination

Ask the patient to place their hands in front of them with their elbows by their side. Get them to copy what you do. In pronation, ask them to make a fist and open their fingers, then ask them to fully supinate and again make a fist. Then ask them to raise their hands above shoulder height to show you the ulna border keeping their fingers straight. All the time you are inspecting for clues to the diagnosis, such as scars, swellings and asymmetrical movements.

Look

Splints

Elbow – Cubitus valgus, rheumatoid nodules, cubital tunnel scar

Forearm – FCU wasting, scars – Nerve decompressions, fracture plating

Fingers – clawing, boutonnière deformity, swan-neck deformity, mallet finger deformity, Wartenberg's sign

Nails – clubbing, pitting

Muscle wasting – Hypothenar, thenar, interossei – Guttering

Swellings – Ganglia, giant cell tumour (GCT), Dupuytren's, rheumatoid nodules

Congenital – Camptodactyly, clinodactyly (Figure 9.1 a and b), polydactyly, syndactyly

Feel

Ask where it is tender
Feel for swellings and tenderness
Feel palm for nodules

Move

Mass movement (make fist) – Look for speed, smoothness and symmetry

Digits – MCP joint 90°, PIP joint 100°, DIP joint 70°, 'tip-to-palm' distance

Thumb – Opposition, flexion, abduction, adduction, retropulsion (lift thumb with palm flat on table)

EDC/EI/EDQ – extend MCP joints

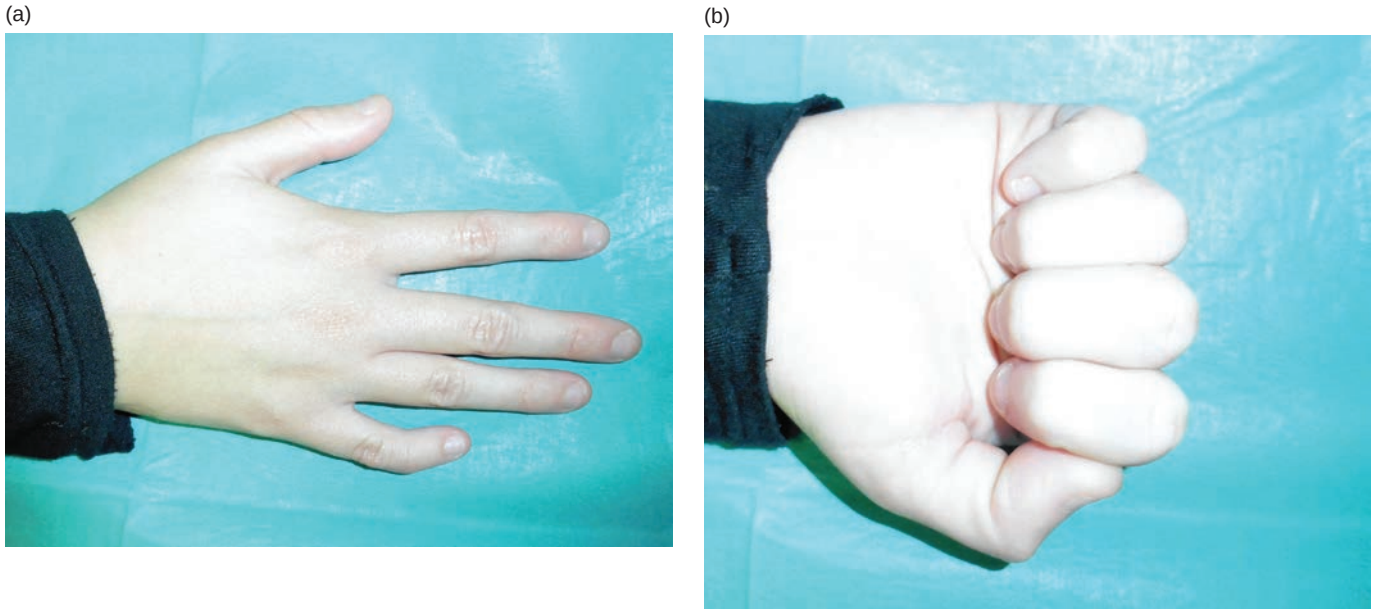


Figure 9.1 Clinodactyly. In this case a uniplanar deformity (a) which corrects on finger flexion (b)

Interossei – 'DAB & PAD' (mnemonic for Dorsal ABduct, Palmar ADduct)

Quadriga effect – Middle to little finger FDP have common muscle belly; flexion of other digits is limited by the shortening of an injured or repaired FDP

Functional assessment

Preparation is vital. Have a key, a pen and a coin easily available.

Ask them to hold the key (key), pick up the coin from your palm (tripod), hold the pen (tip to tip) and then grasp your forearm (power).

Grip (6) – Power – Cylindrical, spherical, hook

precision – Tripod, key (thumb to side index), fine – (tip to tip)

Neurology

Sensation

Median – Index finger pulp/thenar eminence (superficial sensory branch)

Ulnar – Little finger pulp/dorsum fifth metacarpal (dorsal sensory branch)

Radial – Dorsum first web space

Dermatomes – C6 – Thumb, C7 – Middle finger, C8 – Little finger

Motor

Median – FCR, FDS, APB

Anterior interosseous nerve – Supplies FDP (index), FPL, 'OK' sign (Figure 9.2)

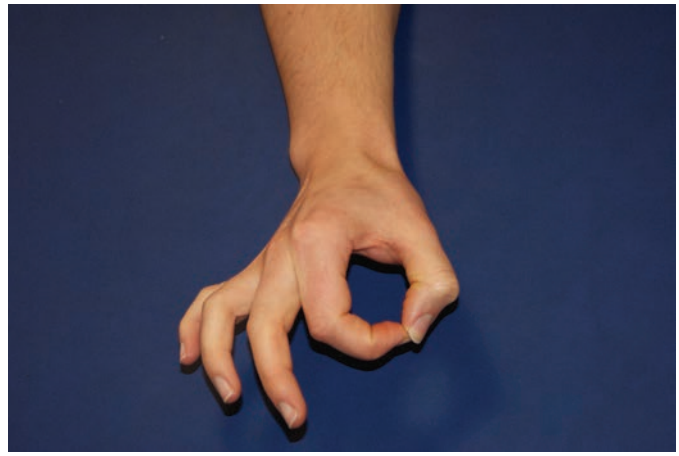


Figure 9.2 'OK' sign – Testing anterior interosseous nerve

Ulnar – FCU, FDP (little finger), ADM, first dorsal interosseous, Froment's test

Radial – brachioradialis, ECRL – Posterior interosseous nerve – ECU, EI, EPL

Pulses

Allen's test – For intact palmar arch. Make fist, compress radial and ulnar arteries at wrist. Relax fingers. Release over one pulse to see if hand reperfuses.

Special tests

Flexor tendons

FDP – Test each individually, resisted DIPJ flexion with PIPJ held extended

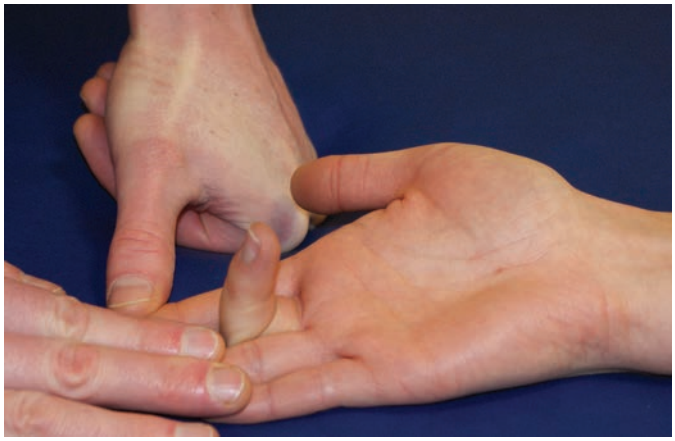


Figure 9.3 Testing for FDS

FDS – Test by holding neighbouring digits extended (to exclude FDP) (Figure 9.3). Index finger has a separate FDP so keep DIPJ extended while testing FDS. FDS to little finger is absent in 10% of people

FPL – Resist IPJ flexion of the thumb with the MCP joint held extended

Tenodesis test (for intact extrinsics)

Passive flexion wrist causes MCP joints to extend, then passively extend wrist and MCP joints flex.

Intrinsics (lumbricals and interossei)

Intrinsic muscles actively flex MCP joints and extend IP joints by direct action across the joints. Extrinsic muscles actively extend the MCP joints and flex the IP joints.

Plus deformity = MCP joints flexed, IP joints extended
Minus deformity = MCP joints hyperextended, IP joints flexed

1. Bunnell–Littler test (for intrinsic tightness). Hyperextend MCP joint. If cannot flex IP joint = intrinsic tightness or tight capsule, so flex MCP joint; if still tight = joint contracture
2. Intrinsic vs extrinsic flexor tightness – Flex wrist (this relaxes long flexors); if you can flex IP joints = tight intrinsics
3. Bouvier's test (to determine in intrinsic minus hand if the extensor mechanism is working normally). Blocking hyperextension MCP joints = allows extension of IP joints by EDC
4. Lumbrical plus finger. Paradoxical IP joint extension on attempted finger flexion. Due to laceration of FDP distal to the origin of the lumbrical

Central slip extensor tendon

Elson's test (acute injury) – Flex PIPJ over the table edge. Resisted PIPJ extension is weak and the DIPJ hyperextends = rupture of the central slip (PIPJ extends indirectly due to pull of lateral bands)

Boyes' test (chronic) – Hold PIPJ hyperextended; there is failure of DIPJ flexion if the central slip is retracted and adherent.

Examination of the wrist

Initial inspection is often unhelpful in wrist cases, with palpation and special tests being more revealing. Potential diagnoses can be grouped into radial sided, central or ulna sided. The order of look, move, feel and special tests is suggested.

- Four articulations (DRUJ, radiocarpal, mid-carpal, carpometacarpal)

Radial side wrist pain (RSWP)

- Tendon entrapments
  - De Quervain's (Finkelstein's test)
  - Intersection syndrome
- Arthritis
  - First carpometacarpal joint arthritis (grind and distraction–relocation tests)
  - STT joint arthritis (pronate wrist against resistance)
  - Radioscaphoid arthritis
- Scaphoid non-union
- Wartenberg's neuritis (entrapment of the superficial branch of the radial nerve deep to brachioradialis)

Central

Kienböck's
Ganglion – Related to scapholunate ligament
Carpal boss
Scapholunate advanced collapsed (SLAC) wrist

Ulnar side wrist pain (USWP)

Distal radioulnar joint (DRUJ)
Triangular fibrocartilage complex (TFCC) tear
Ulnar impaction
Lunotriquetral instability
Extensor carpi ulnaris (ECU) – Tendonitis/instability
Pisotriquetral OA

Look

Splints
Nails – Clubbing, pitting
Deformity – Congenital (Madelung's), distal radius mal-union, thumb base 'squared off'
Swellings – Ganglion, prominent ulnar head – Caput ulnae, synovitis
Scars

Dorsal – Midline/transverse/arthroscopy/first extensor compartment
Volar – Scaphoid surgery/carpal tunnel release/radial (ganglion)

Move

Observe both wrists together, ask the patient to press their palms together to assess wrist dorsiflexion, then the back of the hands together to assess palmer flexion. In pronation assess radial and ulna deviation and finally assess supination.

Look for subtle asymmetry to suggest pathology in the wrist with reduced movement.

Feel

Have a logical sequence starting dorsal and then volar, palpating specific anatomic sites (NB. Lister's tubercle is most easily palpable bony landmark on dorsum of wrist.):

First extensor compartment, ASB, SLL (1 cm distal to Lister's tubercle), DRUJ, ECU tendon, TFCC (foveal soft spot), pisiform, hook hamate, median nerve, FCR, first CMC joint

Special tests – Provocative and instability

Finkelstein's test – Ask the patient to place thumb across palm, then wrap fingers around thumb, finally gently ulnarly deviate the wrist. Take care this is painful!
TFCC tear – Ulna deviate the wrist, and compress and rotate hand
DRUJ – Compress midshaft radius and ulna, and rotate forearm. 'Piano keys' test
Pisotriquetral joint – Palpate for using index finger tip, compress and move radial and ulna
Scapholunate instability

- Kirk–Watson's test – 'Arm wrestling position' – Patient's elbow resting on table and flexed 90°. Examiner's thumb over scaphoid tubercle, index finger over SLL dorsally, examiner's other hand around metacarpals. As moving patient's hand from ulnar to radial deviation, exert pressure with the thumb to prevent scaphoid flexing. Positive test if there's click or pain. Compare to opposite (20% positive in normal)
- Scaphoid thrust test – Similar to above but with more rapid 'thrusting;' of the scaphoid which is felt to move dorsally

Midcarpal instability – Hold forearm and hand, with thumb on dorsum of capitate and pressing volarwards, as wrist is ulnarly deviated a clunk is felt
Lunotriquetral instability (volar intercalated segment instability (VISI) deformity on radiographs) – Reagan's ballottement. Pain and laxity felt on dorsal/volar stressing of the lunate

Rheumatoid hand and wrist

The medical management of rheumatoid arthritis has significantly reduced the requirement for orthopaedic treatment. However, this is still a frequent case in exams and may be an intermediate case. These patients can have pain and the exam is essentially passed by description and a good functional assessment. The appearance of an established rheumatoid hand is very typical and usually easy to identify, the difficulty lies in how to proceed with the examination.

Standard screening assessment allows initial diagnosis to be made, and with arm elevation inspection of the elbows for scars and nodules, plus importantly to comment on the shoulder and elbow function. Performing a hand functional assessment before addressing the specific deformities can be an easier way to proceed.

One strategy is a phrase such as – 'On inspection I can see this patient has bilateral, symmetrical, polyarthropathy suggestive of inflammatory arthritis. It is most likely to be rheumatoid arthritis. I would first like to perform a functional assessment before looking at the specific deformities.'

History

Pain – Site, severity, night pain
Weakness
Paraesthesia
Neck symptoms – Neck pain, radicular pain, myelopathy
Previous surgery
Function
Activities of daily living:

- Shop independently
- Stairs
- Dressing (buttons)
- Washing (face, hair)
- Eating

Previous medical history (and DVT)
Medications (and allergies)
Social – Smoking, alcohol, job, hobbies, partner, stairs
Family history

Examination

Perform screening assessment of hands, elbow and shoulder. State the diagnosis.

Look

Swelling over the dorsum of the wrist (tenosynovitis ± caput ulnae). Caput ulnae – As carpus volar subluxed and supinated. ECU volar-ulnar subluxed, metacarpals radially angulate, swellings over MCP joints, volar-ulnar subluxation of MCP joints, dropped fingers, swan-neck and boutonnière deformities of the digits, Z-thumb, palmar erythema, muscle wasting.

The three most common rheumatoid scars – Wrist arthrodesis, MCP joint replacements, thumb MCP joint fusion.

Move

Mass movement – Ask the patient to make and open a fist.

Functional assessment

- Pick up a coin (tripod pinch)
- Hold a pen (end pinch)
- Hold a key (side pinch)
- Power – Grip around your wrist (grip strength)

Feel

Tenosynovitis; feel the digits to gauge whether they are floppy (arthritis mutilans), and the subcutaneous border ulnar for nodules. Identify MCP joint subluxation. Sensation specific to carpal tunnel syndrome.

Specific lesions

Dropped fingers (NB. IPJ extension due to lumbricals)

Causes

Vaughan–Jackson – Ruptured EDM/EDC, tenodesis test

Ulnar subluxed extensor tendons (sagittal band rupture)

- Finger extension maintained if MCP joint passively extended and tendon centrally relocated
- Posterior interosseous nerve (PIN) palsy – Tenodesis test, radial deviation (ECRL) on wrist extension
- Dislocated MCP joints – Reduce and take radiographs

Locked trigger finger

Mannerfelt lesion

Ruptured FPL by attrition on scaphoid bone spur causes loss of thumb IPJ flexion (treatment: FDS tendon transfer or fuse IPJ).

Differential diagnosis is anterior interosseous nerve (AIN) palsy or trigger thumb.

Memorandum

'This patient has features of a symmetrical polyarthropathy of the small joints of the hand typical of rheumatoid arthritis. There are swellings over the dorsum of both wrists and the MCP joint. There is spindling of the fingers due to soft-tissue swelling at the PIP joints and MCP joints but the DIP joints are spared.'

'There is significant restriction of elbow and shoulder movement.'

'Functional assessment reveals weakness of grip strength, but they have functional key, end and tripod pinch.'

'On closer inspection I can see ulnar deviation of the fingers at the MCP joints, volar subluxation of the MCP joints, radial deviation of the wrists and a prominent ulnar head. There is a Z-deformity of the thumb, swan-neck deformity of the left little and ring fingers.'

'There is drooping of the right little and ring fingers suggestive of possible long extensor tendon rupture[a]. I can see scars of previous surgery over the wrist[b], thumb[c] and fingers[d]. There are boutonnière deformities of the index and middle fingers. There are firm subcutaneous nodules at the elbow, over the extensor tendons and in the palm. The flexor aspects of the fingers appear bulky due to chronic synovitis. There is wasting of the small muscles of the hand. There is thin bruised skin; pale nail beds and nail fold infarcts are present. There is no nail pitting or scaly rash seen[e]. In the palms I am looking for pallor in the palmar creases indicating anaemia[f] and palmar erythema[g].'

Examiner questions

- Can you explain the reasons for the ulna deviation of the digits?
- What causes a caput ulnae deformity?
- What are the important functions of the hand?
- What is a boutonnière deformity?
- What is a rheumatoid nodule?

'Are your hands painful?'

'I would now like to palpate the hands feeling for any areas of tenderness, boggy swellings[h]. There is evidence of bony destruction of the PIP and MCP joints with sparing of the DIP joints. I cannot feel any rheumatoid nodules or Heberden's nodules in the hand. None of the joints is tender or warm at present.'

'Can you make a fist and now straighten your fingers and thumb?'[i]

'There is limitation of flexion and extension of all digits. PIP joint deformities are only partly correctable. There is no extensor tendon rupture. This patient has severe rheumatoid disease affecting both hands. I would like now to assess function of the hands and review radiographs of the hand. To complete my examination I would like to examine the cervical spine and look for extra-articular manifestations of rheumatoid arthritis.'

[a] Dropped finger suggests tendon rupture but remember dropped fingers may also be a result of tendon subluxation, joint subluxation or dislocation, PIN palsy or flexor contracture produced by intrinsic tightness.

[b] Usually arthrodesis of the wrist for strength and stability.

[c] Usually arthrodesis of the MCP joint for strength and stability.

[d] Usually silastic joint replacements for movement and pain relief.

[e] Psoriatic arthropathy is an asymmetrical arthropathy involving mainly the DIP joints with pitting of the finger nails and hyperkeratosis. There is a red, scaly rash over extensor surfaces or scalp.

[f] There are five main causes of anaemia in rheumatoid arthritis: Anaemia of chronic disease, GI bleed, bone marrow suppression, associated with pernicious anaemia and Felty's syndrome.

[g] Redness around the palm sparing the central area is associated with rheumatoid arthritis, pregnancy and liver disease.

[h] Talk to the examiners – Tell them what you are doing as you go along. Do not let the examiners assume/think that because you are not saying anything you know nothing. It is useful to communicate your findings to the examiners at each stage.

[i] Try not to get bogged down in describing one abnormality in the hand; work through problems systematically and be guided by the examiner as to what they specifically want you to concentrate on, particularly with special tests.

Other possible features

- **Carpal tunnel syndrome:** Caused by flexor synovitis. Check for thenar muscle wasting and test the power of abductor pollicis brevis (APB). Don't miss the scar from previous decompression
- **Triggering of digits:** Secondary to tenosynovitis. May cause loss of flexion especially in the thumb
- **Palmar erythema:** Non-specific change indicative of a hyperdynamic circulation
- **Arterial pulses: D**o Allen's test
- **PIN palsy:** May occur at the elbow in rheumatoid patients due to anterior dislocation of the radial head. The patient will present with an inability to extend the fingers and thumb although the wrist can be extended, albeit into radial deviation. (Radial nerve innervates ECRL.) The differential diagnoses are rupture of the extensor tendons at the wrist, ulna subluxation of extensor tendons at MCP joint, subluxation/dislocation of MCP joint or flexor contracture secondary to intrinsic tightness. Tenodesis test will differentiate between ruptured or intact extensor tendons. If the extensor tendons are intact the fingers will passively extend upon wrist flexion. If the extensor tendons are ruptured the fingers will not extend. Management depends on symptoms, functional assessment and any previous or ongoing treatment

Examination corner

Short case 1: Florid rheumatoid hands, elderly female
Time spent discussing clinical features, assessment, management, etc.

Short case 2: Rheumatoid hands
General discussion and classification (Nalebuff) of the classic deformities seen. Patient also had scar from previous elbow arthroplasty.

Short case 3: Atlanto-axial subluxation
The candidate was asked to examine a woman's hand. Clinical features were of rheumatoid hands. The examiners did not want a description, only the spot diagnosis. The examiner then asked the candidate, 'Why is she wearing a cervical collar?'

The candidate, who had noticed it but not mentioned it, said it could be because of atlanto-axial subluxation.

Short case 4: Extensor tenosynovitis in the rheumatoid hand

- Diagnosis
- Differential diagnosis
- Complications
- Tendon rupture and caput ulnae
- Principles of tendon reconstruction in a rheumatoid hand

Short case 5: Rheumatoid hand after a Swanson MCP joint replacement operation
'How would you perform the operation?'

Short case 6: Young rheumatoid female. Right wrist fused. Left wrist replaced – Discuss
Performing bilateral wrist arthrodeses in patients with inflammatory arthritis is controversial. Patients with bilateral wrist fusions are believed to have less dexterity and greater functional compromise than those with one wrist fused and arthroplasty of the other. There is disagreement as to which wrist should be fused. Arthrodesis of the non-dominant hand and arthroplasty of the dominant hand is generally recommended.

Dupuytren's disease (DD)

Commonly occurs in the short cases and hand oral. Pattern recognition is important – A Dupuytren's hand can easily be summarized in a few lines. Don't forget to say the diagnosis early in your examination if it is obvious.

Take care not to diagnose a fixed flexion deformity on inspection; it may be passively correctable on examination. Cords can be very minimal but cause significant contracture, requiring careful fingertip palpation to identify them. Conversely if you can't feel any cords in a fixed flexed finger think of another diagnosis.

Differential diagnosis of DD

- Locked trigger finger
- Camptodactyly
- Skin contractures (secondary to burns or scarring)
- PIP joint volar plate contracture
- Tendon contracture (thickened cord moves on passive flexion of the finger)
- Arthrodesed joint

History

- Age, hand dominance
- Age of onset of the disease
- Rate of progression of the disease
- Functional deficit: Difficulty putting hand in pocket, washing face, wearing gloves,
- Foot or penile involvement
- Family history
- Previous medical history: Diabetes, epilepsy, alcohol, smoking, trauma
- Occupation, hobbies
- Previous hand surgery

Inspection

Look

Pits (Dupuytren's inserting vertically into the skin) and nodules – Early changes

Cords – Pretendinous (Figure 9.4), natatory, lateral, spiral, abductor digiti minimi, commissural

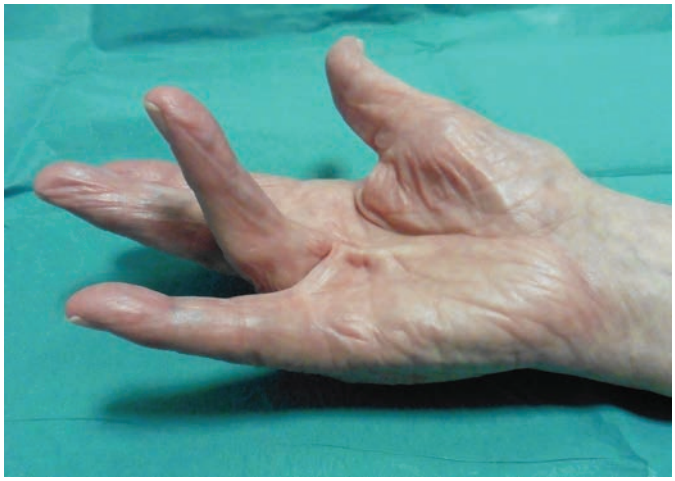


Figure 9.4 Pretendinous cord causing a MCP joint contracture. Assess for PIP joint contracture with MCP joint flexed fully. If skin is mobile over the cord, this can be treated with percutaneous needle fasciotomy or collagenase injection

Scars – Brunner, Z-plasty, amputation!

Dermofasciectomy – Look for hairs in graft and scar for donor site

Garrods pads – Thickening on dorsum of proximal interphalangeal joints

Ask the patient to straighten out their fingers, palms up.

> 'The hand is held with a flexed posture to the little and ring finger at the MCP and PIP joints. There are skin pits and nodules present in the palm. There are no obvious scars suggestive of previous surgery present in either the palm or fingers.'

Ask the patient to flex their fingers fully.

Feel

Palpate with your index finger across the palm and then distally following any cords identified.

> 'There is an abductor cord to the little and a pretendinous cord to the ring finger causing contractures at the MCP and PIP joints. There are no Garrod's pads present'[j].

Measure

As the cords cross more than one joint, flex the PIP joint fully to measure an MCP joint contracture (place a goniometer on the back of the digit), then flex the MCP joint fully to measure a PIP joint contracture. Rarely is the DIP joint involved. The true contracture for each joint can be much less than the apparent contracture on initial inspection.

Sensation and vascularity

It is very important to test for sensation distal to any proposed site of surgery especially if there are scars from previous surgery present (1.5% risk of digital nerve injury for first-time surgery). Assess the circulation with a digital Allen's test (Figure 9.5 – Press either side of the fingertip and milk the blood out proximally to the base. Release pressure on one side and observe if the finger perfuses. Repeat for other digital artery).

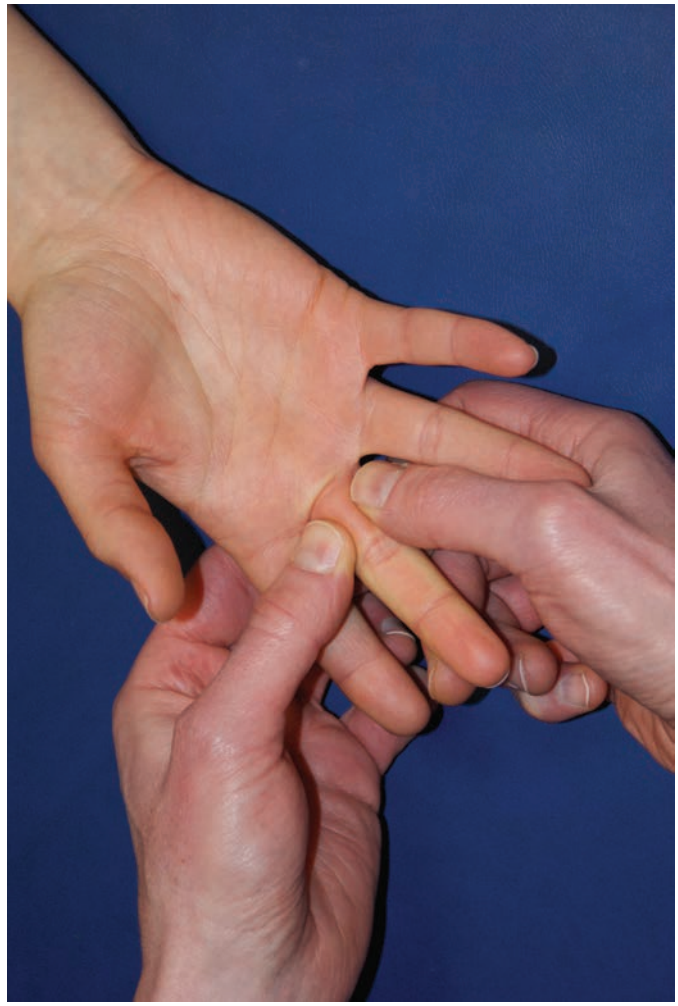


Figure 9.5 Digital Allen's test

EXAMINER: What else would you like to examine?

CANDIDATE: The soles of the feet (Ledderhose's),[k] the dorsal knuckle pads.[10] DD is also associated with Peyronie's disease.[l]

EXAMINER: How would you decide on management?

CANDIDATE: I would perform a Hueston's tabletop test. More precisely I would offer intervention for an MCP joint contracture >30°, or for any significant PIP joint contracture >15°. Other factors such as age, functional deficit, rate of progression and previous surgery are also relevant.

EXAMINER: Consent me for a partial fasciectomy.

[j] The knuckle pads(dorsum proximal interphalangeal joints) (Garrod's pads) can often be thickened.

[k] Can affect the plantar aponeurosis.

[l] Fibrosis of the corpus cavernosum causing curvature of the penis. The examiners are unlikely to expect you to confirm this association!

CANDIDATE: The aim is to excise diseased tissue and restore movement through a zigzag incision. It is performed under general anaesthetic. You will wake up with your hand in a bulky dressing and go home on the same day. A therapist will see you at 48 hours and remove the dressing and commence splinting. Your sutures are taken out at 10–12 days, then you have wound management to soften the skin and splinting at night for 6 months. Surgery is to control the disease and not cure it, and recurrence can occur. Complications include wound infection, haematoma, digital nerve injury (1.5%), stiff hand, reflex sympathetic dystrophy (RSD) and, rarely, amputation.

EXAMINER: Why may a PIP joint contracture not correct fully?

CANDIDATE: The question relates to the position of safety for splinting the hand (wrist extended 20°, MCP joints flexed 90°, IP joints extended fully). Flexion contracture of the PIP joint leads to shortening of the volar plate. An extensive release (check-rein ligaments, sheath, accessory collaterals, ± volar plate) may be needed for a marked contracture (>70°) and this is controversial as it can lead to further scarring, even limiting flexion postoperatively. In the MCP joint, a 90° flexion contracture does not shorten the collaterals due to the cam shape of the metacarpal head, and the joint will always straighten after excision of the Dupuytren's tissue.

EXAMINER: What is the incidence of nerve injury at recurrent surgery?

CANDIDATE: Usually about 1.5% for primary surgery with anything up to 20% reported for recurrent surgery.

Examination corner

Short case 1: Elderly man, bilateral DD

Spot diagnosis
Asked to examine hands and comment on typical features of DD. A few minutes of general discussion about DD
What are the various cords and what are the bands that contribute to each? ('band' is normal, 'cord' is diseased)
Various finger incisions (Brunner's allows excellent exposure of the neurovascular bundles – Z-plasties allow lengthening of the skin)
Role of open palm technique
Diathesis
Recurrence rate

Short case 2: Elderly man, DD right hand

EXAMINER: Would you examine this gentleman's hands, please?

CANDIDATE: On inspection there is a flexed attitude of the little and ring finger of the right hand. Looking at the palm there are cords extending into the little and ring fingers. There are no obvious surgical scars present. 'Can you turn your hands around for me, sir?'

On inspecting the dorsal surface of the hand there are thickenings of skin over the PIP joint knuckles suggestive of Garrod's pads. This gentleman has DD and I would like to assess the degree of flexion contracture of the little and ring fingers.

I took out a goniometer and made a show of measuring angles.

He has a 30° flexor contracture of his little finger MCP joint and 20° of the PIP joint. In the ring finger there is a 20° MCP joint contracture and the PIP joint is minimally affected. There also appears to be a cord running between the thumb and index finger, which I hadn't noticed.

EXAMINER: What do you call that cord?

CANDIDATE: Commissural cord. 'Can you put your hand down flat on the table, please, sir?' The patient is unable to put his hand down flat on the table, the so-called Hueston's tabletop test, indicating that we may need to consider surgery in this gentleman's case. 'Can you feel me touching the side of your finger? Does that feel normal?'

The candidate continued to test digital nerves of each finger whilst the examiners in background were heard to mutter, 'Yes, good'.

CANDIDATE: There is normal sensation present in each digit.

EXAMINER: What are the various bands in the hand?

CANDIDATE: The normal bands in the hand are the longitudinal pretendinous bands, spiral bands, natatory ligaments, Cleland's ligaments, Grayson's ligaments and the lateral digital sheath.

EXAMINER: And what are the diseased cords?

CANDIDATE: Central cord, spiral cord, lateral cord, retrovascular cord and abductor digiti minimi cord.

EXAMINER: Yes, the abductor digiti minimi cord; a lot of people forget about this cord and, as you can see, this gentleman has an abductor digiti minimi cord that should be excised at surgery or else you will not get full correction of the digit.

How are you going to manage this gentleman?

CANDIDATE: I would perform a partial fasciectomy using a Brunner's zigzag incision.

EXAMINER: This patient is listed for surgery next week. What would you be concerned about from an anaesthetic point of view?

CANDIDATE: There is a higher incidence of ischaemic heart disease, chronic pulmonary tuberculosis, chronic lung disease, diabetes and excessive alcohol intake in patients with DD.

EXAMINER: How would you obtain informed consent of the patient?

CANDIDATE: I would mention that surgery is not curative; there may be a recurrence. We are unlikely to achieve full correction of the finger and there is a small possibility of loss of sensation of the digit owing to digital nerve injury. There is also a possibility that the blood supply to the finger can be compromised because of stretching, spasm or division and very occasionally the finger may have to be amputated if the circulation does not recover. The wound can look very alarming postoperatively but this is normal. The hand can become stiff and take several weeks to recover. There is the possibility of a wound haematoma and infection developing in the hand. I would also mention that the hand would need to be splinted at night for several months afterwards to lessen the chance of the deformity recurring.

Short case 3: Elderly man with DD and ring finger MCP joint contracture with isolated palmer cord

Asked to examine hands.

Discussion regarding treatment options. As the cord was well defined in the palm and only causing MCP joint

contracture, treatment options lies between a needle aponeurectomy or collagenase injections. The recent NICE guidelines were explained and supported a needle aponeurectomy. This would achieve good correction, avoid general anaesthetic and the chance of recurrence was less important at the patient's age.

Short case 4: Young man with bilateral DD

- Describe the condition?
- What questions would you ask in the history to evaluate this particular patient for aetiology, prognosis and management?
- Logic of treatment?
- Indication for surgery?
- What are the indications for a dermofasciectomy? What do we mean by 'firebreak'?

Short case 5: DD digital contracture

- Spiral cord components?
- Surgical approach to a spiral cord to avoid damaging the nerve?
- How to release a PIPJ contracture?
- What is Dupuytren's diathesis?

Short case 6: Elderly man with bilateral DD

CANDIDATE: On examination there is DD of both hands with a severe fixed flexion deformity at the PIP joints of the little and ring fingers.

EXAMINER: What do you think?

CANDIDATE:

No Garrod's pads
Positive family history
No ectopic disease
Neglected DD or diathesis.

EXAMINER: What treatment would you offer this patient?

CANDIDATE: My preferred option would be a multidigit partial fasciectomy. I explained this is complex surgery and may require skin grafts from the medial forearm. I would expect a tourniquet time of 2 hours when planning my list. Amputation of the involved digits should certainly be considered although this may be a bit drastic and wouldn't be my first option, but should be discussed with the patient.

Peripheral nerve lesions

These are very common exam cases, often being chronic and painless, but with good clinical signs. The main causes are compression neuropathy or traumatic injury (sometimes iatrogenic!). Knowledge of the anatomy of the brachial plexus, the peripheral nerves and the specific dermatomal and sensory nerve cutaneous supply is essential. A clear understanding of how both motor and sensory testing distally relates to the proximal pathology produces a competent and efficient examination.

History

Age/hand dominance
Occupation/hobbies
Previous trauma/surgery
Co-morbidities
Diabetes/endocrine disorders
Alcohol/smoking
Anatomical and temporal distribution of symptoms
Functional dexterity

Examination

This is the suggested order of examination:

Inspection, sensory testing, palpation, motor testing, provocation tests.

Look – Inspection

Often the limb can look normal on initial inspection. Following the routine screening described earlier will identify a peripheral nerve lesion.

Muscle wasting

Ulnar: Ulna side forearm (FCU/FDP)/hypothenar eminences/first dorsal interosseous/guttering from interossei wasting
Radial: Wasting of radial side of proximal forearm
Median: Thenar wasting – Most radial side specifically abductor pollicis brevis (APB)

NB. Anterior and posterior interosseous nerve lesions show no muscle wasting in the hand.

Attitude

Ulnar: Cubitus valgus, ulna clawing (MCP joint extension and IP joint flexion), Wartenberg's sign (loss third palmar interosseous, with unopposed action of EDM causing little finger abduction)
Radial: Dropped wrist, splints

Scars

Ulnar: Elevate the arms at the shoulders to inspect behind the medial epicondyle for a scar from cubital tunnel release. Traumatic or surgical scar in forearm. Scar from Guyon's canal release over volar aspect wrist
Radial: scar posteriorly over triceps from plating of humerus. Radius plating with scar from anterior approach (Henry) causing superficial radial nerve injury
Median: carpal tunnel scar. Scar radial proximal forearm from pronator syndrome release

Sensation

Ulnar nerve

Test the tip of the little finger and the dorsum of the hand over the fifth metacarpal. This differentiates between a high or low lesion. If the nerve is injured at the elbow sensation will be

lost in both places tested, but if the injury is at the wrist the sensation over the fifth metacarpal will be preserved as the dorsal sensory branch arises 5 cm proximal to the wrist.

Radial nerve

The superficial branch of the radial nerve arises at the level of the elbow and supplies the dorsum of the first web space. The posterior interosseous nerve has no cutaneous distribution.

Median nerve

Test the tip of the middle finger and over the thenar eminence. A lesion above the wrist will cause sensory loss in both areas, but sensation will remain over the thenar eminence if the lesion is in the carpal tunnel. The anterior interosseous nerve has no sensory distribution.

Palpation

Ulnar: differentiate tenderness over the cubital tunnel from over the medial epicondyle (Golfer's elbow)
Radial: differentiate compression over the lateral epicondyle (tennis elbow) from radial tunnel/PIN syndrome which is 5 cm more distal
Median: tenderness over the sites of compression for pronator syndrome in the proximal forearm

Motor testing

It is important to have a logical sequence of muscles tested and to be able to test each part of the peripheral nerves. This allows identification of the level of the injury or dysfunction.

Ulnar: Muscles in forearm – FCU and FDP to little finger (Pollock's test)

Intrinsic muscles – ADM and first dorsal interosseousSpecial test – Froment's

Radial: Main radial nerve – BR and ECRL
Posterior interosseous nerve – ECU, EI and EPL

Median: Muscles in Forearm – FCR and FDS
Intrinsic muscles – APB and OP.

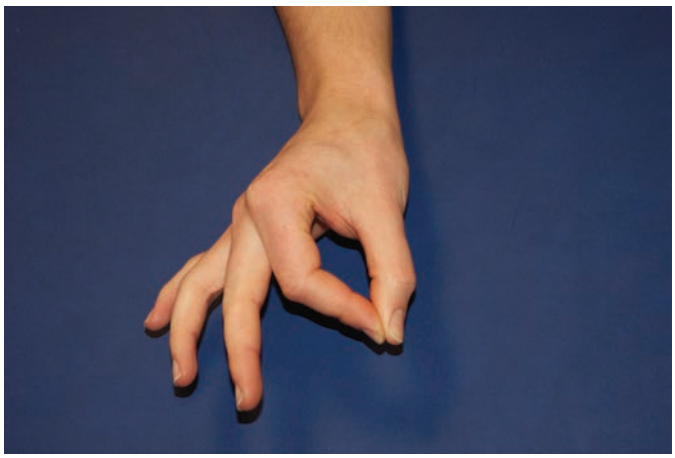


Figure 9.6 Kiloh–Nevin sign (anterior interosseous palsy)

Special test – OK sign (Kiloh–Nevin sign for anterior interosseous nerve palsy) (Figure 9.6)

Provocative testing

These are done last in clinical practice and are unlikely to be performed in the clinical examination, but you should be able to describe them and their significance.

Ulnar: Tinel's test over cubital tunnel and Guyon's canal

Elbow hyperflexion test (Wadsworth)

Median: Tinel's test over carpal tunnel

Phalen's and reverse Phalen's test. McMurtry compression test. Provocation tests for pronator syndrome

Radial: Resisted supination with elbow in extension (compression under arcade of Frohse)

Peripheral nerve injuries (Seddon)	Neuropraxia	Physiological, demyelination
	Axonotmesis	Endoneural tubes in continuity, Wallerian degeneration
	Neurotmesis	Epineurium divided, surgery
Sunderland		Grade I–V (III – Scarring endoneurium, IV – Complete scarring)
Myotome	Muscle mass supplied by a spinal nerve	

Dermatome	Skin area supplied by a spinal nerve
Erb's palsy	Long-standing traction palsy to upper trunk C5/6 Arm internally rotated (suprascapular nerve)/ elbow extended/forearm pronated/wrist-digits flexed
Klumpke's palsy	Claw hand, decreased sensation medial arm (C8, T1)
Claw hand	Combined median/ulnar nerve palsy, rheumatoid arthritis, Volkmann's contracture

Ulnar nerve lesions

Memorandum 1 (stabbing injury forearm)

'On inspection there is a well-healed longitudinal surgical scar over the volar-ulnar aspect of the mid forearm. There is abduction of the little finger[m] and hypothenar muscle wasting[n]. The attitude of the hand is suggestive of ulna claw hand with flexion of the ring and little finger PIP joints. The distal IP joints are also

[m] Due to denervation of ADM.
[n] Due to denervation of the hypothenar muscles.

flexed; suggesting that the FDP is intact[o]. There is also hyperextension of the MCP joints of the little and ring fingers. There is no obvious skin ulceration[p], brittleness of the nails[q] or tropic changes.'

'Can you stretch both arms out please, and then bend them and lift them above your head?'

'There are no obvious deformities such as cubitus valgus or varus suggestive of an old elbow fracture[r]. There are no obvious scars around the elbow'

'I would now like to test for sensation. Can you feel me touch you here and now here? Does it feel the same as here on the other hand?'

'The sensation is lost over both the tip of the little finger and the dorsum of the fifth metacarpal suggesting the lesion occurred proximal to the origin of the dorsal sensory branch.'

Memorandum 2 (cubital tunnel syndrome – Post surgery)

'Would you roll up your sleeves and put your hands palm down out in front of you please?' 'Can you make a fist and then straighten your fingers and turn your hands over please?' 'Please can you bend your elbows and lift your arms above your head?'

'On inspecting the dorsal surface of the hand there is marked interosseous muscle wasting, particularly of the first dorsal interosseous muscle, with hollowing on the dorsal aspect of the first web space. There is some ulna clawing but no muscle wasting on the medial side of the forearm. There is a recent surgical scar on the medial side of the elbow that is compatible with ulnar nerve decompression. Sensory testing reveals loss of sensation at both the tip of the little finger and in the region of the dorsal sensory branch. Palpating gently in the region of the scar I can feel the nerve posterior to the medial epicondyle suggesting it has not been transposed. FCU and FDP have full power, but there is significant weakness of abductor digiti minimi, and the first dorsal interosseous. Froment's test is also positive. I believe this patient has had severe cubital tunnel syndrome and a recent decompression. Recovery may take 12–18 months.'

Examination points for ulnar nerve lesions

Palpation

'I would now like to palpate the nerve at the elbow. Please tell me if this is painful or uncomfortable. I can feel the nerve posterior to the medial epicondyle and as I flex and extend the elbow it does not sublux anteriorly.'

The ulna nerve subluxes in 16% of normal subjects. Occasionally there will be a snap as the nerve dislocates with elbow flexion.

Tinel's test should be performed where the nerve enters the two heads of FCU, but again can be positive in many 'normal' subjects.

Motor testing

Practice a routine to test the nerve in a logical sequence. So test the muscles supplied in the forearm first. FCU (resisted wrist flexion, palpate the FCU tendon) and FDP to little finger (test-resisted DIPJ flexion of little finger).

Then test the nerve as it supplies the ulnar side of the hand (ADM – Resist little finger abduction with your index finger while palpating the muscle belly with your thumb) and finally the terminal supply of the first dorsal interosseous muscle (place the patients hand in neutral forearm rotation then ask them to lift the index finger, then resist as you press with your index finger and palpate the muscle belly with your thumb) (Figure 9.7). Perform Froment's test (for adductor pollicis) last. Practice explaining how it works:

'The patient is asked to hold the piece of paper or book between their extended thumb and index finger. If the ulnar nerve is intact they can grasp it using the adductor pollicis (Figure 9.8a), but if these are weak they will try to resist the paper being pulled away by recruiting the anterior interosseous nerve innervated flexor pollicis longus and flex the IPJ of their thumb (Figure 9.8b).'

Palmar interossei

Card test

'Hold your hand out. Palm down, fingers together please. I'm just going to slide this card between your fingers (middle and index). Keep your fingers straight. Can you grip the card between your fingers and stop me pulling it out? Now between your middle and ring fingers and finally ring finger and little finger.'

In the case of weak palmar interossei it is easy to pull the card out.

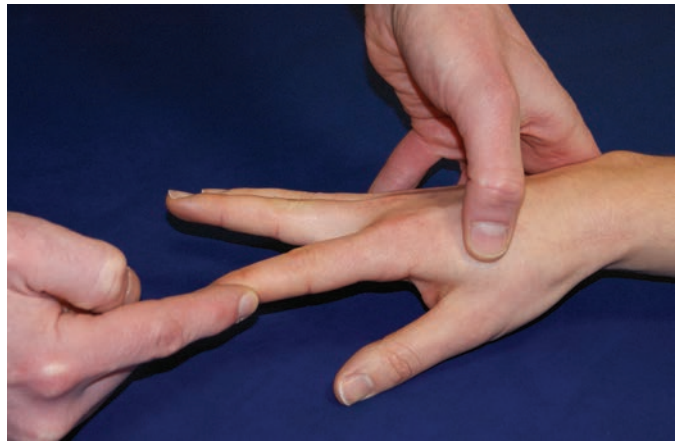


Figure 9.7 Testing first dorsal interosseous – Place index finger in abduction and ask patient to resist pressure while feeling muscle belly

[o] Ulna paradox: Clawing of the hand is more obvious in low ulnar nerve lesions because the FDP is intact and less obvious in high lesions.

[p] Caused by unnoticed trauma on the desensitized medial skin of the dorsum and palm and the medial (ulnar) 1½ digits.

[q] Due to denervation.

[r] Cubitus varus deformity occurs most often with supracondylar fractures whilst cubitus valgus deformity is more suggestive of an old malunited lateral condylar mass fracture.

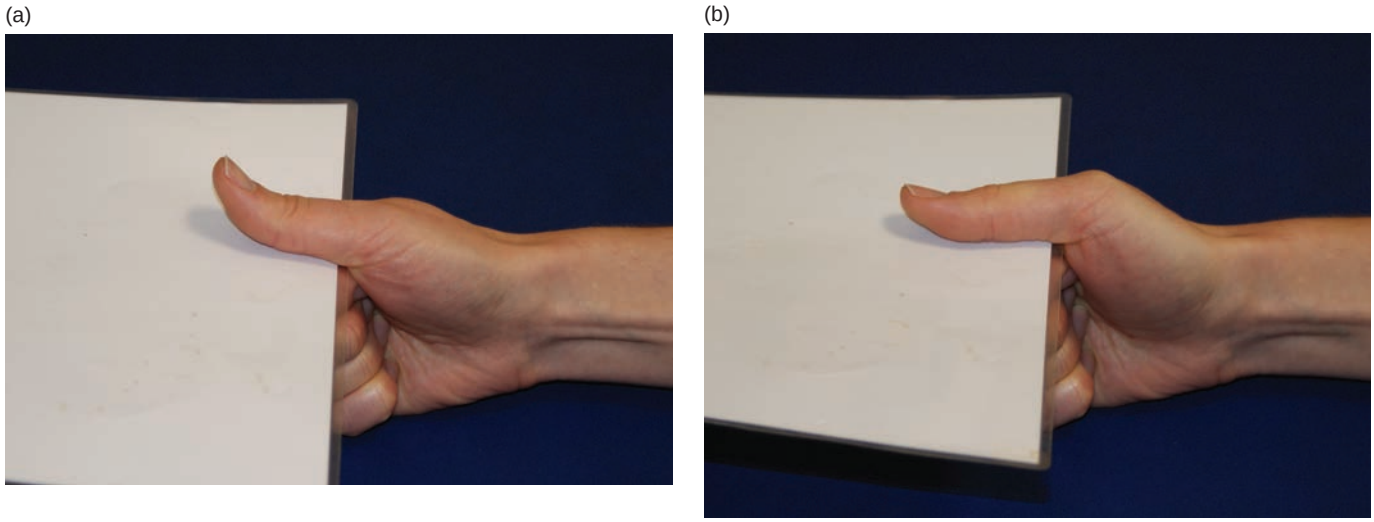


Figure 9.8 (a, b) Froment's test

Dorsal interossei

'Can you cross your fingers?'
'Can you move your middle finger from side to side?'

First dorsal interosseous muscle

Support the patients hand with the index finger uppermost. Ask them to lift the index finger. If there is significant wasting lift the finger yourself and see if the patient can hold it abducted. If they can lift it span the index finger and the muscle and while you try to adduct the finger palpate the muscle for bulk and contracture.

Abductor digiti minimi

'Now push your little finger out against my finger.'

Again test for power but also feel the bulk of ADM while it contracts.

Or

'Can you push your little fingers together?' (More sensitive test.)

FDP little finger[s] (Pollock's test)

If FDP weak, nerve abnormality at elbow (high lesion).

Differential diagnosis

- Cervical radiculopathy
- Thoracic outlet syndrome
- Cervical rib
- Cervical spondylosis
- Pancoast's tumour
- Benediction hand (high median nerve lesion with FDP paralysis to the index and middle finger) vs claw hand (high ulnar nerve lesion with little and ring finger MCP joint hyperextension and IP joint flexion)

[s] Differentiates a high from a low (distal) nerve lesion.

Sites of potential compression neuropathy at the elbow

Arcade of Struthers	Formed by superfcial muscle fibres of the medial head of triceps attaching to the medial epicondylar ridge by a thickened condensation of fascia
Cubital tunnel	Formed by fascia from the medial epicondyle to the olecranon (thickened Osborne's ligament)
Fascia of FCU	Fascial bands connecting the two heads of FCU
Anconeus epitrochlearis	An accessory muscle
Deep flexor-pronator aponeurosis	Exit of the ulnar nerve from FCU

Causes of ulnar nerve palsy (proximal–distal)

Brachial plexus

- Trauma

At the elbow

- Bony abnormalities: Osteophytes, bony spurs, cubitus valgus (tardy ulna nerve palsy)
- Scarring
- Anomalous muscles (anconeus epitrochlearis muscle)
- Tumours
- Ganglions
- Trauma: Old fractures (lateral condylar mass fracture), lacerations, iatrogenic

At the wrist

- Lacerations
- Ganglia

Ulnar tunnel syndrome (rare)

Ulnar nerve compression in Guyon's canal.

Signs of distal ulnar nerve lesion (low lesion)

- No muscle wasting of forearm
- Sensation over the dorsum of fifth metacarpal is intact
- Decreased sensation in ulnar 1½ digits (sensation of the dorsum of the hand preserved)
- Tender over Guyon's canal
- FCU intact
- Ulnar half of FDP intact (ulnar paradox), marked clawing

Tendon transfers for a distal ulnar nerve

1. For weak pinch between the thumb and index finger (thumb adduction and index finger abduction)
   - Split insertion of middle finger FDS to adductor pollicis
   - EIP to first DI muscle
2. For loss of the interossei and ulnar two lumbricals (clawing hand)
   - Zancolli capsulodesis to stabilise the MCP joint in 20° of flexion)
   - Or split tendon transfers of FDS ± EIP to the radial dorsal extensor apparatus. Carried out to restore MCP joint flexion and IP joint extension

Examination corner

Short case 1: Ulnar claw hand, low lesion with pathology at Guyon's canal, no sensory change

EXAMINER: Would you care to examine this man's right hand and tell me what you see?

CANDIDATE: There were various well-healed traumatic and surgical scars over the dorsal surface of the wrist. The volar–ulnar border of the wrist had a recent longitudinal surgical scar over Guyon's canal. Gross interosseous muscle wasting and gross clawing of the hand were evident. I examined for sensory deficit; however, none was present.

EXAMINER: What difference would you expect to find in sensation between a high and low ulnar nerve lesion?

CANDIDATE: There would be decreased sensation at the tip of the little finger but normal sensation on the dorsum of the fifth metacarpal area if the lesion is low as the dorsal branch of the ulnar nerve is spared.

EXAMINER: What is the ulnar paradox?

CANDIDATE: Less clawing of the hand with a more proximal nerve lesion. A more proximal lesion will paralyse FDP to the little and ring fingers, reducing the amount of IPJ flexion.

EXAMINER: Would you care to examine the motor function of the ulnar nerve?

CANDIDATE: Examination included FCU and FDP little finger which were both working, ADM, first dorsal interosseous muscle and Froment's test (positive). I, therefore, explained that it was a low ulnar nerve lesion affecting motor function but not sensory function. My mind went blank as I didn't know any obvious causes for this condition but luckily the examiners didn't probe me

I am also sure I jumbled up ulnar motor testing in a haphazard random way(Not proximal to distal) and in fact began with ADM. The examiners let me off with this as well!

EXAMINER: Let us move on to another case. *(Pass)*

Short case 2: Isolated ulnar nerve palsy with no scars present over limb

What is the commonest cause of an ulnar nerve palsy?
Common sites of nerve compression?
Clinical tests?
Management (conservative and surgical)?

Short case 3: Ulna claw hand

EXAMINER:
Describe the appearance.
Examine the nerves.
What is the differential diagnosis and why?
Level and why?
What is a Martin–Gruber anastomosis?

CANDIDATE: The Martin–Gruber anastomosis occurs when motor fibers normally carried entirely by the ulnar nerve enter the ulnar nerve from the median nerve via branches in the forearm. Disruption of the ulnar nerve above the level of anastomosis may not necessarily result in motor loss of ulnar-innervated muscles.

Radial nerve palsy

Radial nerve palsy is a classic clinical case. Patients with radial nerve injury and fracture fixation are frequently brought to exams. Look for scars of humeral or radius fixation!

Memorandum

'Would you roll up your sleeves and stretch your arms out in front of you please?'

'On inspection there is an obvious left wrist drop[t]. There is gross wasting of the left forearm muscles[u]. There does not appear to be any gross wasting of the triceps muscle[v]. There are no scars or swellings visible. Sensation over the first web space dorsally is

[t] Due to loss of extensor muscles.
[u] Due to loss of extensors, the muscle bulk of which is in the forearm.
[v] In high lesions.

reduced. I would now like to test the motor function of the radial nerve.'

Scars

If you identify a wrist drop ensure that you look for surgical scars from humeral fixation. In particular a posterior midline scar over triceps, but also an anterior scar from a brachialis splitting approach and scars from humeral nailing.

A scar over the volar forearm may be from a radius plating with associated superficial radial nerve injury. Iatrogenic injury to this nerve as it runs under brachioradialis is not uncommon.

Sensation

'I would now like to test sensation.'

'Can you feel me touch you here? Here? Here? Does it feel the same as here on the other hand?'

'There is sensory loss over the first dorsal interosseous muscle. This patient has features suggestive of superficial radial nerve injury. There is evidence that this may have been caused by . . .'

Test triceps[w]

Extend the shoulder

'Can you straighten your elbow?' (Gravity excluded)

Then test resistance. Test triceps reflex.

'He has normal triceps power and no loss of his triceps reflex. Weakness of the supinator and brachioradialis muscle suggests a lesion above the supinator tunnel. Weakness of the triceps suggests a lesion at or above the mid humerus.'

Test brachioradialis

Flex the elbow in the mid prone position

'Can you bend your elbow and stop me straightening you arm?'

'I am now testing brachioradialis muscle. There is a definite contraction of the brachioradialis muscle.'

Remember – Brachioradialis does not cross the wrist joint.

Test supinator

Elbow must be extended, to exclude the action of biceps. Place the forearm in full pronation. It is difficult to isolate supinator.

'Can you turn your hand over (against me)? Don't let me stop you.'

'I am testing the supinator muscle. There is a definite weakness of supination compared to the other side.'[x]

Extensors of the wrist

Place patient's wrist in extension, look for radial deviation.

'Don't let me pull it down.'

'I am testing the extensor muscles of the wrist. He has weakness of wrist extension MRC grade 4 minus.'

Extensors of the fingers

'Can you bend your elbow into your side and give me your hand facing down (palm down)? I will support your wrist. Can you try to straighten your fingers please? Straighten them. Don't let me push them down.'

'He is able to extend his IP joints because of the action of his interossei and lumbrical muscles. He is, however, unable to straighten his MCP joints.'

It is very important to appreciate that extension of the finger IP joints is from ulnar and median nerve function and MCP joint extension requires radial nerve function.

Test EPL

'Please place your palm flat on the table. Can you lift up your thumb?'

Tests retropulsion.

Test EI and EDM

Ask the patient to flex their middle and ring finger and keep their index and little finger extended at the MCP joint. It is easiest to demonstrate and ask the patient to copy you. This is possible due to the independent function of EI and EDM.

Radial nerve vs PIN palsy

Radial nerve palsy

- Sensation lost over dorsum first webspace
- Inability to extend elbow (triceps) if very high lesion
- Triceps intact but wrist drop if lesion between triceps and ECRL innervation

PIN palsy

- No sensory loss
- Nerve supply to ECRL and brachioradialis intact
- Wrist extends with radial deviation
- Unable to extend MCP joints, no thumb retropulsion (EPL)

Causes of a radial nerve palsy

Axilla

- Saturday night palsy: Neuropraxia from prolonged local pressure
- Ill-fitting crutches

Midhumerus

- Fracture of the humeral shaft (or Holstein-Lewis type injury)
- Tourniquet palsies
- Lacerations, gunshot wounds

At and below the elbow

- Entrapment syndromes (FREAS; a mnemonic for **F**ibrous tissue bands, **R**adial recurrent vessels, fibrous **E**dge of ECRB, **A**rcade of Frohse, **S**upinator)

[w] Triceps weakness suggests a lesion at the midhumeral level. Loss of all triceps activity suggests a high (plexus) lesion.

[x] Loss of supinator suggests a lesion proximal to the supinator tunnel.

- Rheumatoid elbow
- Dislocated elbow
- Monteggia fracture
- Surgical resection of the head of the radius
- Mass lesions (ganglions)

Tendon transfer (Jones' transfer)

- Wrist extension: Pronator teres to ECRB
- MCP joint extension: FCU (around ulna) to EDC or FCR (through interosseous membrane)
- Extension and abduction thumb: Palmaris longus to EPL

Supinator tunnel

The fibres of the supinator muscle are arranged in two planes, between which lies the deep branch of the radial nerve (PIN). The supinator arises from the lateral epicondyle of the humerus, the elbow joint and superior radial ulnar joint and the supinator crest and fossa of the ulna. It inserts into the posterior, lateral and anterior aspects of the neck and shaft of the radius as far as the oblique line.

Examination corner

Short case 1: Humeral shaft fracture with associated radial nerve palsy

Demonstration of clinical signs – Posterior midline humeral scar

When do you operate on humeral fractures?
What method?
What approach?
What size of plate do you use?

Short case 2: Resolving radial nerve palsy (Saturday night palsy)

'I identified on inspection that the patient had a wrist drop, I could not see any scars to suggest surgery. I tested his sensation in the distribution of the first dorsal web space and this seemed to be intact. I then proceeded to demonstrate the function of the radial nerve from proximal to distal. Explaining to the examiner why I was doing so. There was full power of triceps.'

'When I asked the patient to flex their elbow I could detect some contraction in brachioradialis and on wrist extension the wrist went into radial deviation suggesting that ECRL was intact but not ECRB or ECU. There was no function in EI, EDC or EPL. I suggested to the examiner that I felt the patient had a resolving radial nerve palsy with the muscles supplied by the main radial nerve trunk functioning, but the posterior interosseous was yet to recover.'

'We had a discussion about the use of external wrist splints vs the use of a temporary internal tendon transfer (PT to ECRB) to maintain wrist extension.'

Median nerve

The common compression syndromes are carpal tunnel syndrome and the less common pronator syndrome. An isolated anterior interosseous nerve palsy is frequently brought to exams.

Memorandum 1 (low median nerve lesion)

'On inspection of the left hand there is obvious thenar muscle wasting. The thumb appears to be lying in the plane of the palm – A simian thumb (ape-thumb deformity). There is atrophy of the pulp of the index and main fingers, dystrophic nail changes present, generalized nicotine-stained fingers and possibly a cigarette burn over the radial border of the distal phalanx of the index finger. There is no obvious ulceration seen in the hand or fingers and no visible scars are present.'

'The thumb cannot be opposed to the fingertips to produce useful function. Testing for APB revealed MRC power grade 4 minus compared to the opposite normal side with reduced muscle bulk and tone present. However, testing for FPL revealed normal power.'

Memorandum 2 (higher lesion)

'In addition, there is wasting of the left forearm. The index finger is held in a position of extension – Benediction attitude. On asking the patient to make a fist the index finger remains pointed – Finger pointing sign.'

'I would now like to test for sensation.'

'Can you feel me touch you here? Here? Does it feel the same as here on the other hand?'

'There is sensory loss over the palmar aspects of the lateral 3½ digits and thenar eminence.'

'I would now like to test for power.'

'Lay your hand on the table, palm up please.'

Median nerve motor testing

Flexor carpi radialis

'Can you bend your wrist and stop me pushing it back?'

Feel the tension in the FCR tendon

Flexor digitorum superficialis

'I am going to hold your fingers out straight, and can you bend your middle finger.'

Flexion at the PIP joint is from FDS function

Abductor pollicis brevis

'I am going to hold your wrist so you don't move your hand. Now lift your thumb up off the table to touch my finger. Push against it.'

Resisted thumb abduction and feel for contraction and bulk (Figure 9.9). The APB is the most radial of the thenar muscles and most specifically median nerve innervated. This is, therefore, the best muscle to test for the median nerve in the hand.

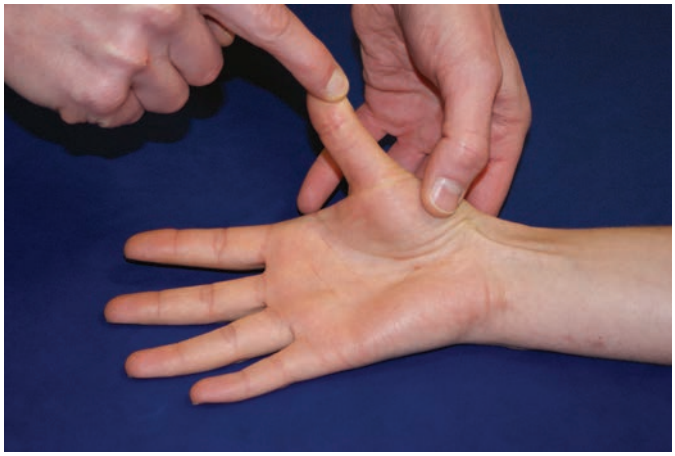


Figure 9.9 Testing APB – Resisted thumb abduction while feeling muscle belly of APB

Opponens pollicis

'Please can you touch the tip of your little finger with your thumb? Now stop me from pulling them apart.'

OK or Kiloh–Nevin sign

'Please can you make a circle with your thumb and index finger like this? And pinch them together?'

This is the test for the anterior interosseous nerve. If it is intact, the FPL and index finger FDP will flex the thumb IP joint and index finger DIP joint, and it is possible to pinch while making the OK sign. If the anterior interosseous nerve is deficient the IP joints will collapse into extension on pinching. The differential diagnosis for loss of thumb IPJ flexion is an FPL rupture.

Low nerve lesion

- Loss of APB and variable loss of FPB (and opponens pollicis)
- Weakness of thumb abduction and opposition

High nerve lesion

Low lesion plus:

- Loss of flexion IP joint thumb (FPL)
- Loss of flexion index and middle fingers (FDS, FDP)
- FCR

Tendon transfers for low lesion

For thumb opposition (loss of APB)

- Ring finger FDS transfer to APB, or EIP to APB
- MCP ± IP joint fusion

Tendon transfer for high lesion

For index and middle finger flexion

- FDP index and middle finger sutured side-to-side (tenodesed) to the neighbouring intact FDP of the ring and little fingers (FDS cannot be used, as it is supplied by the median nerve)

For flexion IP joint thumb

- Interposition graft with Pl to FPL
- FDS (ring) to FPL

For thumb opposition

- EIP to APB
- Pl to APB (Camitz)

Causes of a median nerve palsy

At the elbow

- Fractures
- Elbow dislocations

Distal to the elbow

- Pronator entrapment syndromes

In the forearm

- Lacerations
- Gunshot wounds
- Forearm bone fractures

Wrist

- Especially lacerations
- Colles' fractures
- Carpal tunnel syndrome

Carpal tunnel syndrome

This is the commonest hand condition and although rarely would be an isolated hand case, its frequency means it may be present with another pathology. You are, therefore, likely to be asked about it at some point in the exam.

History

- Age/occupation
- Hand dominance
- Numbness
- Pins and needles
- Night symptoms
- Clumsiness
- Diabetes, hypothyroid, neck symptoms

Examination

- Routine hand examination with particular attention to:
- Muscle wasting
- Sensory deficit
- Motor deficit
- Decreased sweating
- Ulnar nerve signs
- Provocative tests: Tinel's sign, Phalen's sign, median nerve compression test

Check that the patient is adequately exposed before starting your examination. Make sure their shirt is rolled above their elbows.

'In normal clinical practice I would start by examining the cervical spine, shoulders and elbows but for the moment I will just concentrate on the hands.'

'This (middle-aged) lady complains of pain, numbness and paraesthesia in the palm and fingers. This is particularly severe at night and causes her to get up and shake her hands to relieve symptoms. There is thenar muscle wasting and sensory loss over her radial 3½ digits. There is definite weakness of abductor pollicis brevis compared to the opposite side.'

'Can you pull your thumb into your palm and now push your thumb up to the ceiling?'

Resist this movement with the index finger of one hand whilst simultaneously feeling for the contraction of abductor pollicis brevis with your thumb of the same hand.

'There is also weakness of opposition of the thumb. The presence of the long flexor and variability of the nerve supply makes testing for flexor pollicis brevis of doubtful value.'

'Tinel's sign is positive for median nerve irritation and, likewise, Phalen's sign is also positive at 20 seconds.'

Look and be seen to be looking at your watch and test for at least a minute before saying it is negative.

'The median nerve compression test was positive. The flexor muscles of the forearm are not involved, suggesting a distal median nerve lesion.'

Or:

'The symptoms the patient describes are suggestive of carpal tunnel syndrome. There is normal sensation in the palm, particularly over the thenar eminence. Tinel's and Phalen's tests were positive for median nerve compression but the nerve itself showed no motor deficit.'

Signs

Wasting thenar eminence (LOAF: Mnemonic for *l*ateral two *l*umbricals, *o*pponens pollicis, *a*bductor pollicis brevis, *f*lexor pollicis brevis)

Decreased sweating and increased temperature at the thenar eminence

Decreased sensation in the radial 3½ digits (palmar branch proximal to tunnel)

Reduced power APB

Carpal compression (Durkin's test) – Most sensitive

Test	Sensitivity (%)	Specificity (%)
Tinel's	74	91
Phalen's	61	83

Causes

Can be congenital or acquired. Majority of cases are idiopathic.

Congenital

- Persistent median artery (thrombosis of such an artery can cause an acute onset of carpal tunnel syndrome)
- High origin of lumbrical muscles

Acquired

- Inflammatory: Synovitis, rheumatoid arthritis, gout
- Traumatic: Colles' fracture
- Fluid retention: Pregnancy, renal failure, myxoedema, diabetes, congestive cardiac failure, steroids
- Space-occupying lesion: Lipoma, ganglion

Differential diagnosis

- Cervical radiculopathy
- Collagen vascular disorders
- Thoracic outlet syndrome
- Raynaud's disease
- RSD
- Spinal cord lesions – Tumour, syrinx
- Peripheral neuropathy: Alcohol, diabetes

Examination corner

Short case 1: A 60-year-old woman

'I was directed to her left hand, which had marked APB muscle wasting, and was told she had had some tingling/pain in her index/middle fingers. I said I would like to commence the examination proximally from the neck but was told to concentrate on the hand. Comparing both hands I commented on unilateral thenar eminence wasting with no dorsal interosseous wasting.'

'The examiners asked what I thought the diagnosis could be. I answered carpal tunnel syndrome and was asked what I would want to examine – I mentioned sensation, motor power and provocation tests.'

'I was then asked then to examine motor power: APB was very weak but (ulnar) intrinsic power normal, which I commented on.'

'I then tested FDP to index, which appeared to be a little weaker than in the opposite hand. Challenged, I said that non-dominance plus relative disuse may make it weaker although the possibility of proximal median nerve compression should be considered.'

'I was then asked to demonstrate the carpal tunnel syndrome provocation tests, which were all strongly positive.'

'Whilst walking to the next patient I was asked about management. I mentioned that conservative measures were unlikely to help given the marked wasting, which was suggestive of chronicity – Therefore, carpal tunnel decompression would be indicated. Asked what I would advise her about the outcome, I mentioned that there was a good likelihood of early night-pain relief but numbness could take up to 1 year to settle and the wasting could be permanent.'

Short case 2: Bilateral carpal tunnel syndrome
History/exam
Benefits of simultaneous vs sequential surgery

Short case 3: A 35-year-old woman with carpal tunnel syndrome
Short history
Examination starting with the neck
Provocation tests
Tests for proximal sites of compression of median nerve

Pronator syndrome

Entrapment of the median nerve around the elbow.

History

- Ache or discomfort of the forearm after heavy use
- Weakness or clumsiness of the hand
- Paraesthesia in all or part of the median nerve

Examination

- Local tenderness to deep compression with reproduction of symptoms
- Tinel's sign is negative at the wrist but may be positive at the proximal anterior aspect of the forearm
- Negative Phalen's
- Weakness of thenar muscles but sparing of AIN innervated muscles

Provocation tests

- Elbow flexed, forearm pronated, resisted forearm supination (bicipital aponeurosis)
- Elbow extended, forearm supinated, resist forearm pronation (two heads of pronator teres)
- Resisted middle finger PIP joint flexion (proximal arch of FDS)

Sites of compression (four sites)

- Supracondylar process humerus (ligament of Struthers)
- Bicipital aponeurosis
- Between heads of pronator teres
- Proximal arch of FDS

EPL rupture

A classic clinic case: Either a rheumatoid patient or post-Colles' fracture. A traumatic laceration is unlikely.

Memorandum

'On inspection there is generalized soft-tissue swelling over the dorsum of the right wrist joint. There is also a deformed appearance of the wrist suggestive of a recent fracture. The attitude of the right thumb is one of flexion at the IP joint, and reduced span. Active dorsiflexion of the wrist is reduced to 30° compared to almost 80° on the opposite normal side. Similarly, palmar flexion of the wrist was 50° compared to 70° on the opposite side. Pronation and supination are normal. There was no active retropulsion of the right thumb, suggestive of EPL rupture. I was not able to feel any contracture of the EPL tendon. There was some mild swelling and tenderness around Lister's tubercle in the line of the EPL tendon. The index finger can point in isolation indicating the extensor indicis is present.'

Management

Initially non-operative management to see if patient is functionally limited or if the thumb is catching due to lack of span before considering surgery.

Surgery is an EIP to EPL tendon transfer. Requires a GA or regional block and the patient will need hand therapy for supervised mobilization and a splint for 4–6 weeks postoperatively.

Three incisions

- Transverse over index finger metacarpal head (remember EIP lies ulnar to EDC)
- Transverse or longitudinal incision proximal to extensor retinaculum
- Oblique incision over the thumb MCP joint to identify EPL tendon distal to the rupture

Short case 1

EXAMINER: What tendon is used to replace the function of EPL?
CANDIDATE: EIP.
EXAMINER: How do you test for the presence of EIP?
CANDIDATE: By asking the patient to point with the index finger.
EXAMINER: How many incisions do you do in this tendon transfer?
CANDIDATE: Three.
EXAMINER: Show me where exactly on my hand you would place these incisions and what you are trying to achieve with each.
CANDIDATE: At this stage the oral deteriorated rapidly as I got mixed up with the reason for the various skin incisions. *(Fail)*

Hand oral 2

What tendon rupture can you get after distal radius fracture?
Which type of fracture is particularly associated with it?
An undisplaced or minimally displaced colles fracture
Which tendon would you transfer?
Show me the incisions.

Chapter 10

Spine clinical cases

Prasad Karpe

Introduction

Over the last few years, spinal cases have become more relevant in the FRCS exam. Although candidates will not be expected to manage complex spinal conditions as a Day 1 General Orthopaedic Consultant working in a District General Hospital, knowledge of emergency management of important spinal conditions is a must. Also, many patients with spine complaints like limb radiculopathy or neurogenic claudication may be referred to your general orthopaedic clinic.

All candidates not necessarily may have done a spine job and some may find the spine cases quite perplexing. But like everything in life, it's all about planning. Just like the old saying, 'If you fail to plan, you plan to fail.'

In other words, with adequate preparation, spine cases should be an area for scoring marks.

It is essential to know two kinds of scenarios for the FRCS exams:

1. Common cases, e.g. prolapsed intervertebral disc presenting with leg pain and/or back pain
2. Rare but cannot be misdiagnosed scenarios like cauda equina or discitis

Spine case could be an intermediate case (15 min with 5 min each for history, examination and management) or a 5-minute short case.

Intermediate case

1. Cervical spondylotic myelopathy
2. Prolapsed cervical disc with radiculopathy and/or weakness (usually chronic)
3. Lumbar canal stenosis
4. Prolapsed lumbar disc with radiculopathy and/or weakness (usually chronic)
5. Scoliosis – congenital, idiopathic, or any other type
6. Kyphosis (ankylosing spondylitis)
7. Spondylolisthesis

Short cases

Any of the long cases can pop up as short cases with focused examination like inspection, palpation or check neurology. You should be slick enough to do it within 3–4 minutes with at least a minute for discussion.

It is uncommon to have a cauda equina or acute fracture case for the clinicals but these are frequently asked for the vivas.

Preparation

1. History
2. General examination of the spine
3. Neurological examination
4. Upper and lower motor neuron lesion
5. Investigations – MRI, CT, plain x-rays, nerve conduction studies
6. Management – non-operative or operative
7. Evidence-based practice: current literature, NICE guidelines, British Orthopaedic Association Standards for Trauma (BOAST) guidelines, Spine Patient Outcomes Research Trial (SPORT) trial, etc

History

To get the right answers, one needs to ask the right questions. Time limitations further add to the problem. For the FRCS Orth exams, one gets only 5 minutes for history taking and that sometimes includes summary too. But, if you ask the right questions, 5 minutes are more than enough!

By the end of your history, you should have a diagnosis, if not a differential diagnosis. There are many factors running in the background that do help in your history taking like:

- Clinical letter provided to you just prior to you entering the examination room. (Please read it carefully)
- Patient age

  Adolescent girl – Adolescent idiopathic scoliosis
  Middle age – Prolapsed disc
  Old patient – Lumbar canal stenosis, osteoporotic fracture or tumors
- Calipers, foot drop splint point towards neurology
- Syndromic patient with obvious features should ring a bell to alert you if you are dealing with non-idiopathic scoliosis or kyphosis

A word of advice. After you introduce yourself to the patient, focus entirely on the patient/family maintaining eye contact. Pretend as though the examiners do not exist. Treat it is just

like an everyday clinical scenario. It helps to focus on the case and looks more professional.

The following is a format for history taking for a spinal intermediate case like lumbar canal stenosis or prolapsed intervertebral disc. Some questions may change depending upon the case, but format should essentially remain the same. Specific scenarios are covered later.

After the introduction, handshake, name, age, etc, the first question is usually an open question like:

'Mr ABC, How can I help you today?' or 'Mr ABC, your GP writes to me (clinical letter) that you are suffering from low back pain, can you please tell me something more about it?'

1. **Pain**
   a. **Neck/low back pain**
      – Where?

      Ask patient to point out with one finger. Low back pain (LBP) could be sacroiliac joint, lumbar spine, buttock pain (neurogenic or vascular claudication). Site itself is a very important clue
      Lumbar facet pain is central back pain or may be paravertebral or sometimes radiate to buttocks or posterior thighs up to the knees. (Below knees radiation points to radiculopathy due to nerve root irritation)
      Sacroiliac joint pain is usually one-sided and located in the buttock, just to the side of the midline. The pain may radiate down the back of the thigh to the knee. Typically, it is difficult to find a comfortable position when lying in bed
      Shoulder pain can be confused with cervical neck pain associated with radiculopathy. However, shoulder pain does not radiate below elbows

   b. **Duration**: Acute (trauma or infection), subacute or chronic
   c. **Aggravating and relieving factors**: Discogenic pain is worse with sitting and bending forwards, relieved when lying down
   d. **Treatment for pain**: Analgesics, acupuncture, etc. You don't want to offer same treatment in your management if it hasn't worked in the first place
   e. **Pain in any other joints**: Are you dealing with polyarticular disease like ankylosing spondylitis or, say, rheumatoid arthritis
   f. **Radiculopathy** (arm or leg pain)

      Ask specifically where does the arm pain or leg pain radiate: E.g. middle finger radiation means C7 radiculopathy or C7 nerve root compression that can be due to prolapsed disc at C6–C7
      Or pain radiating to dorsum of the foot means L5 nerve root compression due to poster lateral disc at L45 or far lateral disc at L5–S1

   g. **What's worse**: Is it the radiculopathy or back/neck pain?

      This is an important question as the treatment differs. Discectomy helps radiculopathy but may worsen back pain in the long run. So also, neuropathic pain modulators like pregabalin are preferred over simple analgesics to treat radiculopathy

2. **Walking**
   a. **How far can you walk?** (In terms of minutes /blocks or yards)

      If the patient says he/she can walk only for 5 minutes, it is important to know what stops the patient from walking further. Is it the cramping in the legs (claudication) or breathlessness/chest pain or palpitations? It is not uncommon for old patients to have other co-morbid conditions. Patients with neurogenic claudication may also experience worsening of numbness, paresthesias rather than pain alone

   b. **If there is claudication history**

      Differentiate between neurogenic and vascular claudication. Neurogenic Neurogenic claudication improves with bending forwards (shopping-cart sign), improves on cycling and climbing stairs is better than descending them. Pulses will be normal and there may be neurology. Vascular on the other hand improves on standing, worsens on going uphill due to increased metabolic demand, pulses are weak, neurology will be normal and there may be associated skin changes. Associated skin changes in peripheral vascular disease include thin shinny skin with hair loss or trophic changes in the nails

   c. **Has the walking distance reduced?**

      This means that nerve compression is worsening. Rest pain implies critical compression. (Does this patient need early surgery?)

3. **Weakness in any of the limbs?**

   Foot drop: L4 and/or L5
   Hand/grip weakness: C8 or T1
   Triceps weakness: C7

   In other words, knowing your myotomes and dermatomes helps in history taking and arriving at a diagnosis with the history itself

4. **Bowel or bladder weakness**

   This question cannot be missed. 'Do you have any problems passing water?' If the answer to this is yes then:

   'Can you feel your bladder filling up?'
   'Can you feel your back passage when you're cleaning yourself?' (Loss of perianal sensations)
   'Do you have control when you pass water?'

Incontinence is usually a last sign of cauda equina, which usually means irreparable damage
Loss of bladder control on coughing, standing or straining, usually in females, signifies stress incontinence due to weak sphincter control. This should be differentiated from cauda equina. There is no other neurology in stress incontinence

5. '**Are you able to …?' questions**

   These questions help us to assess how disabled the patient is or is able to do his/her activities of daily living. Similar to questions asked in Harris Hip Score for hip history:

   'Are you able to do your washing and dressing?'
   'Are you able to stand for prolong period of time?'
   'Are you able to sit for prolong period of time?'
   'Are you able to sleep comfortably?'
   'Is you social life restricted or normal?'
   'Do you use a walking stick or crutch?'

6. **Red flags**: Night pain, weight loss, fever and history of malignancy
7. **What treatment have you had already?** Physiotherapy, braces, injections, surgery. If yes, did it help? And for how long? Details of surgery?
8. **Past history**: Four questions

   Medical: Diabetes, hypertension, etc
   Major past operations
   Medications: Steroids, blood thinners, immunosuppressant's (affect surgery)
   Allergies

9. **Social history**: Four questions again

   Occupation?
   Smoking?
   Alcohol?
   Where do you live? House or a bungalow? Are there any stairs?

10. **What are your expectations or what do you want from me?**

    There's no point in discussing complex surgeries when the patient wants relief from his/her radiculopathy that can be managed with neuropathic medications or root blocks

The last five questions are similar for all intermediate cases. Please do not miss these, otherwise it may be embarrassing in management if the examiner tells you later that the patient is smoker or is on steroids, some important points that can alter management!

General examination of the spine

Follow basic orthopaedic concept: Look, Feel and Move. Only additions are neurological examination and special tests.

1. **Inspection**

   a. **General appearance of the patient**

      Does the patient have features suggestive of a syndrome, e.g. café-au-lait spots in neurofibromatosis? Or long arm span with long slender fingers suggestive of Marfan's syndrome?
      Look in the vicinity for walking sticks, calipers, braces or custom-made shoes and comment on them

   b. **From front**

      Both shoulders appear same level?
      Both anterior superior iliac spine (ASIS), knees same level? (Leg length discrepancy can lead to scoliosis)

   c. **From sides**

      Normal or loss of cervical and lumbar lordosis?
      Flexion in knees? (Compensation for loss of lumbar lordosis)
      Exaggerated thoracic kyphosis? (ankylosing spondylitis)

   d. **From back**

      Step sign? (Spondylolisthesis)
      Scoliosis? Describe which side, what level and how many curves; e.g. single right-sided convex thoracic scoliosis with/without both shoulders at same level and with/without pelvic obliquity
      Any obvious skin abnormalities like café-au-lait spots, previous scars, lipoma, etc
      Ask patient to bend over to see for Adam's forward bending test to see for prominence of scoliosis and/or rib hump

   e. **Gait**

      Symmetrical, presence of all three rockers, adequate step length with or without any assistance
      Comment on any abnormalities like crouched gait in lumbar canal stenosis or presence of foot drop in L4–L5 root involvement

2. **Palpation**

   Gently tap over the midline with a fist and ask for any spinal tenderness. Also look into the patients' eyes when doing this. Some spine surgeons do palate facet joints bilaterally to elicit tenderness.
   Step sign of spondylosisthesis is better palpated than inspected.

3. **Movements**

   CERVICAL FLEXION: Can you please touch your chin to your chest?
   EXTENSION: Can you please look up to the ceiling?

LATERAL FLEXION: Can you try touching your ear to your shoulder?
ROTATION: Please look towards the right and left?
LUMBAR FLEXION: Can you please bend forwards? Keep hand on the back to feel for the spine bending forwards to check if it's the hip or spine that flexes. Some patients use hip flexion to compensate for the spine. Discogenic back pain is worse with flexion.
EXTENSION: Can you please bend backwards? Painful extension is one of the few positive signs in degenerative lumbar canal stenosis and facet arthritis.
LATERAL FLEXION: Please bend on either sides? Check how far the hands reach up to the knees.
ROTATION: Can you please turn to either side with hands on your hips?

4. **Special tests**

Straight leg raise test for prolapsed lumbar disc
FABER test for sacroiliac joint pathology
Shoeber's test for ankylosed spine

These are discussed later with each scenario.

Neurological examination

By the time you approach the exams, you should be able to do a complete neurological examination in under 5 minutes.

Make the patient lie on a couch to make patient comfortable. This examination can be further divided into:

1. **Nutrition**: Presence or absence of any wasting. Wasting is more marked in a lower motor neuron lesion
2. **Tone**: Normal, increased or decreased
3. **Power**: As per Medical Research Council (MRC) grading

Some candidates waste time checking each movement at every joint. In spine, this is not rewarding. At the end of the examination, you should be able to tell what root/spinal level is involved. Following is easy and quick way to check power:

C5 – Shoulder abduction
C56 – Elbow flexion
C7 – Elbow extension
C8 – Finger flexion or ask patient to press on your fingers
T1 – Finger abduction
L12 – Hip flexion
L34 – Knee extension
L4 – Ankle dorsiflexion with some contribution from L5
L5 – Big toe extension
S1 – Ankle plantar flexion

4. **Sensations**

Check both sides simultaneously to compare and save time. In other words, stroke your finger on identical dermatomes on both limbs. A sensible question would be 'Does this feel the same on both sides?' If sensations are reduced in lower limb dermatomes, then check dermatomes in abdomen and thorax. Sensory level is suggestive of cord compression
Non-dermatomal pattern of loss of sensations is seen in peripheral neuropathy like diabetes (glove and stocking) or psychogenic disorders

5. **Reflexes** (Tables 10.1 and 10.2)

Never forget the Babinski test in lower limbs and Hoffmann's sign in upper limbs that help to differentiate between upper and lower motor neuron lesions. Hoffmann's is like the Babinski reflex of the upper limb. Flicking the terminal phalanx of the middle finger produces a flexion of the terminal phalanx of the thumb (positive Hoffmann's). If there is time, check joint position and vibration sense especially in cervical spondylotic myelopathy.

At the end of neurological examination, don't forget to check both pedal pulsations and mention to the examiner that you would like to examine the hips, sacroiliac joints and may be the knees. It is preferable to check both pedal pulsations and do a FABER test (flexion, abduction and external rotation) prior to doing neurological examination. A negative FABER test suggests that the hips, knees and sacroiliac joints are essentially normal

Table 10.1 Upper limb reflexes

Reflex	Nerve segments
Biceps jerk	C5–C6
Triceps jerk	C7–C8
Brachioradialis jerk	C6–C7

Table 10.2 Lower limb reflexes

Reflex	Nerve root segments
Knee jerk	L3–L4
Ankle jerk	S1–S2

Upper and lower motor neuron lesion

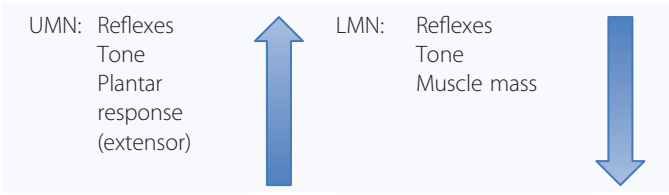
This is medical school knowledge, absolute basics.

Why is this important? It helps in localizing the site of the lesion.

Following are the rules:

1. In an upper motor neuron (UMN) lesion, the tone is increased, there is hyperreflexia and there is extensor plantar response. There is wasting, but the wasting is less marked as compared to lower motor neuron (LMN) lesion.

In a LMN lesion, wasting is marked, tone is reduced and there is hyporeflexia. Also, there is no extensor plantar response. So:



2. Any lesion up to the spinal cord just short of anterior horn cell is an UMN lesion. So, a stroke affecting the brain or syringomelia affecting the cervical central cord is a UMN lesion. Peripheral neuropathy or, say, poliomyelitis affecting anterior horn cells in the spinal cord is a LMN lesion
3. The spinal cord ends at lower part of L1 or upper part of L2. So, if clinical examination suggests a UMN picture, the lesion has to be above L1–L2

   So, neurological presentations could be:

   a. Normal upper limbs and LMN picture lower limbs: Lesion below L1–L2; e.g. cauda equina, peripheral neuropathy or lumbar plexus injury
   b. Normal upper limbs and UMN picture lower limbs: lesion above L12 and below cervical spine. In other words, lesion should be in the thoracic spine
   c. UMN picture in both upper and lower limbs: lesion in either brain and or cervical spine. Note, there could be an additional lesion in the thoracic spine

Investigations

1. **Blood investigations**

   Depending on what you suspect at the end of your history and examination, talk about blood investigations. In some cases, you may take the examiner directly to radiological investigations

   Examples for blood tests in a patient with suspected metastatic region in spine with unknown primary are:

   a. Baseline bloods (including infective markers): FBC, U&E, ESR, CRP, LFT
   b. Metabolic markers: Ca, PO_4, vitamin D, Serum Alkaline PO_4
   c. Tumor markers: PSA, myeloma screen, Bence Jones protein, CEA, etc

2. **X-rays**

   Like foot and ankle, almost always standing x-rays preferably the whole spine unless contraindicated. Views asked are anteroposterior (AP) and lateral (and bending views in scoliosis for preoperative planning only).

   a. **AP view**

      Are the vertebral bodies and spinous processes aligned?
      Are the vertebrae with pedicles intact? (Winking owl sign for pedicle destruction)
      Is there loss of disc height?
      Is there facet arthritis?
      Is there a scoliosis? If yes, describe the curve; e.g. single thoracic left-sided convex scoliosis with apex roughly at T5 vertebrae
      Risser's grade in scoliosis to check for curve progression?

   b. **Lateral view**

      Normal curves? (i.e. thoracic kyphosis (primary curve) with cervical and lumbar lordosis(secondary curves))
      In utero, spine assumes a C-shaped (kyphosis – primary curve) to cope for the small space. During the first few months of life, once the child develops neck control, the cervical lordosis or secondary curve develops to allow infant to visually access his environment. The secondary lumbar curve (lordosis) develops after the fifth month when the child starts unsupported sitting. This curve, which is well-suited to upright posture, continues to develop till child starts standing, walking and running
      Is there any loss of cervical or lumbar lordosis?
      Is there any exaggerated thoracic kyphosis?
      Presence of fractures, lysis, listhesis; reduced disc height or fuzzy end plates? (?Discitis)

   c. **Sagittal balance**

      The whole purpose of a standing whole spine x-ray is to see for sagittal balance, pelvic incidence, sacral slope and pelvic tilt. It is more important to correct sagittal imbalance (kyphosis) than coronal imbalance (due to scoliosis) or else surgery may fail. Sagittal imbalance shifts the center of gravity either in the front or back of the body and makes locomotion difficult. A normal or neutral sagittal balance means a plumb line falling from center of C7 should fall on the posterosuperior edge of S1 vertebra. If the C7 line falls in front of S1 then it is positive sagittal balance and if it falls behind S1 then its negative sagittal balance. The osteoporotic thoracic spine with previous fractures easily illustrates this. These patients have positive sagittal balance and, hence, their center of gravity shifts forward. This in turn predisposes them for further wedge compression fractures with worsening of kyphotic deformity (Dowager's hump)

3. **Magnetic resonance imaging (MRI)**

   Closer to your exams, you should be well-prepared regarding MRI in terms of uses, how it works, T1 and T2 images, etc. You may be given a spine MRI scan for the

clinical or vivas and asked to talk about it. After commenting on what spine level it is, the following points should be of help:

a. *Is this a sagittal, parasagittal or axial image?*
   Axial images are obvious but learn to differentiate sagittal and parasagittal images. Sagittal images pass through the center of the spine and unless there's no scoliosis , one should be able to see the entire spinal cord and thecal sac. Parasagittal images are useful to detect nerve root compression on either side
b. *Is this a T1, T2 or STIR image?*
   T1 image water is dark and fat is bright. T1 is better for anatomy. So if there is compression (after seeing T2 image), T1 helps to precisely see what's the extent of compression. So also, in metastases, once cancer cells replace the fat marrow, loss of fat signal is seen on T1 images. So for checking metastases, see T1 images.
   T2, water is bright (cerebrospinal fluid (CSF)) and fat is bright as well to some extent. An easy way to remember is that water is H_20, which has a 2. Young disc has more water content and, hence, is bright on T2. Water is seen more in inflammation-like infections/fractures. Hence, a bright signal in T2 in a vertebral body could mean fracture or infection
   Ask for STIR (short tau inversion recovery) images, which is fat-suppressing imaging that helps to localize infections or fractures
c. *Then comment on any thecal sac compression, disc protrusion, extrusion, sequestration, and any fractures, evidence of osteomyelitis*

4. **Computerized tomography (CT) scan**

   If there is any fracture or difficult to visualize region, like the cervicothoracic spine, request a CT scan. A CT scan also helps see for bony compression, like ossified posterior longitudinal ligament, or to get more detail of the bony anatomy and fractures. Also, a CT myelogram is performed in patients when MRI is contraindicated
5. **Bone scan**

   For infections, primary tumours (osteoid osteoma) and metastases
6. **Biopsy**

   Suspected tumors or infections. You may be asked the principles of biopsy here
7. **Nerve conduction studies**

   Nerve conduction studies help to localize a lesion or confirm a diagnosis. These are not always necessary, but may be helpful in certain scenarios like double-crush syndrome with carpal tunnel or ulnar nerve compression associated with nerve lesions in neck. They also help in diagnosing peripheral neuropathy or motor neuron disease (along with electromyography)

Management

Based on the history, patient expectations, examination and investigations, you will be asked to offer a plan for management of your patient.

1. **Non-operative**
   Unless there is instability (like fracture) or neurology, almost always the first line is non-operative management that includes:
   - Analgesia
   - Activity modification
   - Physiotherapy
   - Cognitive behavior therapy
   - Braces, e.g. scoliosis
   - Injection treatment (facet joint, epidurals, nerve root blocks)
2. **Operative**

   Indications
   - Neurology
   - Worsening of neurology
   - Instability
   - Failure of non-operative treatment

Evidence-based practice

Please read the current literature on common spinal conditions. Quoting literature does improve your final score. Following are some of important literature for spine:

- BOAST guidelines
- NICE guidelines for osteoporotic spinal fracture, cervical disc replacement, suspected spinal metastases, etc
- SPORT trial for lumbar canal stenosis, prolapsed lumbar disc, spondylosisthesis

Knowledge of current literature about fusion for low back pain, cervical disc replacement vs fusion for cervical disc protrusion and recent concepts about vetebroplasty and kyphoplasty should be known for the exams.

Prolapsed lumbar disc

Prolapsed lumbar disc is a common spine pathology, so a common case in exam. It is usually an intermediate case and, therefore, 5 minutes each for history, examination and management.

Know the types of disc based on location – Central, posterolateral (commonest) and far lateral. The L45 posterolateral disc will compress on the L5 nerve root (traversing root), while the L45 far lateral disc compresses on the L4 nerve root (exiting root).

Also know the difference between protrusion, extrusion and sequestrated disc.

History

Follow the usual questions asked in history taking:

- Male preponderance (3 : 1) with peak incidence in the fourth and fifth decade of life. Ninety per cent of these

patients improve with non-operative management in the first 3 months. (In other words, an exam case may be a patient who belongs to the latter 10%.)
- Low back pain and or radiculopathy (enquire about the specific location or dermatome – Clue of the root involved)
- Motor weakness if any
- Bladder or bowel disturbances – Cauda equina(rare in exams)
- Activities of daily living
- Red flags
- Treatment had so far – Physiotherapy, epidural or nerve root injections, surgery, medications
- Past history, social history and expectations – Patient wants to get rid of back or leg pain or both?

Examination

Usual orthopaedic examination – Look, Feel, Move, Special tests and Neurology.

Look: May have a list away from the side of pathology to take the pressure off the nerve. Gait may be altered if there is weakness, e.g. L4–L5 weakness presents as a high steppage or foot drop gait. So also a Trendelenberg gait in L5 weakness (gluteal)

Feel: Central or paraspinal tenderness

Move: Flexion reduced in discogenic back pain (also sitting and axial loading) while extension reduced if there is facet arthritis

Special tests: In other words the nerve root tension signs. Many are described, but it is not feasible for all

a. L5 or S1 prolapse: ***Straight leg raise (SLR) test*** is best done supine. Reproduces leg pain and paresthesias in 30–70° of leg flexion. The ***Lesegue sign*** is SLR aggravated by forced ankle dorsiflexion, while the ***bowstring sign*** is SLR aggravated by compression on popliteal fossa
b. L2, L3 and L4 discs (higher disc prolapse): The ***femoral nerve stretch test***, which is done in prone position

Neurological examination: As mentioned previously in this chapter, check for motor and sensory weakness, but don't forget reflexes and Babinski. If reflexes are brisk, then start doing a neurological examination of the upper limbs. Always mention that you would like to do a neurological examination of the upper limbs as well for the sake of completion

Investigations

AP x-ray: Scoliosis, facet arthritis, count number of lumbar vertebrae (T12 rib helps) to see for lumbarisation of S1 or sacralisation of L5

Lateral x-ray: Loss of lumbar lordosis, facet arthritis, reduced disc height

MRI: Sagital T2: Loss of high signal in the disc (degeneration) with prolapse, extrusion or sequestration and comment on the level. See the axial T2 to see for the central, posterocentral or far lateral location of the disc

Management

Ninety per cent of disc prolapses are at L5–S1 or L4–L5. Ninety per cent of disc prolapse sciatica improves within 3 months without surgery, so you may have a patient with no signs but a big disc on MRI scan

Non-operative

The first line is non-operative management that includes:
- Analgesia
- Activity modification
- Physiotherapy (extension exercises)

Medications like pregabalin and gabapentin help relieve leg pain. Ninety per cent of patients improve with these interventions.

The second line of treatment if the above fails is epidural or selective nerve root blocks. These have long lasting improvement of about 50% and better outcomes are with extruded discs than contained discs.

Operative

Indications:
- Neurology (cauda equnina – Emergency)
- Worsening of neurology
- Failure of non-operative treatment (after 6 weeks)

Indications, procedure and complications of microdiscectomy are frequently asked. Approximately 90% patients have long lasting outcomes with surgery. SPORT trial outcomes at 2 years suggest that there were no significant differences in the primary outcome measures for operative compared with non-operative groups. However, statistically significant outcomes for surgical intervention if sciatica is bothersome. Leg pain and positive sciatic stretch test are good predictors for positive outcome after surgery.

Intermediate case: Lumbar disc

GP note: 'Forty-five-year-old Mr Smith c/o LBP and right leg pain since past 4 months. Can you please take a history, examine this gentleman and discuss management.'

CANDIDATE: Hello Mr Smith, I am Mr K, one of the exam candidates, is it OK if I ask you some questions and examine you?

MR SMITH: Sure doctor, its nice meeting you.

CANDIDATE: Your GP tells me you have back and right leg pain. Can you tell me something more about it? (*Open-ended question*)

MR SMITH: I never had any problems with my back anytime in the past, but about 4 months ago when I was bending forward to lift my suitcase, I felt a cramp go down my right leg. This pain was quite severe, worse than a toothache and is the same since then. This was followed with low back pain, which is not as bad as the leg. This leg pain concerns me and is affecting my daily routine.

CANDIDATE: Can you point and show me where is your low back pain and how does your leg pain radiate?

MR SMITH: *Points to lumbosacral region and showing leg pain radiation along L5*

CANDIDATE: *(Thinking about L5 root . . . means posterolateral L4–L5 disc or far lateral L5–S1.)* Anything that worsens or helps your pain?

MR SMITH: My back pain is worse with sitting but standing and on lying down it is OK (*discogenic back pain*). My leg pain though is constant. I have tried many pain medications, physiotherapy, but this leg pain is showing no signs of going away. I can't walk too far without pain and my sleep is also disturbed. I have no social life now and this leg pain is affecting me mentally as well.

CANDIDATE: Do you have pain in any other joints?

MR SMITH: No.

CANDIDATE: How far can you walk?

MR SMITH: I can walk non-stop for about 15 minutes but can feel this leg pain constantly.

CANDIDATE: Do you have any weakness in any of your limbs?

MR SMITH: No.

CANDIDATE: Do you have any problems with your waterworks?

MR SMITH: No.

CANDIDATE: Have you lost weight recently or had episodes of fever?

MR SMITH: No.

CANDIDATE: What treatment have you had so far?

MR SMITH: Physiotherapy and painkillers. Not much help though.

CANDIDATE: Clearly you are struggling.

(Past history: four questions)

Any medical problems like diabetes, hypertension, etc?

MR SMITH: No.

CANDIDATE: Any major past operations?

MR SMITH: No.

CANDIDATE: Do you have any allergies?

MR SMITH: No.

CANDIDATE: Are you on any medications like steroids, blood thinners?

MR SMITH: Apart from pain medications like ibuprofen, paracetamol, I am not on any other medications. My GP started me on gabapentin, but I did not take them due to work issues.

(Social history: four questions again)

CANDIDATE: What do you do for a living?

MR SMITH: I am a bus driver but I have not worked for the past 4 months. *(Clearly, gabapentin or pregabalin should be used with caution due to side effects of sedation.)*

CANDIDATE: Do you smoke?

MR SMITH: No.

CANDIDATE: Do you have alcohol socially or everyday?

MR SMITH: No, I don't fancy alcohol.

CANDIDATE: Do you live alone? And in a house or a bungalow?

MR SMITH: In my house with my partner.

CANDIDATE: What are your expectations or what do you want from me now?

MR SMITH: If only you could somehow take this leg pain away, I will be grateful.

EXAMINER: So, what do you think is happening?

CANDIDATE: A 45-year-old gentleman is complaining of leg pain along L5 dermatome as well as back pain. Leg pain is worse than his back, I am thinking of a prolapsed disc at L4–L5 or a far lateral disc at L5–S1.

EXAMINER: Please go ahead with the examination.

CANDIDATE: *(After making the patient stand.)* . . . On inspection from the back, the pelvis appears at the same level but there is a listh towards the left. The skin on the back appears normal. On inspection from the sides, there is loss of normal lumbar lordosis.

Mr Smith can you please walk for me? His gait is unassisted, but I notice that he is taking steps very cautiously.

Mr Smith, can you walk tiptoes and on your heels?

He can do heel and toe walking that suggests that he has no gross weakness of L4–L5 and S1.

On palpation form the back, (looking at the patients face) he has central and paraspinal tenderness.

On checking movements, all his movements including flexion, extension lateral flexion and rotations are reduced.

I am now doing the special tests (making the patient lie down) SLR on the left is around 80° but on the right it's reduced to 40°. Faber's test is bilaterally negative and both pedal pulsations are well felt.

Neurological examination reveals normal tone, no motor deficit but he has reduced sensations in his right L5 dermatome. Reflexes are normal and plantars are down going.

These examination findings suggest a disc prolapse involving a right L5 nerve root.

EXAMINER: What would you like to do?

CANDIDATE: I would first like to have standing x-rays of his lumbar spine followed by an MRI.

EXAMINER: These are his x-rays. (Figure 10.1 a and b)

CANDIDATE: Standing AP x-ray of full spine shows coronal tilt towards the left. Lateral x-rays shows loss of lumbar lordosis and reduced disc height at L4–L5.

EXAMINER: What is the cause of his scoliosis?

CANDIDATE: This is a compensatory scoliosis to take the pressure away form the right L5 root.

EXAMINER: These are his MRI images. (Figure 10.2 and 10.3 a and b)

CANDIDATE: MRI T2 sagittal image showing a disc protrusion at L45, with loss of disc height and low signal suggesting dehydration. There is also loss of disc signal at L5–S1 suggesting dehydration.

The axial T1 and T2 images shows a posterolateral disc at L4–L5 on the right compressing on the L5 nerve root.

EXAMINER: So what would you like to do now?

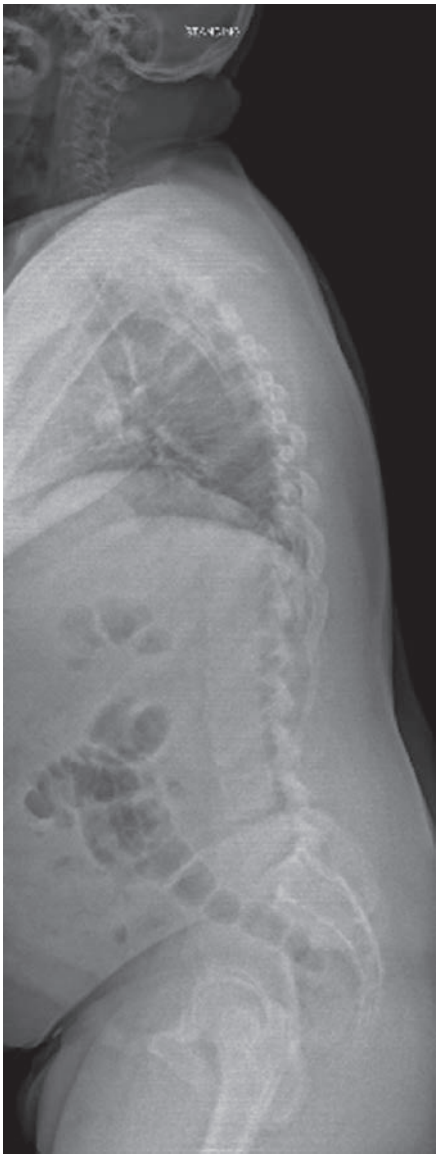
CANDIDATE: He has tried the initial non-operative measures that have failed. I will now offer him selective right L5 nerve root block explaining to him the complications and 50% chance of success.

EXAMINER: What are the complications of blocks?

CANDIDATE: No relief, worsening of symptoms, infection, bleeding, nerve damage with paralysis, further treatment may be needed.

EXAMINER: Nerve block has failed, what will you do now?

(a) Lateral spine



(b) AP spine

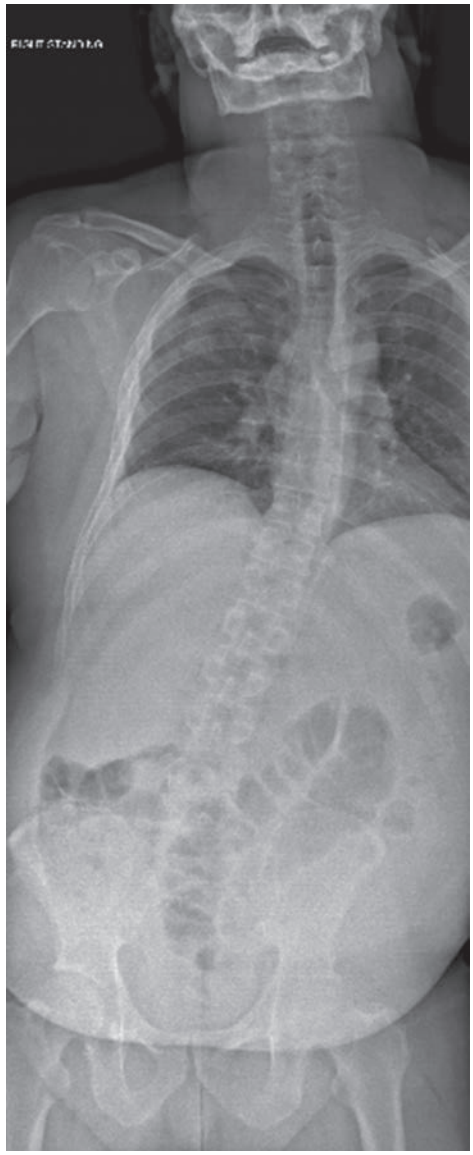


Figure 10.1 (a, b) Standing x-rays of lumbar spine

CANDIDATE: I will discuss surgery with him. I will refer him to a spinal surgeon for right-sided microdiscectomy at L4–L5.

(*The examiner could ask you to consent a patient for the surgery, complications, procedure and outcomes.*)

EXAMINER: The patient wants to know what will be the success of the operation?

CANDIDATE: This operation is primarily meant for relief of leg pain. Ninety per cent of operated patients have long-lasting improvement, more so as he has positive predictors of good outcome following surgery, like chief complaint of leg pain and a positive sciatic stretch test. While he will realize the benefit of no leg pain early, there is no difference in outcomes between operative and non-operative after 4 years.

Bell rings.

EXAMINER: Thank you.

Lumbar canal stenosis

This is a very common intermediate case for exams. Diagnosis is mainly based on history, as there are few physical signs. It is frequently associated with arthritis of the lower limbs (degenerative process) and, hence, always mention to the examiner that you will like to examine the hips and knees as well. It is vital to distinguish between vascular and neurogenic claudication, as the treatment for each is entirely different. Sometimes, both may coexist in the same patient.

Neurogenic claudication improves with bending forwards (shopping-cart sign), worsens climbing downhill, pulses will be normal and there may be neurology. Cycling has no effect and often symptoms of pins and needles are bilateral in both legs with back pain as well.

Vascular claudication on the other hand improves on standing, worsens on going uphill or cycling due to increased metabolic demand, pulses are weak and neurology will be normal. Symptoms are felt in the calf and bending forwards has no effect.

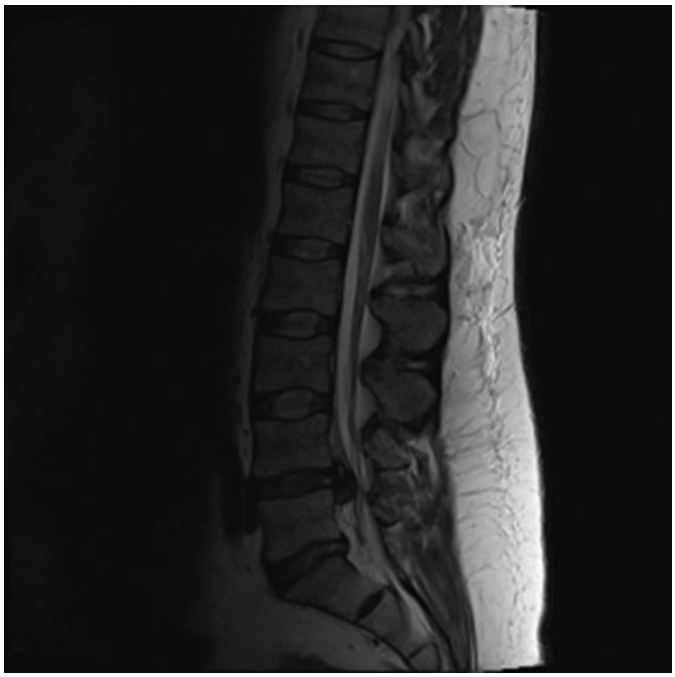


Figure 10.2 MRI scan T2 sagittal view

Classification of lumbar canal stenosis

a. **Primary**: 10% of cases. Congenital short pedicles with medially displaced facets. Present from birth with the short pedicles seen on lateral x-rays
b. **Secondary** (or acquired): More common and can be due to:
   - Degenerative – Due to disc protrusion, facet hypertrophy, ligamentum flavum hypertrophy or spondylolisthesis
   - Iatrogenic or post surgical
   - Trauma
   - Inflammatory – Paget's, ankylosing spondylitis

Also know the anatomical classification of lumbar canal stenosis, which includes central, lateral and foraminal stenosis.

Intermediate case: Lumbar canal stenosis

GP note: 'Fifty-nine-year-old Mr Jack c/o LBP and difficulty in walking since past 8 months. Can you please take a history, examine this gentleman and discuss management.'
(NB. In this scenario, only a summary of the history.)

EXAMINER: Can you summarize your history please?

CANDIDATE: Mr Jack is a 59-year-old retired plumber who complains of low back pain and bilateral neurogenic claudication. I say it's neurogenic claudication because his leg symptoms improve on bending forwards and climbing uphill. Cycling has no effect on his leg symptoms and he has no history of previous peripheral vascular disease and he is a non-smoker. His walking distance has gradually reduced from 15 minutes 6 months ago to rest pain now. His sleep is disturbed now due to leg pain and his leg

(a) (b)

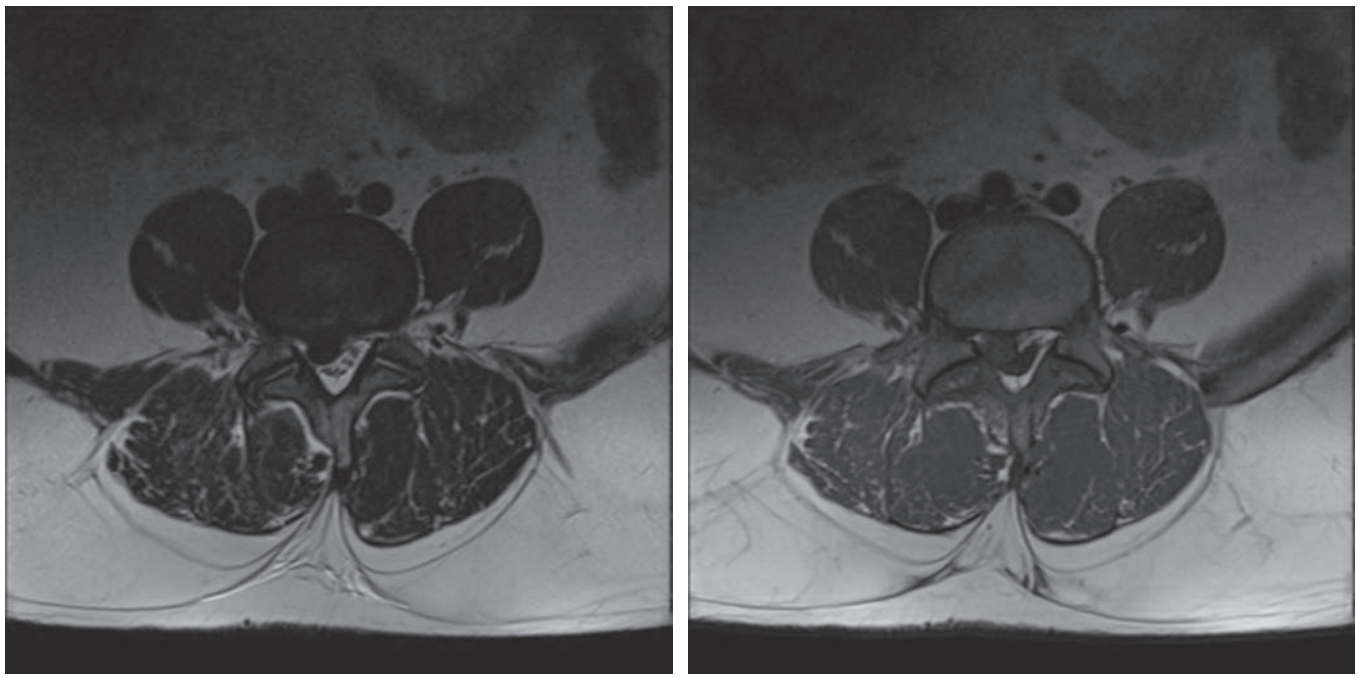


Figure 10.3 MRI scans (a) T2 Axial L4/L5 (b) T1 Axial L4/L5

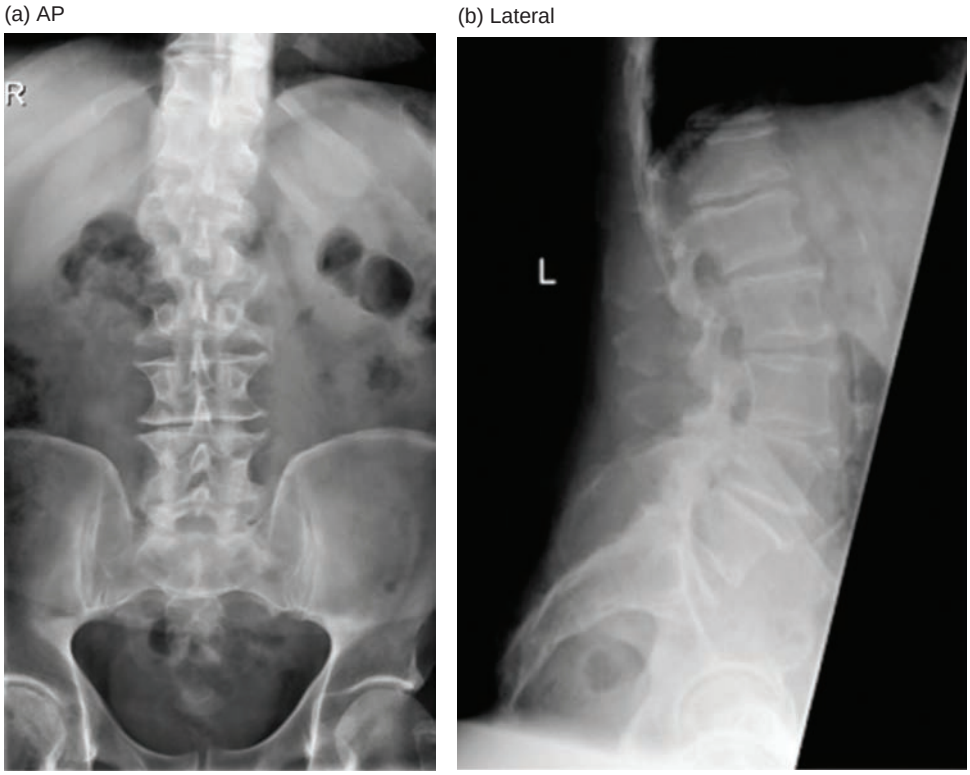


Figure 10.4 (a) AP and (b) lateral x-rays

symptoms are worse than his back. He has no bowel or bladder symptoms but has a past history of diet controlled diabetes for 10 years. He has tried physiotherapy, analgesics and caudal epidural injections that have given him no great relief. He wants to improve his walking and relief from his leg pain.

EXAMINER: (*interrupting*) What are you suspecting based on your history?

CANDIDATE: Lumbar canal stenosis

EXAMINER: Please carry on with your examination.

CANDIDATE: On inspection, Mr Jack has difficulty in standing erect, adopting a simian stance with hips and knees slightly flexed. He walks with a stooped gait and has generalized lumbar spinal tenderness on palpation.

There is normal spinal flexion but moderate and painful restriction of spinal extension.

Both pedal pulsations are well felt and he has no nail changes or loss of hair suggestive of any vascular involvement (*Never forget this in a lumbar canal stenosis case.*)

SLR bilaterally is 80° suggestive of no root irritation (though disc prolapse does not usually occur in this age group, rarely patients with canal stenosis can have acute disc prolapse in background of lumbar canal stenosis with exacerbation of their symptoms. Such patients can have root irritation signs. So also, patients with foraminal stenosis can present with nerve root signs.)

Neurological examination reveals normal nutrition, normal tone and no motor or sensory deficit. His reflexes are absent in both knees and ankles and Babinski is equivocal.

EXAMINER: What is the cause of loss of reflexes in this patient?

CANDIDATE: Lumbar canal stenosis or peripheral neuropathy due to diabetes. Also, age-related nerve degenerative process could also lead to loss of reflexes.

EXAMINER: What would you like to do?

CANDIDATE: I would like to investigate him with baseline blood investigations including blood sugar levels and HbAc to confirm he has had good control of his diabetes. Also, standing AP and lateral x-rays of his lumbar spine along with an MRI of his lumbar spine.

EXAMINER: Please comment on his AP and lateral x-rays? (Figure 10.4 a and b)

CANDIDATE: Standing AP lumbar spine x-ray showing mild scoliosis with loss of disc height and osteophyte formation. The lateral x-ray also shows loss of disc height with anterior and posterior osteophyte formation. There is grade 1 listhesis at L34 with facet arthritis at L4–L5 and L5–S1.

EXAMINER: Please comment on his MRI scans. (Figure 10.5 a–c)

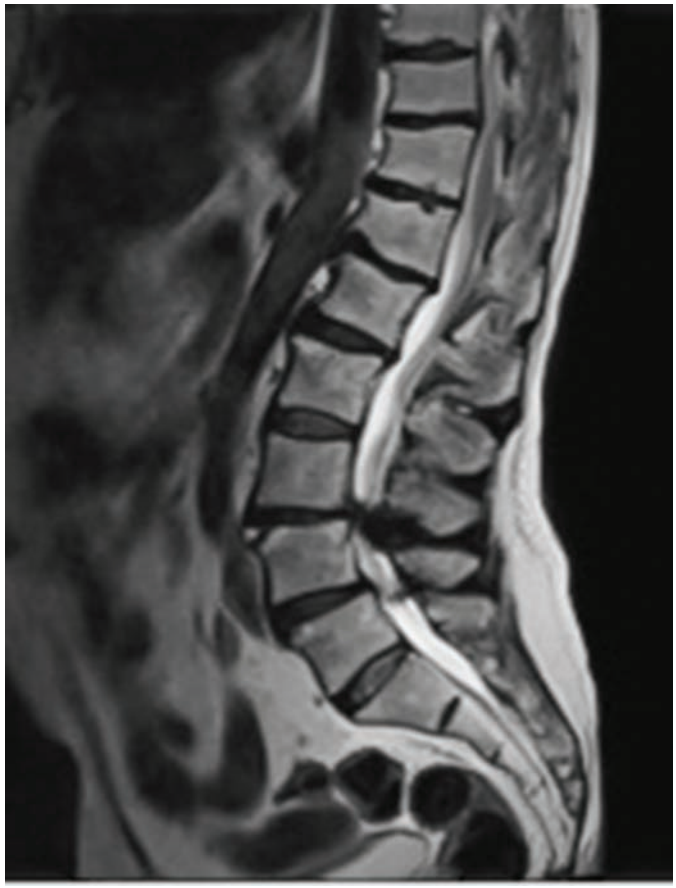
CANDIDATE: T2 sagittal MRI lumbar spine showing significant central stenosis at L3–L4 and also some compression at L4–L5. Axial T2 again suggests significant central stenosis with ligamentum flavum and facet hypertrophy at L3–L4 and moderate stenosis at L4–L5.

EXAMINER: With these investigations, how would you now manage this patient?

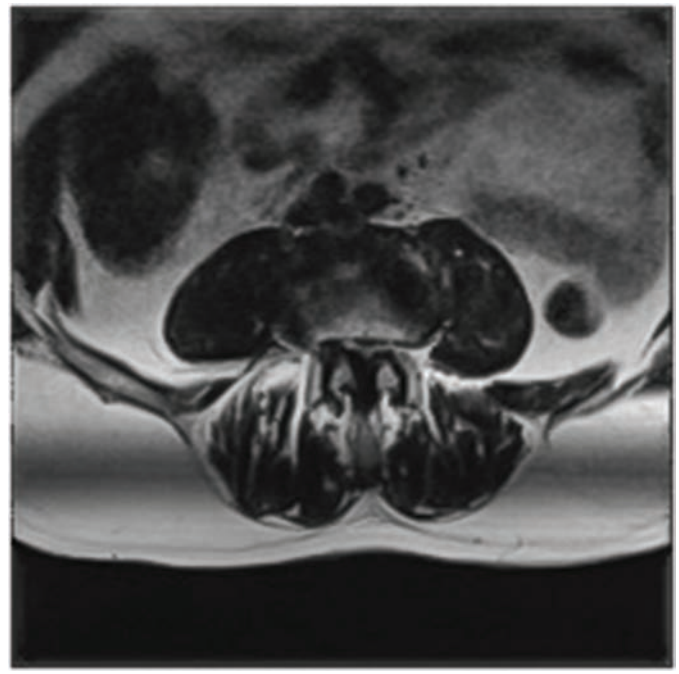
CANDIDATE: Though his MRI scan can explain all his symptoms, I would consider nerve conduction studies to rule out peripheral neuropathy due to diabetes especially if his blood investigations show poor control of his diabetes.

EXAMINER: I would like to argue otherwise, as he has no glove and stocking type of sensory loss. Consider that the nerve studies are normal.

(a) T2 Sagittal



(b) Axial L4/L5



(c) Axial L4/L5

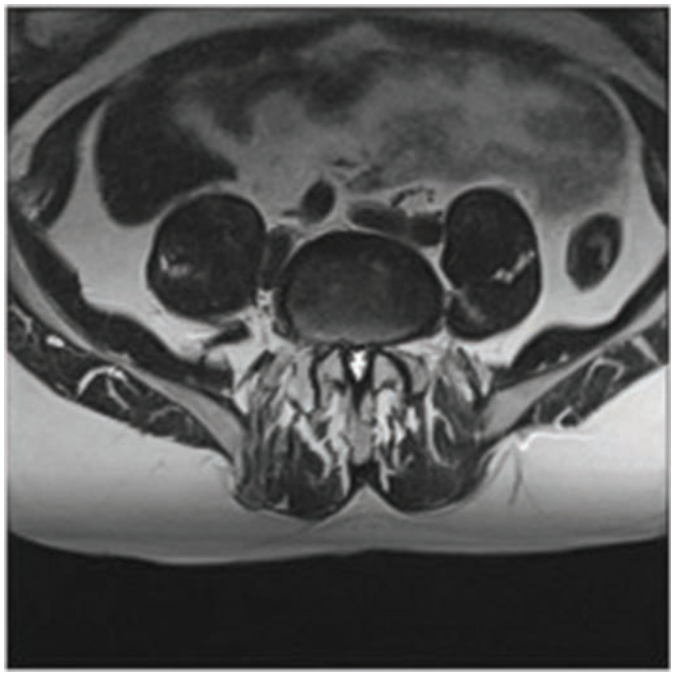


Figure 10.5 (a–c) MRI scans

CANDIDATE: He has tried non-operative measures as well as injections and now has rest pain. I will now refer him to a spinal surgeon for decompression and/or fusion.

EXAMINER: Would you offer fusion or not for this patient?

CANDIDATE: I will need flexion extension x-rays to help me with that decision. Fusion is indicated if there is instability like degenerative scoliosis, spondylolisthesis or removal of >50% of the facets during decompression. This patient probably needs fusion as he would need a large portion of the hypertrophied facet removed during surgery, as his compression is due to facet hypertrophy as well. Besides, he also has grade 1 spondylolisthesis at L3–L4.

EXAMINER: What are the outcomes after surgery?

CANDIDATE: In most patients there is 80% likelihood of excellent-to-good outcome 2 years after surgery. The SPORT trial provided a level 2 evidence that patients with symptomatic spinal stenosis treated surgically compared to those treated medically/interventionally maintain substantially greater improvement in pain and function through 4 years.

EXAMINER: Thank you.

Cervical spondylotic myelopathy

This is classic intermediate case material. This topic could very easily catch you out if you are not familiar clinically with either examination of the neck or specifically the clinical findings you would look for in cervical myelopathy.

Nerve lesions in the upper limb could be due to:

1. Cervical radiculopathy (spinal nerve root)
2. Cervical myelopathy (spinal cord)
3. Peripheral nerve lesions (median, ulnar nerve or radial)
4. Brachial plexus
5. Thoracic outlet syndrome (cervical rib)

The symptoms of cervical spondylotic myelopathy may include gait difficulties, decreased manual dexterity, paresthesias or numbness of the extremities, urinary frequency or urgency, generalized and extremity weakness.

Besides the routine history, of neck and/or arm pain ask for questions that help to find out sensory loss, ability to walk, bladder/bowel function and fine/gross motor loss in upper and lower limbs:

- Can you write?
- Do you drop things?
- Can you dress yourself?
- Has there been any change in passing urine (urinary frequency or urgency)?
- Has there been any disturbance in bowel function (sphincter disturbance)?
- Can you walk independently? Do you need aids to walk? Do you tend to lose balance?
- Do you have any loss of sensations?

Sometimes, the only symptom a myelopathy patient presents is loss of balance. Their diagnosis is then confirmed by clinical examination with upper motor neuron signs in lower limbs and upper or lower motor neuron signs in upper limbs.

Intermediate case: Cervical spondylotic myelopathy

GP note: 'Mr Cooke, a 61-year-old electrician c/o LBP and difficulty in walking in the last 8 months. Can you please take a history, examine this gentleman and discuss management.'

(NB. Don't be surprised with the above scenario. Cervical spondylotic myelopathy (CSM) is easily missed as it's a degenerative process associated with low back pain (LBP). In this scenario, only a summary of the history is provided.)

EXAMINER: Can you summarize your history please?

CANDIDATE: Mr Cook is a 61-year-old electrician who complains of low back pain, mild neck pain and gait disturbances since the past 8 months. He also has weakness in both his hands dropping objects and difficulty manipulating fine objects. He feels unstable on his feet, using a Zimmer-frame to walk and is presently housebound. He has no bladder or bowel disturbances and does not complaint of any sensory loss.

He has tried physiotherapy in past. He has no significant past medical problems and his main concern is loss of balance that is progressively getting worse.

EXAMINER: (*interrupting*) What are you suspecting based on your history?

CANDIDATE: Lumbar canal stenosis, cervical spondylotic myelopathy or peripheral neuropathy. I need do a neurological examination to arrive at a final diagnosis.

EXAMINER: Please carry on with your examination but focus on his neck.

CANDIDATE: On inspection from the sides there is loss of cervical lordosis. There are no scars on the back or front of the neck. He walks with a broad based ataxic gait and Romberg's test is positive.

EXAMINER: What is the cause of ataxic gait and positive Romberg's?

CANDIDATE: Joint position sense (or proprioception) is carried by the dorsal columns of the spinal cord. As these signals do not reach the brain, the patient suffers from sensory ataxia and to maintain balance walks with a broad based gait.

Romberg's test relies on the brain (cerebellum) receiving three sensory inputs. These are vision, vestibular apparatus in the inner ear and joint position (proprioception) carried by the dorsal columns of the spinal cord. If the visual pathway is removed by closing the eyes but the proprioceptive and vestibular pathways are intact, balance will be maintained. But if proprioception is defective, two of the sensory inputs will be absent and the patient will sway and lose balance.

EXAMINER: Please carry on with the examination.

CANDIDATE: Cervical spine flexion, extension, rotation and lateral flexion are moderately reduced. Shoulder movements are normal.

Neurological examination reveals normal nutrition but increased tone in both upper and lower limbs . He has grade 5 power in both C5, but grade 3 power in C6, C7, C8 and T1 bilaterally. Power is grade 4 both lower limbs. Sensations are normal both upper limbs and lower limbs but reflexes are brisk in all four limbs with up going plantars and positive Hoffmann's in both upper limbs. Joint position is lost in all four limbs. This is suggestive of upper motor neuron type of picture with the lesion being either in the brain or cervical spine. However, since he has no brain symptoms like headaches or weakness in cranial nerves, I would consider cervical myelopathy as my first diagnosis.

EXAMINER: What would you like to do?

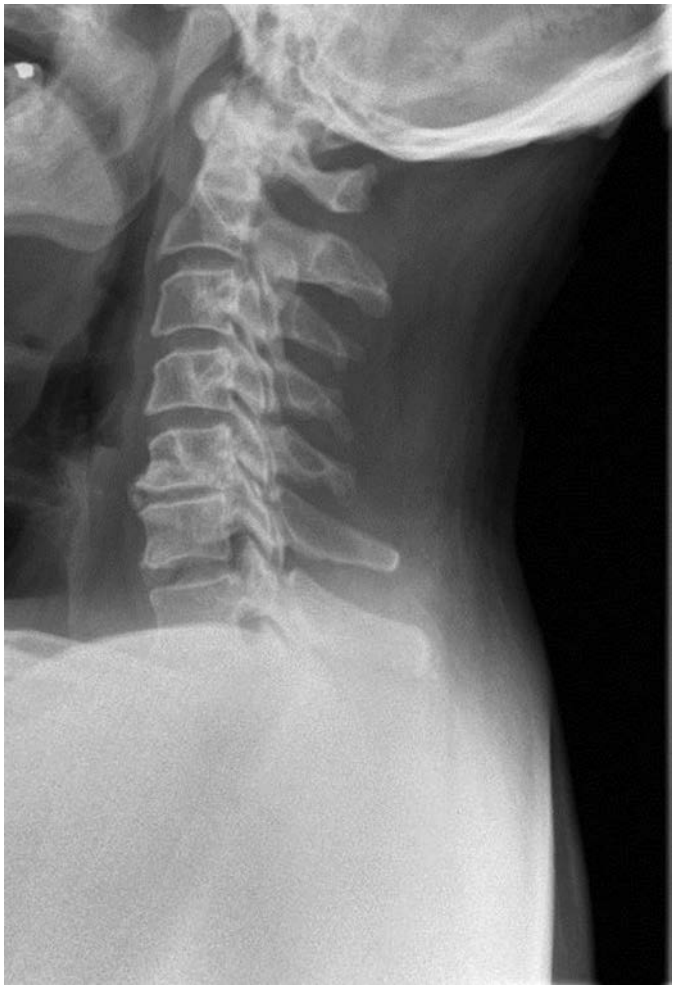


Figure 10.6 Lateral cervical spine x-ray

CANDIDATE: I would like to investigate him with x-rays and MRI of his cervical. Ideally, MRI of his entire spine as he still could have additional compression in his thoracic and lumbar spine.

EXAMINER: Consider MRI thoracic and lumbar is normal. Please comment on this lateral cervical spine x-rays? (Figure 10.6)

CANDIDATE: Lateral cervical spine x-ray showing some loss of cervical lordosis with anterior and posterior osteophyte formation at C5–C6. There is loss of disc height at C5–C6. There is no evidence of listhesis. I would like to see an x-ray AP view and MRI cervical spine.

EXAMINER: The AP view is normal; these are his MRI scans (Figure 10.7 a and b).

CANDIDATE: T2 sagittal MRI cervical spine showing significant central stenosis at C4–C5 and C5–C6. There is evidence of bright signal of the spinal cord at this level suggestive of myelomalacia. Axial T2 again shows significant central stenosis at these levels.

EXAMINER: With these investigations, how would you now manage this patient?

CANDIDATE: He has significant functional impairment with 2 level disease. The compression is mainly at the disc level with no compression at the vertebral level. I will refer him to a spinal surgeon for decompression and fusion.

EXAMINER: Would you offer anterior or posterior decompression and why?

CANDIDATE: Anterior surgery is indicated if the compression is mainly anterior (e.g. ossification of the posterior longitudinal ligament (OPLL)) or there is compression at two or fewer disc segments. Also, if there is fixed cervical kyphosis, anterior surgery should be considered.

Posterior cervical decompression with laminectomy and fusion should be considered in patients with multilevel compression and there is no fixed kyphosis.

This patient has two-level compression, mainly anteriorly; hence, I will offer anterior cervical discectomy and fusion with cage and bone graft.

EXAMINER: What about isolated laminectomy without fusion?

CANDIDATE: Isolated laminectomy is rarely indicated due to the risk of post-laminectomy kyphosis.

EXAMINER: The patient has read about cervical disc replacement for treatment of his neck problems over the Internet?

CANDIDATE: Although NICE recommends cervical disc arthroplasty in cervical radiculopathy and myelopathy, this patient has significant facet arthritis as seen on his x-rays and MRI. Besides kyphosis and probably osteoporosis at his age are contraindications.

EXAMINER: What are the outcomes after anterior surgery with fusion?

CANDIDATE: Although the primary goal of surgery in myelopathy is to prevent progression, most patients actually note neurological improvement after successful decompression and fusion. Ikenaga et al.[1] showed stable clinical results of >10 years in his series of 31 patients with anterior decompression and fusion. Adjacent disc degeneration had minimal effects on the long-term outcome.

EXAMINER: Thank you.

Cervical radiculopathy

The term implies compression on the nerve root with no compression of the central spinal cord. Radiculopathy can be due to disc osteophyte complex or a soft disc. Most common levels are C5–C6 and C6–C7. Remember that C5–C6 disc herniation will compress on the C6 nerve root while C6–C7 herniation compresses on the C7 nerve root.

Patients present with arm and /or neck pain. There will be no upper motor signs. If there are upper motor signs, suspect myelopathy due to cord compression.

Besides the routine history of neck pain, ask for more details about the radiculopathy. In other words, for exact path of radiation and to which fingers. For example, C6 radiation is to thumb, C7 to middle finger and C8 to little finger.

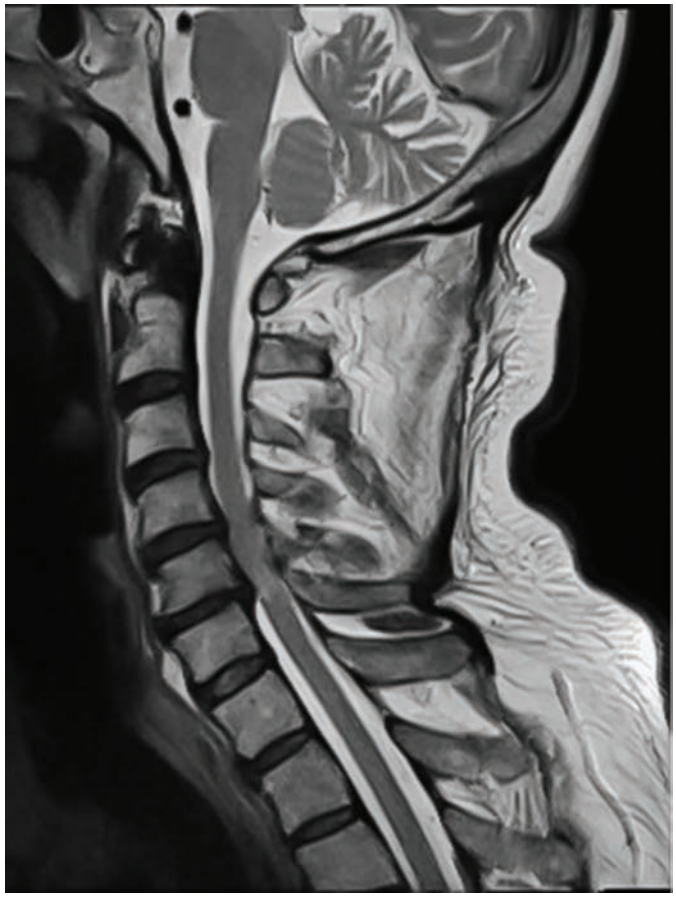
Enquire about sensory and motor loss and loss of function. Find out what all treatment has ben provided so far. This is of course apart from the routine history. Do not forget double crush syndrome with nerve lesion in neck and peripheral compression like carpal tunnel.

Intermediate case: Cervical disc prolapse with radiculopathy

GP note: 'Thirty-seven-year-old Mrs Brown who works as a receptionist c/o neck and left arm pain 6 months. Can you please take a history, examine this lady and discuss management.'

EXAMINER: *(After the history taking)* Can you summarize your history please?

(a) T2 Sagittal



(b) T2 Axial C5/C6

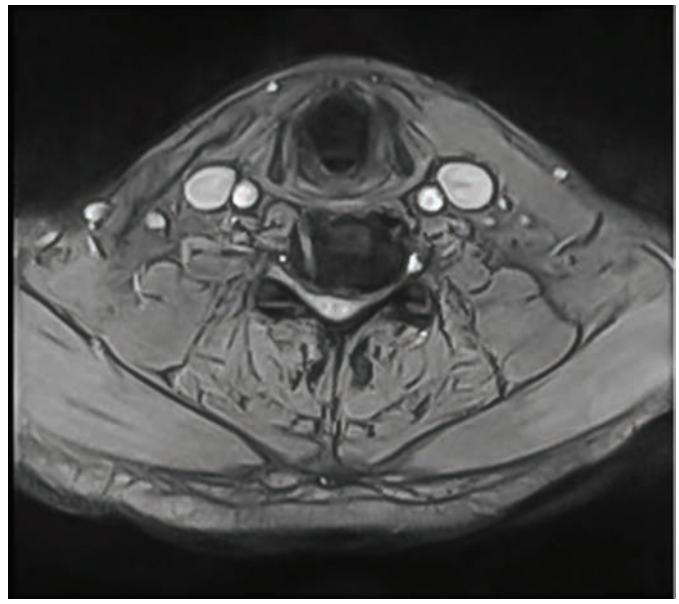


Figure 10.7 (a, b) MRI scans

CANDIDATE: Mrs Brown is a 37-year-old receptionist who complains of neck and left arm pain along C6 distribution. Arm symptoms are worse than the neck. She has no weakness in the arm and has tried physiotherapy and pain injections in her neck. She denies any bowel or bladder disturbances or gait disturbances.

EXAMINER: (*interrupting*) What are you suspecting based on your history?

CANDIDATE: C5–C6 disc prolapse with left C6 nerve compression.

EXAMINER: Why not a central disc protrusion with cord compression?

CANDIDATE: She denies any symptoms in her legs, no bowel or bladder disturbances or gait disturbances suggestive of myelopathy. However, I need to do a clinical examination to check for any long tract signs to rule out upper motor neuron lesion or central cord compression.

EXAMINER: Please carry on with your examination.

CANDIDATE: On inspection from the sides there is loss of cervical lordosis. There are no scars of previous surgeries on the back or front of the neck. Her gait is normal.

Palpation reveals mild paraspinal tenderness.

Cervical spine flexion, extension, rotation and lateral flexion are moderately reduced.

Spurling's test is positive on the left (simultaneous extension, rotation to affected side, lateral bend, and vertical compression reproduces symptoms in ipsilateral arm).

Neurological examination reveals normal nutrition and normal tone. She has grade 3 power in her left biceps. Rest of the motor examination was normal. She also has reduced sensation in left C6 dermatome.

Reflexes are normal. Hoffmann's is negative in both upper limbs suggestive of no upper motor neuron lesion.

I would like to examine both shoulders to complete my examination.

EXAMINER: Consider both shoulders are normal, what would you like to do?

CANDIDATE: I would like to investigate with x-rays and an MRI of her cervical spine.

EXAMINER: Please comment on this lateral cervical spine x-rays?

CANDIDATE: Lateral cervical spine x-ray (Figure 10.8) showing straightening of the cervical spine. There is reduced disc height at C56 with posterior osteophyte formation. I would like to see AP view and MRI cervical spine.

EXAMINER: AP view is normal. These are his MRI scans. (Figure 10.9 a and b)

CANDIDATE: T2 sagittal MRI cervical spine showing disc protrusion at C56. Axial T2 again shows left-sided disc protrusion at C56. The disc bulge is not dark suggesting this is a soft disc. There is no compression of the spinal cord or any altered cord signal.

EXAMINER: How would you now manage this patient?

CANDIDATE: She has tried analgesics, physiotherapy and injections. Her symptoms are for 8 months now and she is struggling with her activities of daily living as well as her job. I will discuss surgery with her and refer her to a spinal surgeon.

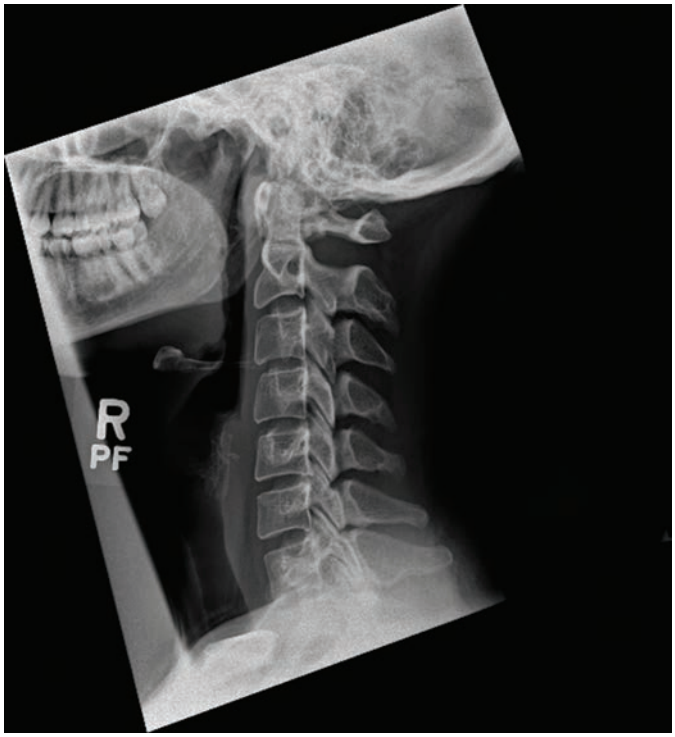


Figure 10.8 Lateral cervical spine x-ray

EXAMINER: What surgery?

CANDIDATE: Options include anterior cervical discectomy and fusion, anterior cervical discectomy and disc replacement or posterior foraminotomy.

Her main concern is arm pain with no or mild neck pain. Target level is C5–C6. She has no facet arthritis at C5–C6. I will offer her a C5–C6 discectomy and disc replacement.

EXAMINER: I would offer her C5–C6 discectomy with fusion.

CANDIDATE: This is debatable. Some surgeons consider fusion as gold standard. NICE has published guidelines for cervical disc replacement. They have also provided evidence for the same. In a randomised controlled trial of 541 patients, patients reported greater improvement from baseline in the mean Neck Disability Index (NDI) score in patients treated with prosthetic cervical disc insertion compared with fusion at 3-month follow-up; but this difference was not significant at 6-, 12- or 24-month follow-up. The arthroplasty group had lower rate of secondary surgeries as well.

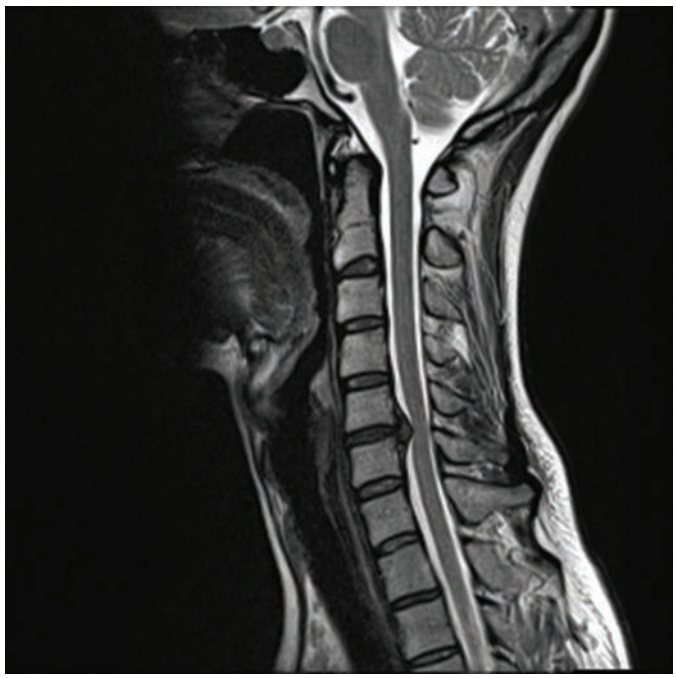
EXAMINER: You mentioned about posterior foraminotomy?

CANDIDATE: Ideal indication for a posterior foraminotomy is a soft one-level disc with unilateral compression. This has a good success rate avoiding risk of anterior injury. This is also an option in this case, but type of surgery is dependent on surgeon choice.

EXAMINER: If this were a revision anterior surgery, would you have any concerns?

CANDIDATE: Yes. I will refer her to ENT specialist for laryngoscopy to check vocal cord function. She could have asymptomatic damage to the unilateral recurrent laryngeal nerve on the side of the previous approach. If that nerve is damaged, anterior approach on the non-operated side could lead to damage of the recurrent laryngeal nerve on that side,

(a)



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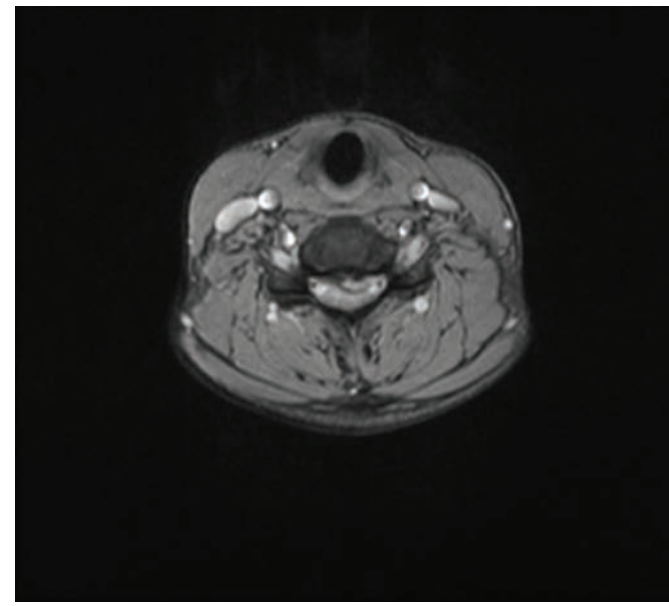


Figure 10.9 (a, b) MRI scans

resulting in bilateral recurrent nerve palsy. This can be catastrophic leading to difficulty in breathing and inability to speak. Checking the vocal cords by laryngoscopy is vital for medicolegal purpose too.

EXAMINER: Thank you.

You may then be asked to consent a patient for anterior surgery, talk about the approach or complications of anterior cervical spine surgery.

Ankylosing spondylitis

Ankylosing spondylitis is a chronic autoimmune inflammatory spondyloarthropathy primarily affecting the axial skeleton with variable involvement of peripheral joints and non-articular tissues. It is seronegative arthritis (Rh factor negative) with positive HLA B27 (90% of patients). It usually affects males and has a strong genetic predisposition.

Diagnosis

As per the Modified New York criteria[2] (NICE guidelines), for diagnosis of ankylosing spondylitis one radiological criterion and at least one clinical criterion are to be satisfied.

Radiological criterion:

- Sacroiliitis at least grade 2 bilaterally or grade 3 or 4 unilaterally

Clinical criteria:

- Low back pain and stiffness for >3 months that improves with exercise but is not relieved by rest
- Limitation of motion of the lumbar spine in both the sagittal and frontal planes
- Limitation of chest expansion relative to normal values correlated for age and sex

All reasonable measures should be taken to ensure that symptoms are due predominantly to ankylosing spondylitis and that alternative causes, including spinal fracture, disc disease and fibromyalgia, are excluded.

Orthopaedic manifestations

- Bilateral sacroiliitis progressing to frank ankyloses
- Spine: loss of movements, ankyloses, kyphotic deformity, fractures
- Other joint involvement: hips, knees, shoulders and ankle
- Enthesopathy: inflammation of enthuses or tendon insertion (tendoachilles most commonly affected)

As evident above, there are many orthopaedic facets to this condition. Examiners can focus on one or two of these for a short case, or a more thorough assessment can form the basis of an intermediate case. There are good clinical signs present with a lot to discuss.

Non-articular manifestations

- Eyes: Acute anterior uveitis
- Heart: Conduction defects, aortitis, aortic regurgitation or stenosis
- Respiratory: Pulmonary fibrosis
- Renal: Amyloid nephropathy
- Gastrointestinal: Associated with Crohn's disease and ulcerative colitis

Besides orthopaedic complaints, take a history regarding the non-articular manifestations as since these may significantly affect the outcome.

Intermediate case: Ankylosing spondylitis

GP note: 'Forty-year-old Mr Smith complains of LBP and back stiffness. Can you please take a history, examine this gentleman and discuss your management.'

EXAMINER: Can you summarize your history please? (after history taking)

CANDIDATE: Mr Smith is a 40-year-old gentleman complaining of back pain since the past 17 years, insidious on onset and gradually getting worse. Pain is in his entire spine, worse in the sacroiliac region and thoracolumbar spine, and accompanied by stiffness. He has developed a stooping posture of his spine that causing difficulty in seeing forwards when he walks. He has no weakness in any of his limbs or problems with his waterworks.

He has no major complaints form his hips, but has occasional pain in his heel. However, his spine is his main concern.

Mr Smith has difficulty in breathing but has no eye, heart or renal problems. He has no allergies . . .

EXAMINER: *(Interrupting)* Please go ahead with the examination.

CANDIDATE: *(After making the patient stand)* On general inspection we have a male patient who is somewhat stooped as he walks into the room. On examination from behind the shoulders are at same level, there is loss of normal lumbar lordosis, a fixed kyphosis of his thoracic spine. The chin to brow angle appears normal indicating no cervicothoracic kyphosis. His visual axis does not appear to be horizontal. His pelvis is level but he tends to stand with a slightly flexed attitude of his hips and knees. The patient has a classic question mark posture with pronounced thoracolumbar kyphosis and flexion attitude of the hips and knees.

On palpation, he has no significant tenderness of his spine, but he does have tenderness of his left sacroiliac joint.

On examination of his neck movements there was very limited flexion and extension present. There is almost no lateral movement of his cervical spine. Examining his lumbar spine reveals a gross restriction of all movements. In particular he tends to flex his hips when bending forwards to compensate for a stiff spine.

Schober's test for lumbar forward flexion was 3 cm (normal 5 cm or more), which is markedly reduced. The wall test unmasks a fixed kyphotic deformity of the spine. He is unable to stand with his back flush against the wall. Flexion, abduction and external rotation of the hip joint (FABER test) produces severe pain of the sacroiliac joints.

Maximum chest expansion from full expiration to full inspiration measured at the level of the nipples is reduced to 3 cm compared to a normal expansion of 7 cm. The patient is breathing predominantly by diaphragmatic excursion, which is the cause of his protuberant abdomen.

Neurological examination of both lower limbs reveals normal tone, power, sensations and reflexes.

I would now like to do a detail examination of his hips.

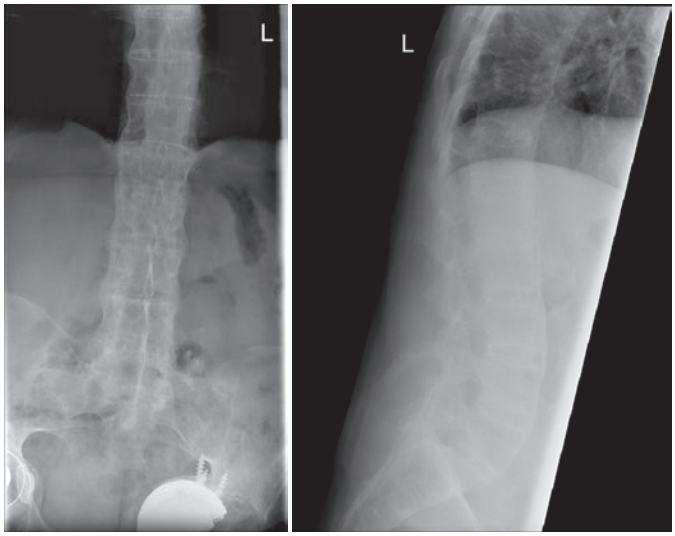


Figure 10.10 AP and lateral radiographs demonstrating bamboo appearance AS spine

EXAMINER: Consider that the hips are asymptomatic and are normal on examination. So what is your diagnosis?

CANDIDATE: I would first like to have standing x-rays of his entire spine with sacroiliac joints to make a diagnosis?

EXAMINER: Why?

CANDIDATE: Mr Smith has sign and symptoms of ankylosing spondylitis. However, to label a patient with this diagnosis, I need one radiological and any one of the clinical criteria. He has all three clinical criteria, which are:

Low back pain and stiffness for >3 months that improves with exercise but is not relieved by rest

Limitation of motion of the lumbar spine in both the sagittal and frontal planes

Limitation of chest expansion

I need one radiological criteria that is sacroiliitis.

EXAMINER: These are his x-rays (Figure 10.10)

CANDIDATE: AP x-ray showing left sacroiliac joint erosion and right sacroiliac joint ankylosis. The spine AP x-ray shows syndesmophytes formation with ossification of the spinal ligaments leading to 'Bamboospine' appearance. The lateral x-ray shows squaring of the vertebrae with marginal syndesmophytes. I can't appreciate any fractures on the x-rays.

I need to see full spine x-rays to see for sagittal balance as well as pelvis with both hips. This AP x-ray has limited view of the sacroiliac joints as well.

EXAMINER: What is the difference between osteophytes and syndesmophytes?

CANDIDATE: Osteophyte formation begins at the site of Sharpey fibers attachment between the annulus fibrosis and anterior margin of vertebral body just above or below the vertebral endplate. Osteophytes begin typically by growing outward and finally meet the osteophyte on the other side of the disc space forming bridging osteophytes. Syndesmophytes on the other hand are ossifications of the annulus fibrosis and are more vertically oriented attaching right at the endplate margin.

EXAMINER: Do you want a MRI scan of the spine in this patient?

CANDIDATE: No. He denies any history of trauma; there is no neurology, acute onset of pain or point tenderness in his spine. MRI is indicated if suspecting fracture, or presence of any neurology especially since patients of ankylosing spondylitis are known to have epidural hemorrhage. MRI of the sacroiliac joint is also indicated in early part of the disease process to identify sacroiliitis.

EXAMINER: So, how do you manage this patient?

CANDIDATE: MY main concerns in this patient are pain and thoracolumbar deformity. I would like to have a multidisciplinary approach involving the rheumatologist, physiotherapist, orthotics, pain team and a spinal surgeon as well.

EXAMINER: Consider that his pain is now under control but he has problems regarding his posture with loss of horizontal gaze.

CANDIDATE: I will refer him to a spinal surgeon for considering a kyphotic deformity correction. The spinal team may consider osteotomy at the thoracolumbar region, like a pedicle subtraction osteotomy. Indications for osteotomy are poor function, lack of horizontal gaze, cosmesis and fatigue pain from neck and hip extensors.

EXAMINER: If this patient presents with acute back pain, are you concerned?

CANDIDATE: Yes, this could be due to a vertebral fracture especially if history of fall or trauma. Fractures are not uncommon due to altered biomechanical properties of the spine. The ossified spine creates long lever arms limiting the ability to absorb even small impacts. Besides, there is osteoporosis due to stress shielding, immobility and inflammatory process. These fractures are invariably missed and are associated with high incidence of neurological complications and pseudoarthrosis.

EXAMINER: Thank you

Other points for discussion

1. Preoperative anesthesia concerns
2. Consenting a patient for spinal surgery
3. Approaches and complications of spinal surgery
4. Medical management of ankylosing spondylitis
5. Patient with spine and hip involvement, order of surgery
6. Cervicothoracic fractures and deformity management

Scoliosis

Scoliosis is defined as spinal curvature in the coronal plane >10°. However, there is usually associated deformity in sagittal plane and transverse plane (vertebral rotation). It can feature as intermediate case or a short case in the exam.

Etiology

Scoliosis is a descriptive term and not a diagnosis.

1. **Idiopathic** (80% cases)
   a. Infantile: 0–3 years
   b. Juvenile: 3–10 years
   c. Adolescent: 10+ years

2. **Congenital** (present at birth)
   a. Failure of formation – Hemi vertebrae
   b. Failure of segmentation – Unilateral unsegmented bar
   c. Mixed
3. **Neuromuscular**
   a. Upper motor neuron: Cerebral palsy
   b. Lower motor neuron: Polio
   c. Muscular weakness: Muscular dystrophies
4. **Others**
   a. Syndromes: Marfan's, Ehlers–Danlos, neurofibromatosis
   b. Tumours: Osteoid osteoma
   c. Trauma
   d. Compensatory: Leg length discrepancy

Questions to be answered in assessing a case of scoliosis

1. Etiology? Idiopathic or non-idiopathic (neurology, syndromic features)
2. Region involved? Lumbar, thoracic, thoracolumbar, involvement of pelvis or cervical spine
3. Structural or non-structural scoliosis? Structural scoliosis is irreversible lateral curvature of the spine with rotation of the vertebral bodies. Non-structural scoliosis is a reversible lateral curvature with no rotation of the vertebral bodies
4. Neurological involvement? Asymmetric abdominal reflexes, weakness in any of the limbs, upper motor neuron signs
5. Risk of progression? Age of the patient, menarche in females, Risser's grading of pelvis or PA view of hand and wrist
6. Severity of the curve? Mild (10–25°), moderate (26–40°), severe (>40°)
7. Other systems involved? Syndromic scoliosis may have significant cardiorespiratory decompensation, sufficient to be unfit for major surgery
8. Is it painful? Painful scoliosis is always pathological, e.g. osteoid osteoma

Reading x-rays of the spine in scoliosis

- Confirm if standing and make sure entire spine is in view with PA, lateral and side-ending films (to assess flexibility of the curve)
- Region involved – Lumbar, thoracic, thoracolumbar curve
- Number of curves – Single or double
- Apex of the curve – Vertebrae furthest away from the midline
- Cobb angle measurement – Identify the end vertebrae that have the pedicle levels with the greatest tilt from the horizontal. Cobb angle is the angle between these two vertebrae
- Risser's sign – Ossification of the iliac apophysis begins laterally (anterior superior iliac spine (ASIS)) and proceeds medially (posterior superior iliac spine (PSIS)) to eventually cap the entire iliac crest. Risser's 1–5 is a measure of skeletal maturity and, therefore, a predictor of curve progression. Risser's 0 means no ossification center visible. Risser 0 and Risser 5 are similar on x-rays with no appearance of ossification centers. However, they are easily distinguished by age with Risser's 0 at 5 years and Risser's 5 after 16 years of age

What are the indications of MRI?

- Presence of neurology
- Abnormal abdominal reflexes
- Severe curves or rapid progression of curves
- Severe kyphosis
- Atypical curve: left thoracic, apical kyphosis, short angular curve
- Foot deformities

Intermediate case: Idiopathic adolescent scoliosis

This is the commonest form of scoliosis, usually a female patient accompanied by her parent. Most common is right thoracic curve. This form of scoliosis is not associated with significant back pain, fatigue or neurological symptoms. If present, it is non-idiopathic scoliosis that needs to be investigated to find the cause. Some patients with adolescent idiopathic scoliosis (AIS) may have low back pain that is common in adolescence in general. However, it is felt that the curvature itself does not result in back pain.

GP note: 'Referring to you 12-year-old Ms Leanne who's mum is concerned about a curvature in her back. Mum and daughter both are worried if this may worsen. Can you please take a history, examine this pleasant girl and discuss management.'

(In this scenario, only a summary of the history.)

EXAMINER: Can you summarize your history please?

CANDIDATE: Ms Leanne is a 12-year-old girl, otherwise fit and well. Her mum noticed Leanne developed a spinal curvature when she was 10 years of age. This has gradually progressed but is not associated with any pain. She denies any difficulty in breathing or any chest pain. She has no weakness in her legs or problems with her bowel or bladder. Leanne has noticed that her clothes don't fit well as they did previously. There is no family history of similar problems. She has tried a brace which her mum feels is not helping. She had her menarche at 11 years of age. The family's main concern is cosmesis and progression of the deformity.

EXAMINER: (*interrupting*) What are you suspecting based on your history?

CANDIDATE: Adolescent idiopathic scoliosis

EXAMINER: Why not non-idiopathic scoliosis?

CANDIDATE: Female, age of presentation, no back pain, no neurological complaints, no symptoms of other systems. Besides AIS is the most common type of scoliosis. However, I need to do examination and investigations to confirm my diagnosis.

EXAMINER: Please carry on with your examination.

CANDIDATE: On general examination, she is moderately built with no obvious features of any syndrome. On inspection from the back, the left shoulder appears to be at a higher level. There is waistline asymmetry with the body shifted to the right. There is a right convex thoracolumbar scoliosis. There is a rib hump on the right that becomes more prominent on bending forward. The skin over the back appears normal with no café-au-lait spots, tuft of hair or lipoma. On inspection from the sides, there is hypokyphosis of the thoracic spine with normal lumbar and cervical lordosis. On inspection from the front, the chest appears to be normal with no deformity.

The gait appears normal and she is able to do heel toe walking.

On palpation, there is no localized or generalized tenderness in the spine. The spinal ROM is reduced in all three planes.

On sitting the curve does persist. On laying her supine, there is no leg length discrepancy. There is no motor, sensory deficit in both lower limbs and tone is normal. Reflexes are normal and plantars are down going. Abdominal reflexes are normal.

EXAMINER: What is the significance of normal abdominal reflexes?

CANDIDATE: It suggests there is no intra-spinal cord pathology like a syringomyelia, diastometamyelia, tethered cord and Arnold–Chiari malformation. If they are abnormal, she will need an MRI scan of her spine to rule out any intraspinal pathology. Syrinx may cause the scoliosis in the first place. The syrinx needs to be surgically treated by a neurosurgeon prior to scoliosis correction. If scoliosis correction is carried out first, then it carries a risk of temporary or permanent neurological damage.

EXAMINER: What would you like to do?

CANDIDATE: I would like to have standing full spine PA, lateral and side-bending views.

EXAMINER: Please comment on these x-rays? (Figures 10.11, 10.12, 10.13 a and b)

CANDIDATE: Standing AP spine x-ray shows a single right-sided convex thoracolumbar scoliosis with the apex at T12. There is loss of coronal balance with the C7 plumb line falling to the right of central sacral line. The Cobb angle roughly measures around 50°. The pelvis shows Risser's 1 stage base of <25% of the calcification of the lateral iliac apophysis.

The lateral spine x-ray shows loss of thoracic kyphosis but the sagittal balance is well maintained.

The side-bending x-ray shows that the curve does not correct fully on the side of the convexity.

EXAMINER: Do you need a MRI scan?

CANDIDATE: No. MRI scan is not indicated. She has no features of any syndrome, there is no neurology, abdominal reflexes are normal, its not a atypical curve like a left-sided curve, acute angular curve, there are no foot abnormalities or presence of significant kyphosis. If she had any of these, I would have requested an MRI scan of the full spine.

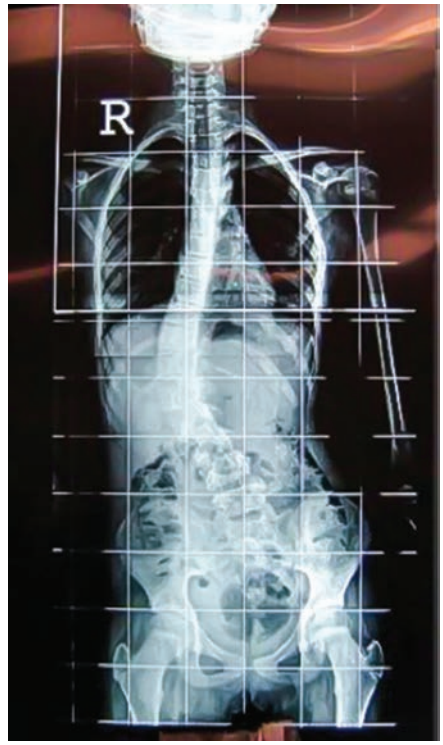


Figure 10.11 Standing AP spine x-ray

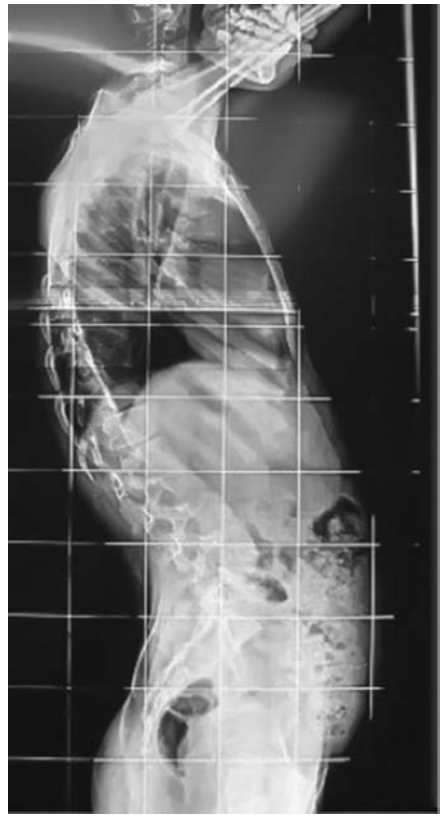


Figure 10.12 Lateral spine x-ray

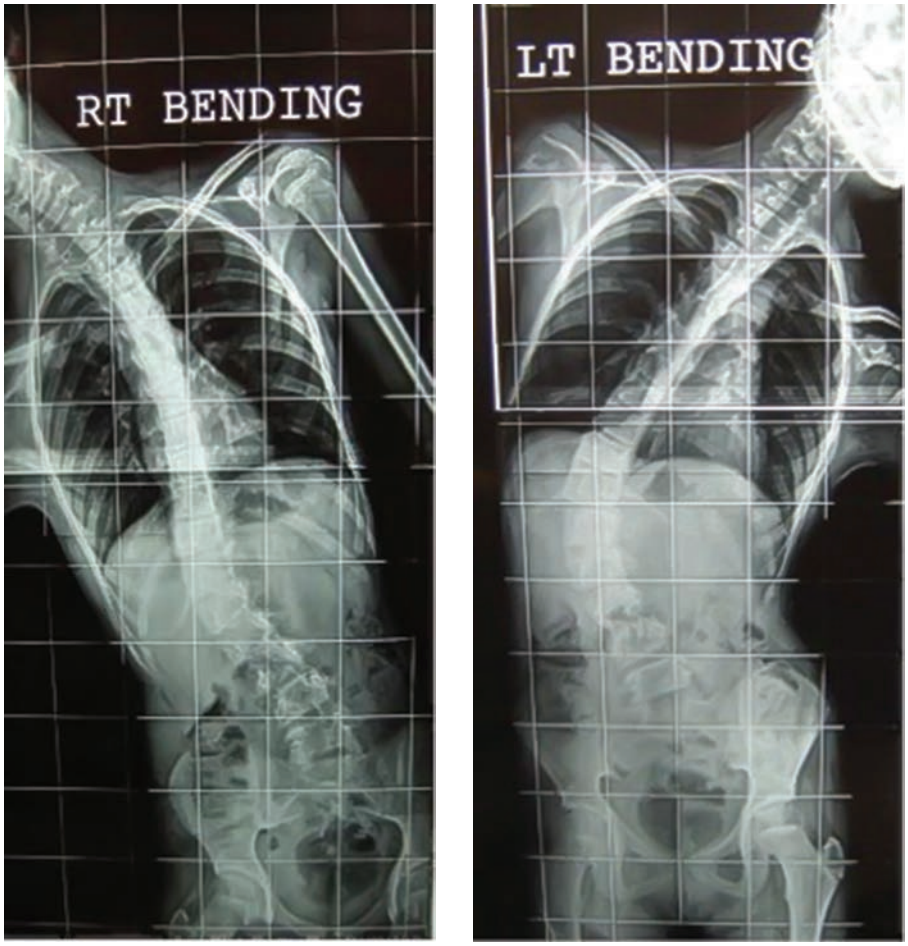


Figure 10.13 (a, b) Side-bending spine x-rays

EXAMINER: How would you manage her now?

CANDIDATE: I would refer her to a spinal surgeon for considering scoliosis correction surgery.

EXAMINER: Why not bracing?

CANDIDATE: This is a severe curve, more then 40° and this needs surgery. Besides, she is Risser's 1 with menarche just about a year ago, she is definitely likely to progress. Besides, she is not happy with the shape of her back.

EXAMINER: What do you think they will do?

CANDIDATE: They will consider correction with posterior spinal fusion. If the curve is very severe, like 70°, they would consider anterior + posterior spinal fusion.

EXAMINER: You mentioned Risser's sign as to progression of the curve. Is there any other investigation that can help?

CANDIDATE: Yes, PA x-ray of the hand and wrist. It helps in determining skeletal maturity. Skeletal maturity is defined as Risser's 4, 2 years post menarche or <2 cm change in height in two visits 6 months apart.

EXAMINER: What is the role of SSEP (somatosensory evoked potential) during scoliosis correction?

CANDIDATE: SSEP is intraoperative neurophysiological monitoring that is indicated in corrective spinal surgery procedures when there is potential risk to the spinal cord, like severe curve corrections. They provide information about the central and peripheral nervous system and guide the operating team to prevent iatrogenic damage. If neurological injury is suspected, then the operating team may consider a Stagnara wake-up test.

EXAMINER: Thank you.

Other points for discussion

1. Preoperative anesthesia concerns – Respiratory compromise
2. Consenting a patient for scoliosis spinal surgery
3. Approaches and complications of scoliosis spinal surgery
4. Neurophysiological monitoring
5. Estimating residual growth (Risser's, Tanner's stages, hand and wrist x-ray, menarche)
6. Classification of AIS (Lenke, King classification)

Short case: Non-idiopathic scoliosis

EXAMINER: Please examine this 8-year-old boy focusing mainly on the spine.

CANDIDATE: On general examination, the left lower limb appears to be smaller than the right. He has a short limb gait.

On inspection from the back, there are multiple café-au-lait spots measuring >15 mm. I would like to examine for axillary and inguinal freckling that would suggest type 1 neurofibromatosis.

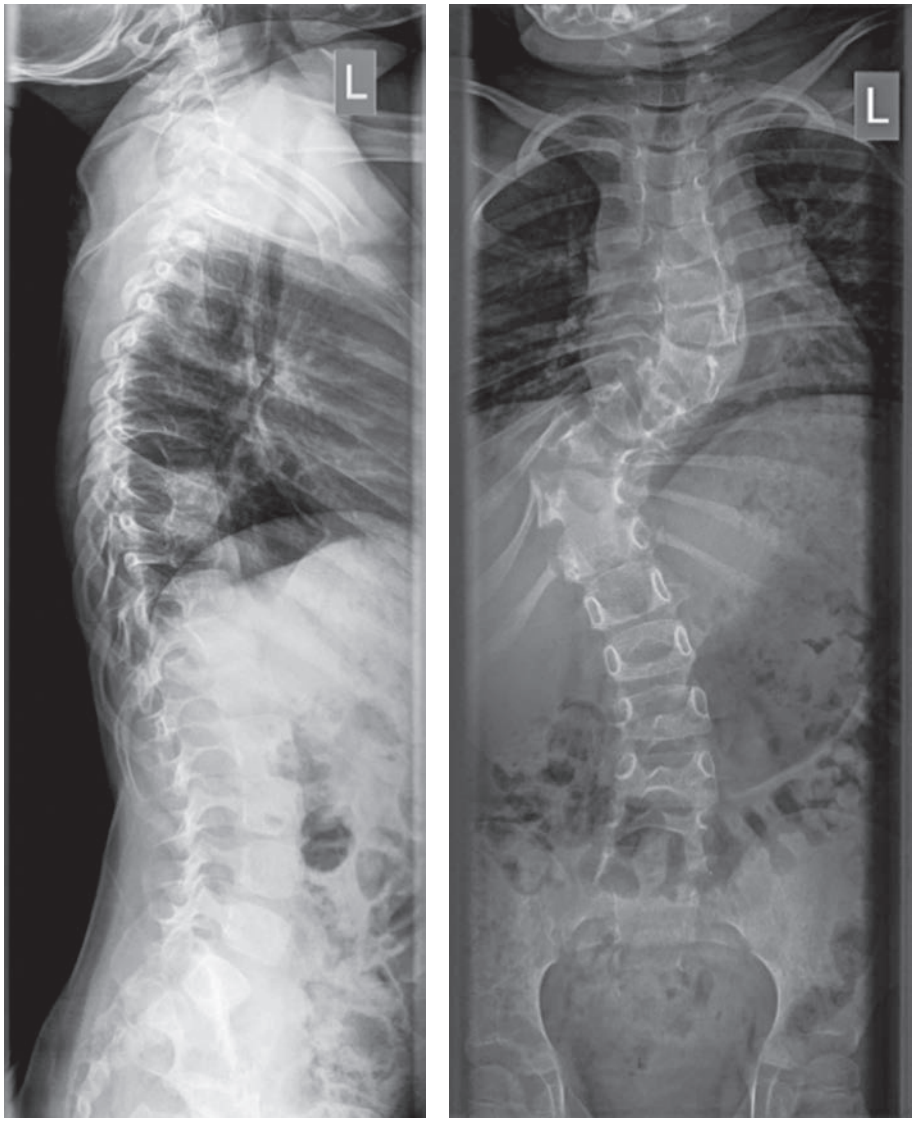


Figure 10.14 Standing AP and lateral spine x-rays

EXAMINER: Yes, it is neurofibromatosis; now please focus on the spine.

CANDIDATE: On inspection from the back, there is a right convex thoracic scoliosis with the apex roughly at T10. Both shoulders appear to be at the same levels.

On inspection from the sides, there appears to be normal sagittal balance. On inspection from the front, the chest appears to be normal with no deformity.

He is able to do heel–toe walking.

On palpation, there is no localized or generalized tenderness in the spine. The spinal ROM is reduced in all three planes.

On sitting the scoliosis persists, suggesting it's not due to leg length discrepancy.

On laying him supine, there is leg length discrepancy; the left limb is shorter than the right. There is no motor deficit in both lower limbs and tone is normal. Sensations are reduced in right L4, L5 and S1 dermatome. Reflexes are normal and plantars are down going. Abdominal reflexes are normal.

EXAMINER: How would you manage him?

CANDIDATE: I need to take a detail history to find out his symptoms. I also need investigations like full spine standing x-rays and MRI scan of the spine. I also need to know if this is dystrophic or non-dystrophic type of neurofibromatosis scoliosis. Dystrophic scoliosis is short-segmented sharp curve with involvement of ribs and vertebrae. It is associated with kyphosis and high rate of post-surgery pseudoarthrosis. Non-dystrophic curves behave like adolescent idiopathic scoliosis.

This curve appears to be dystrophic being short segment.

Also, I need to know if he has other orthopaedic complaints like tibial pseudoarthrosis. I can already see that he has hemi-hypertrophy of his right lower limb.

EXAMINER: Please comment on these standing x-rays (Figure 10.14)?

CANDIDATE: Standing AP spine x-ray shows a short right-sided convex thoracolumbar scoliosis with the apex at T10. There are features of dystrophic vertebral scalloping, pencilling of ribs. The Cobb angle roughly

measures around 70°. It is difficult to comment on Risser's grading as I can't see the entire iliac crest.

The lateral x-ray depicts normal coronal balance. There is evidence of thoracic hypokyphosis.

I need an MRI scan of his spine to see for any intraspinal lesions.

EXAMINER: Consider his MRI scan to be normal.

CANDIDATE: Unusual for the MRI scan to be normal considering the x-ray changes of enlarged neural foramina, scalloping of vertebrae and sensory deficit.

However, if MRI scan shows no intraspinal lesions and spine cosmesis is his main concern, I will refer him to a spine-deformity correction surgeon.

EXAMINER: What are the associated complications?

CANDIDATE: High rate of pseudoarthrosis up to 40% with posterior surgery. Some surgeons recommend bone grafting as well in same sitting.

EXAMINER: Thank you.

Short case: Foot drop

EXAMINER: Can you please examine the right foot of this 40-year-old patient? You can ask one question if you like.

CANDIDATE: Hi, My name is Mr K, one of the exam candidates. Can you please tell me what's wrong with your right foot?

PATIENT: I have weakness in my right foot since past 1 year. It all started with back and right leg pain that is not there anymore; however, this weakness concerns me.

EXAMINER: Please carry on with the examination.

CANDIDATE: On inspection, there is wasting of the muscles in the anterior and lateral compartment of the leg. He is using ankle foot orthotic splint on the right side. There are callosities in the forefoot over the plantar aspect.

Can you please walk for me?

He walks with a high steppage gait with exaggerated flexion of the hip and the knee to prevent toes from catching on the ground during the swing phase. There is absence of the first rocker on the right and his foot slaps on the ground at initial contact. Heel walking is absent on the right.

There are no scars in the low back, buttocks or around the neck of fibula suggestive of previous surgery or trauma.

I would like to perform a detail neurological examination.

EXAMINER: Please focus in the foot.

CANDIDATE: Ankle dorsiflexors (L4–L5, mainly L4) is grade 1/5, plantar flexion 5/5 (S1, S2), EHL 5/5 (L5), eversion (L5–S1) 5/5, inversion(L4–L5) 3/5. Sensations are reduced over the L4 dermatome (medial aspect of the foot). Tone is normal and Babinski is downgoing suggesting the lesion is LMN type.

SLR bilaterally is 80°.

Examination suggests a L4 nerve root involvement, most probably from his spine as he had back and leg pain. However, to confirm this, I need to do a detail neurological examination.

EXAMINER: I say it's coming from a common peroneal nerve injury following a fibula neck fracture. Am I right in saying so based on your examination?

CANDIDATE: I need to check the knee extensors (L3–L4) and the hip abductors (L4–L5, mainly L5). If the power is normal in the hips and knees, with history of trauma to the fibula neck, it would suggest common peroneal nerve involvement and not spinal etiology.

EXAMINER: If it's coming from the spine, what level is the compression?

CANDIDATE: L4 nerve root involvement due to posterolateral disc L3–L4 or far lateral disc L4–L5.

EXAMINER: What are other causes of foot drop?

CANDIDATE:

- Brain: cerebral palsy, stroke, multiple sclerosis
- Spine: Prolapsed disc, polio, syringomelia
- Nerve: Peripheral neuropathy (diabetes), hereditary motor and sensory neuropathies (HSMN), fibula neck fracture, total hip arthroplasty/total knee replacement iatrogenic injury, knee dislocation, nerve tumors
- Muscle: Muscular dystrophies

EXAMINER: Do you think he will benefit from spinal surgery if he had prolapsed disc?

CANDIDATE: He denies any leg pain now and SLR is not restricted. In other words, this is a painless foot drop that I feel will not benefit from lumbar discectomy or decompression.

Management now is mainly orthotics and physiotherapy. In rare circumstances surgery in the form of tendon transfer or arthrodesis.

EXAMINER: Thank you.

Short case: Spondylolisthesis

EXAMINER: Can you please examine this pleasant 17-year-old girl focusing on her spine?

CANDIDATE: Hi, My name is Mr K, one of the exam candidates. Can you please walk for me?

Her gait appears normal.

On inspection from the back, both shoulders and pelvis are at same level. There is no evidence of any scoliosis. There are no scars of previous surgery. On inspection from the sides, there is loss of lumbar lordosis with positive sagittal balance. The knees and hips are in flexed posture, probably trying to maintain sagittal balance.

On palpation, there is a step, roughly at L5–S1 level. There is central and paraspinal tenderness at this level. Spinal flexion and extension are reduced.

On lying down supine, there is no leg length discrepancy. SLR is about 50° bilaterally with hamstring tightness.

Neurological examination reveals normal tone. EHL (extensor hallucis longus) and ankle dorsiflexors on the right are 4/5; power is normal in remaining myotomes. Sensations are reduced in right L5 dermatome. Reflexes are normal bilaterally.

EXAMINER: What's your diagnosis?

CANDIDATE: Considering her age with a positive step sign and positive sagittal balance L5–S1 spondylolisthesis.

EXAMINER: Please comment on her lateral x-ray (Figure 10.15).

CANDIDATE: Assuming this is a standing x-ray, there is L5 lysis with spondyloptosis of L5 over S1. Pelvic incidence is also increased. I need full spine x-rays to check for sagittal balance besides her spine AP x-rays. Clinically and based on this lumbar spine lateral radiograph, she seems to

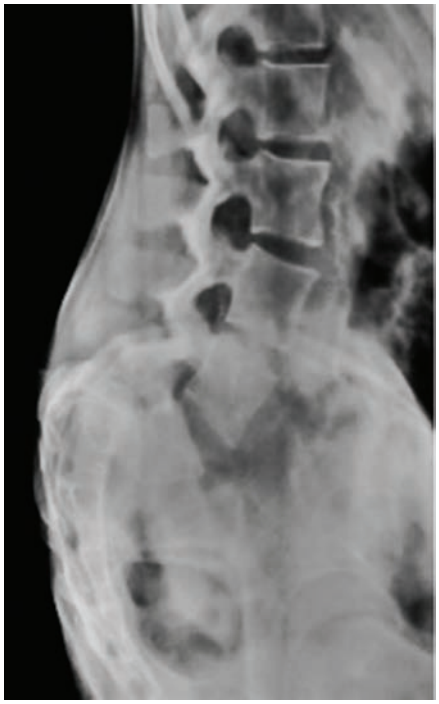


Figure 10.15 Lateral x-ray

have positive sagittal balance which is expected to be present in this high grade of listhesis. I will need MRI of her lumbar spine as well.

EXAMINER: This is her MRI scan (Figure 10.16 a and b).

CANDIDATE: MRI T1 and T2 sagittal image showing spondyloptosis at L5–S1.

EXAMINER: Unfortunately, I don't have her axial images. What do you expect in those images?

CANDIDATE: I would expect to see L5 nerve root compression more on the right as she does have right leg pain.

EXAMINER: How would you treat her?

CANDIDATE: I need to take a history first. I need to know her symptoms.

EXAMINER: You can ask her two questions.

CANDIDATE: Can you please tell me if you have any problems with your back?

PATIENT: I have back and nasty right leg pain for the past 2 years.

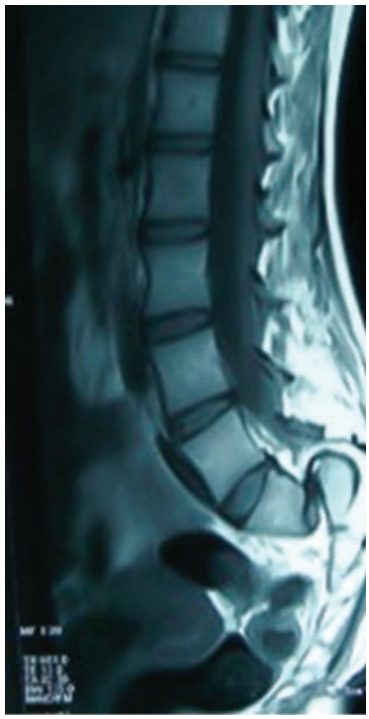
CANDIDATE: What all treatment have you had?

PATIENT: Painkillers, rest, physiotherapy. I am sick of it now. It's affecting my life. I used to be a gymnast before. No more.

EXAMINER: Would you offer her surgery?

CANDIDATE: Yes I would refer her to a spinal surgeon to consider surgery. She has tried non-operative measures so far. Surgery would be in the form of reduction of the listhesis and fusion with bone grafting. This is high grade and will probably need fusion up to L4.

(a)



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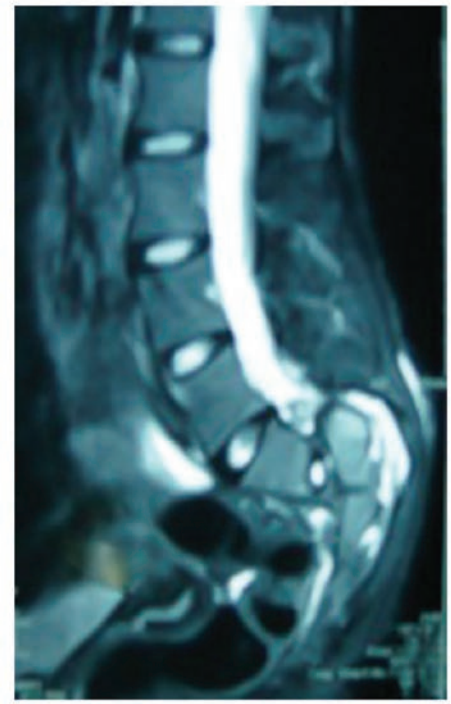

Figure 10.16 (a, b) MRI T1 and T2 sagittal images

EXAMINER: Can you think of any differences in isthmic spondylolisthesis like this one and a degenerative spondylolisthesis?

CANDIDATE: Degenerative spondylolisthesis usually affects older patients. Isthmic spondylolisthesis on the other hand affects children and adolescents especially those involved in repetitive hyperextension, like gymnasts, weightlifters.

L4–L5 is most common level in degenerative spondylolisthesis while L5–S1 is the most common level in pediatric spondylolisthesis.

And finally, the exiting nerve root in involved in isthmic listhesis while the traversing nerve root is usually involved in degenerative listhesis. So, L5–S1 lytic listhesis will involve L5 root while S1 root will be involved in degenerative listhesis L5–S1.

EXAMINER: Thank you.

Other points for discussion

1. Spondylolisthesis classification: Wiltse–Newman, Meyerding grading
2. Non-operative management
3. Indications for surgery
4. Reduction or in-situ fusion
5. Define pelvic incidence, sacral slope and pelvic tilt; what's their significance?
6. Degenerative spondylolisthesis and its management. Presentation similar to lumbar canal stenosis ± back pain. Decompression alone or with fusion?
7. SPORT trial outcome for spondylolisthesis

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Chapter 11

Hip clinical cases

Suresh Thomas and Paul A. Banaszkiewicz

Clinical examination of the hip

During the exit fellowship examination a candidate will have to demonstrate not only that he/she knows how to examine the hips of a patient but to also ensure that the examiner is able to see and appreciate each part of the examination. It will become immediately apparent to the examiner if the candidate has a routine for examining the hip. It is useful to have a set standard system that is second nature to you so that you appear competent and no steps are forgotten.

Examination of the hip in the month prior to the FRCS Orth exam should become a subconscious act to you. This means that even during the most stressful of situations in the real exam it will flow naturally, without one having to think about what comes up next or worry that one has missed some vital test out. If you are able to achieve this competency in your examination technique you will be more relaxed during the real exam and will be able to appreciate the significance of the clinical findings that you elicit.

In its most evolved form this involves anticipating expected clinical findings based on what you have already uncovered clinically and formulating ideas about the possible diagnosis as you go along.

By all means develop your own routine that works best for you but don't stray too far from the norm. Just as important, do not jump around and get the order of the hip examination out of sync. This is particularly annoying to examiners and suggests a disordered thought process and a lack of a systematic approach in your clinical practice.

Preliminaries

Preliminaries are very important in the exam setting even for the short cases where time is tight:

- Always introduce yourself to the patient
- Ask permission to examine the hip
- Ask if their hip is painful
- Explain to the patient that you are going to be moving their hip about and will do your best not to hurt them
- Make sure that you watch their face throughout the examination and avoid sudden movements
- Tell the examiner that you would like to start by undressing the patient to his/her underwear including removing socks. They will probably indicate that this is not necessary
- Although glaringly obvious at all times be careful to maintain the patient's modesty, because very occasionally candidates can become so focused on some minutiae finer detail of the examination process that they forget this. This is a pass/fail issue that will be discussed between examiners
- Don't forget to wash your hands between EVERY case

When examining the hip:

- Think about what you will find
- Listen to what the examiner says
- Look as though you know what you are doing and have examined a hip beforehand
- Appear confident to the examiners

Examine in turn:

- Inspection (Figure 11.1)
- Gait[a]
- Trendelenburg's test
- Palpation
- Thomas' test
- Movements (active and passive)
- Limb length inequality
- Neurovascular status

Trendelenburg's test

This is performed to assess the integrity of the abductor mechanism of the hip, which consists of a fulcrum, lever arm and power. This is a first order lever mechanism. The fulcrum is taken to be at the centre of the hip joint, lever arm represented by the neck of the femur and power represented by the controlling group of muscles. In practice it is easier if you first demonstrate the Trendelenburg test to the patient showing them what you want them to do. It avoids any misunderstanding (Figure 11.2).

[a] Keep the patient walking; it is difficult to take everything in immediately. Equally don't keep the patient walking forever if you can't work out the gait pattern – Move on with the exam.

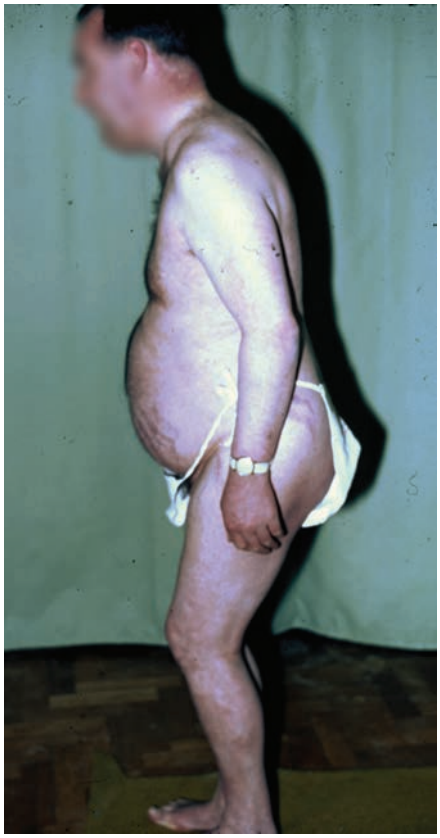

Figure 11.1 Inspection from the side. An increased lumbar lordosis suggests a compensatory mechanism to conceal a fixed flexion deformity of the hip

Figure 11.2 Trendelenburg's test. There are several modified methods of performing Trenedelenburg's test. Learn one method well and stick to it. Be very clear about what you are testing and be able to talk your way through the test as you perform it in front of the examiners. If all else fails remember the "sound side sags".

True positive

Power failure (weakness of abductors)

- Generalized muscular weakness or paralysis (polio, Duchene muscular dystrophy)
- Generalized neurological weakness (spinal cord lesions, myelomeningocele)
- Localized gluteal muscle paralysis or weakness (superior gluteal nerve injury, post-total hip arthroplasty (THA) exposure with failure of adequate repair, trochanteric osteotomy

Lever failure

- Intracapsular neck of femur fracture (NOF); extracapsular NOF, short neck in coxa vara, Perthes disease

Fulcrum (pivot) failure

Dislocation hip

- Developmental dysplasia of the hip (DDH)
- Femoral head destruction secondary to septic arthritis

False positives

Gluteal inhibition due to pain secondary to:

- OA
- Avascular necrosis (AVN)

If pain is not considered a true positive. Hip pain makes proper assessment of these cases difficult. It has been suggested that a 10% rate of false positives occur.

False negative

- Arthrodesed or ankylosed hip

The patient is able to maintain hip abduction with no abductor function. Sometimes the hip can be so arthritic that it will not move when standing on the affected leg and, therefore, the pelvis will stay level.

False-positive and false-negative responses may occur, but their interpretation can be clarified if the test is properly performed.

The test is invalid if:

- Poor balance
- Lack of co-ordination
- Unable to understand instructions

The presence of pain, poor balance and either lack of co-operation or understanding by the patient can lead to false-positive tests, because the test cannot be properly performed. The reason for some false-negative tests is that the

subject uses muscles above the pelvis to elevate the non-weight-bearing side of the pelvis, or shifts the torso well over the weight-bearing side; these can be called 'trick movements'. So long as you know the principles of the Trendelenburg test and appear confident in your approach this should be enough to score a basic pass. The examiners may ask 'if you know any other ways to perform the test' so be prepared to be at least able to discuss alternative methods. An increasingly popular slick method of performing the test is with the examiner seated in a chair but beware this may not always be possible in the short case examination hall.

There has been a recent update of the description of the test calling it a "single leg phase stance phase." This requires the patient to stand with feet shoulder width apart raising the unaffected leg to 45° knee and 45° hip flexion. The test is completed after 6 sec and is positive if the trunk falls more than 2 cm.

Thomas' test (Figure 11.3)

This is classic test material. The examiners will almost certainly ask you to demonstrate Thomas' test as part of an intermediate or short-case examination of a hip condition. The test is usually well described by most candidates but often poorly performed in the pressure/stress of the real exam. Candidates should practice this test repeatedly and be prepared to demonstrate it well. Do not hurt the patient. and generally best to avoid the test if THA on the opposite side for fear of dislocation.

Perform the test on the good side first. Ask the patient to hold their affected limb with both hands. Place your left hand under the patient's lumbar spine. With your other hand holding the good leg, control full extension of the limb. Full extension is normally achieved with the lumbar lordosis still obliterated.

If there is a fixed flexion deformity the patient will arch their lumbar spine and the lumbar lordosis will reappear (pressure is relieved in your left hand).

Figure 11.3 Thomas' test. The angle subtended between the back of the thigh and the bed will be the angle of fixed flexion deformity. Do not maximally flex up the opposite hip as this may flex the pelvis and lead to a false impression of a FFD. It is perhaps easier to perform the test kneeling down to the side of the couch rather than standing rigidly bolt upright

At the point where this occurs, bend the patient's knee so that their heel touches the couch and measure the angle between the couch and thigh = angle of flexion deformity.

Leg length inequality

Identify any obvious leg length difference:

- Is it real or apparent?
- If it is real is it in the femur or tibia?
- If in the femur is it above or below the greater trochanter?

Shortening of a limb could be compensated by tilting the pelvis (ipsilateral anterior superior iliac spine (ASIS) down), flexing the opposite limb at hip and/or knee, equinus position of the ankle.

True shortening

The affected leg is physically short compared to the opposite side. This could be above or below the trochanter. Measurement is taken from ASIS to the medial malleolus. If there is a deformity in one leg, the other leg must be placed in an identical (mirror equivalent) position and the pelvis square.

Apparent shortening

Apparent shortening is measured from the xiphisternum to medial malleolus (pelvis need not be square). It is taken as the sum of true shortening plus shortening from any fixed deformity.

This measurement helps in assessing the extent of natural compensation developed for concealing the actual disparity at the hip joint especially by tilting the pelvis sidewards (fixed abduction and fixed adduction deformity). On many occasions this natural compensation improves cosmetic appearance.

- If the true shortening is equal to apparent shortening it indicates no compensation
- If the true shortening is more than apparent shortening it indicates that part of the shortening has been compensated
- If the true shortening is less than the apparent shortening it suggests a fixed adduction deformity besides shortening without compensation

In recent exam sittings there has been less concern with measuring for apparent shortening and a more concentrated focus on true shortening. A safe middle ground approach would be to measure for true shortening and reserve measuring for 'apparent' discrepancy only when there is an incorrectable tilt of the pelvis.

Examination of leg shortening standing

- Blocks can be used to measure for any leg length difference in the standing weight-bearing position
- The pelvis is tilted to a lower position on the short side
- Correct the pelvic tilt and when the ASIS is at the same level place blocks under the foot. This gives the length disparity. Many clinicians regard this as a better method of assessment of length discrepancy than measuring

supine as the patient is actively involved in this assessment. Be careful if a fixed obliquity is caused by lumbosacral disease, as this cannot be corrected for by using this method
- A more accurate method that may be brought up in a discussion is either a scanogram or spiral CT scout film
- If a patient has a fixed deformity of a joint the Block test is not accurate and, therefore, the limb needs to be measured in the position of deformity using a tape measure

Examination of leg shortening supine

- Apparent shortening is roughly estimated measuring the distance from xiphisternum to the medial malleolus. The lower limbs are kept parallel to one another and in line with the trunk. The pelvis need not be square.
- Next go on to measure for true shortening. Ensure the pelvis is square
- Legs should be kept in identical position if possible
- It may not be possible to do this if there is a pelvic tilt with adduction contraction
- A deformity is usually unmasks on squaring the pelvis
- Measurement is taken from ASIS to medial malleolus with the limb in the deformed position
- When the normal limb is measured, it is necessary to keep it in the same position as the affected limb
- Any pelvic tilt due to postural scoliosis should be adjusted by the position of the patient

In simple terms the distance between the ASIS and the medial malleolus is 'less' in adduction and 'more' in abduction and not the 'same'. Therefore, both the limbs should be either in 'adduction' or 'abduction' to get the true length of the lower legs. This can be done by bringing both the ASIS at the same level by 'squaring' the pelvis before recording the real lengths.

Abduction/adduction contracture

Deformity is unmasked by squaring the pelvis.

A patient with an adduction deformity compensates and may appear straight by tilting the pelvis on the affected side up. Hence, the ASIS is raised and the leg appears short.

A patient with an abduction deformity compensates and may appear straight by tilting the pelvis on the affected side down. Hence, the ASIS is lowered and the leg appears long.

If the pelvis is square and ASIS are at the same level, there is no deformity.

Comment to the examiners on the presence of an adduction contracture:

> 'I am unable to place the legs perpendicular to the pelvis because of an adduction contracture and, therefore, I must place the other leg in the same position.'

This is difficult and cannot be done simultaneously. The most practical way is to cross the legs sequentially. Cross one leg and measure and then cross the other leg and measure.

Again comment to the examiners on the presence of an abduction contracture:

> 'I am unable to place the legs perpendicular to the pelvis because of the abduction contraction and must place the normal leg in the same position.'

Abduct the unaffected hip to the same degree. Measure leg lengths.

Flexion contraction knee

Comment on this to the examiners:

> 'I am unable to place the legs straight because of the fixed flexion of the knee.'

You must place the other leg in the same position. One would flex the unaffected knee over a bolster or pillow to the same degree and then measure leg lengths.

Valgus knee

Comment on this deformity to the examiners:

> 'I am unable to place the leg straight because of the valgus knee. I am unable to place the opposite leg in the same position.'

Note the difficulty and, therefore, you must measure component parts of the leg. This approximates to a true leg length. You measure from the ASIS to the tibial tuberosity and then from the tibial tuberosity to the medial malleolus.

Examination corner

For a short case the examiners may just specifically ask a candidate to examine for leg length inequality. This is not always performed well by candidates whilst the textbooks can be often misleading and contradictory.

In the ideal world the legs should be parallel to each other and the sides of the examination couch and be perpendicular to an imaginary line joining the ASIS.

Apparent leg length discrepancy

Measure for apparent leg length discrepancy first. The patient should be lying supine in a comfortable position with the affected leg in the line of the trunk. The lower limbs should be in a parallel position. To achieve this the unaffected leg is moved to make the limbs parallel. No attempt is made to correct any pelvic tilt or abnormal limb position. The measurement is taken from any central fixed point on the trunk (central point of the suprasternal notch, xiphisternum) to the medial malleolus. Textbooks also mention the umbilicus but some old school examiners may comment that it is not a fixed structure and may not be midline if diseased or had previous umbilical surgery.

True leg length discrepancy

With measurement of true leg length the pelvis will then need to be squared up to reveal any concealed fixed abduction or adduction deformity. The legs are put into equivalent/identical positions. The affected leg is moved to square up the pelvis (level the pelvis) by exaggerating the noted deformity. The normal limb is then moved to make it in an identical position to the affected leg. Check the level of the malleoli.

Measure from the ASIS to the medial malleolus. The ASIS can be difficult to reliably identify in obese patients or if distorted from iliac chest bone graft harvesting. Make sure you accurately define the ASIS by hooking your fingers up from below.

Difficulty may also be encountered with fixed deformities of either the hips or knees. If necessary sequentially measure from ASIS, greater trochanter, medial joint line of the knee and tip of the medial malleolus.

Shortening cases for the FRCS (Tr & Orth) exam

Level 1 difficulty

This may involve a femoral fracture with mal-union and shortening. The mal-union usually will involve a rotatory element.

There could be an associated arthrofibrosis of the knee with limited knee movement and knee pain.

History

The patient may have been involved in a road traffic accident (RTA) and so a full history is required concerning the mechanism of injury.

It is important to know all the various surgical treatments undertaken.

What are the current on going problems?

Examination

There will generally be various surgical and non-surgical scars present.

Get slick at describing the scar patterns present.

Left lower limb external rotatory position at rest with equinous position of the ankle.

Discussion

Know your definitions of true and apparent leg length discrepancy.

What can be done about post-trauma painful arthrofibrosis of the knee in a young male patient?

There has presumably been a failure of conservative treatment and, therefore, arthroscopic lysis of adhesions should be considered. Distension of the capsule, excision of adhesive bands and release of scar tissue is performed in a systematic fashion. If this is not successful or the arthrofibrosis severe then an open release may be indicated.

Limitations to extension are generally secondary to pathology in the intercondylar notch region. Limitations of knee flexion are generally secondary to scar tissue within the medial and/or lateral gutters or within the suprapatellar pouch region.

In a patient who has significant pain, consider infection and/or complex regional pain syndrome (CRPS) as a contributing cause of knee stiffness.

Discussion of the Ilizarov frame correction of the deformity.

Level 2

The femur and tibia of the involved limb are short but no attempt has been made to lengthen either of them.

The clubfoot or polio case of a hypoplastic femur and tibia causing a leg length discrepancy. There is shortening in the tibia and femur so Galleazzi's test is more complicated to interpret. The knee of the longer leg is projecting more towards you from the end of the bed and higher up from the side.

Level 3

The femur and tibia of the involved limb are short but there has been an attempted lengthening of one or both bones.

Candidates may get confused as they are instinctively expecting both bones to be shortened because the leg is hypoplastic with poor musculature.

Examples would include:

1. The clubfoot case with a short femur but longer tibia than the opposite normal side from an Ilizarov lengthening procedure.
2. The polio case where there has been an attempt at lengthening the tibia using a monolateral external fixator to correct the limb length discrepancy (LLD) (Figure 11.4). This is puzzling as you would normally expect the tibia to be both hypoplastic and short. The give away clues are healed external fixator scars in the lower leg. Galleazzi's test is difficult to interpret if a candidate does not appreciate what's been done (Figures 11.4–11.6).

Level 4

Two long bones of the same limb are shortened with possible lengthening attempted of one or both bones. The normal opposite limb has been shortened to reduce the LLD either the femur or tibia or both.

The hypoplastic left leg of clubfoot. Multiple scars of clubfoot surgery on the actual foot itself. A long surgical scar posteriorly over the tendo Achilles from previous lengthening of the soft tissues. The left foot is small and hypoplastic.

The normal right femur is shorter than the involved left femur as epiphysiodesis has been performed on the distal right femoral epiphysis. The normal right tibia is longer than the opposite tibia as no epiphyisiodesis of the proximal right tibia has been performed as well as no lengthening of the left tibia.

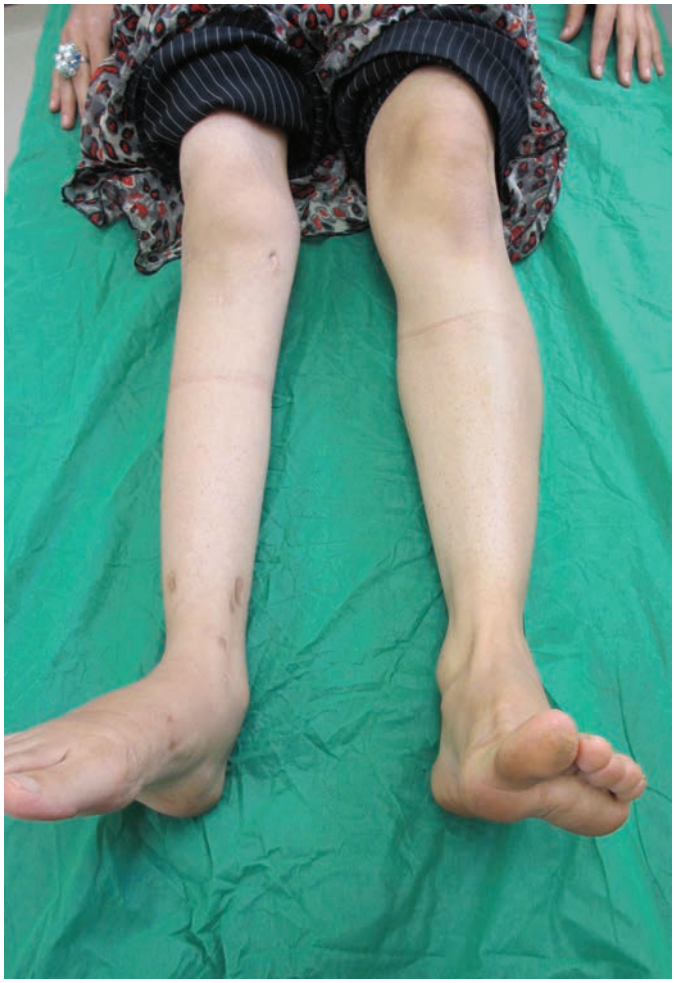

Figure 11.4 Clinical picture of polio affecting the right leg with previous external fixator lengthening of the right tibia. Note residue valgus deformity, right foot.

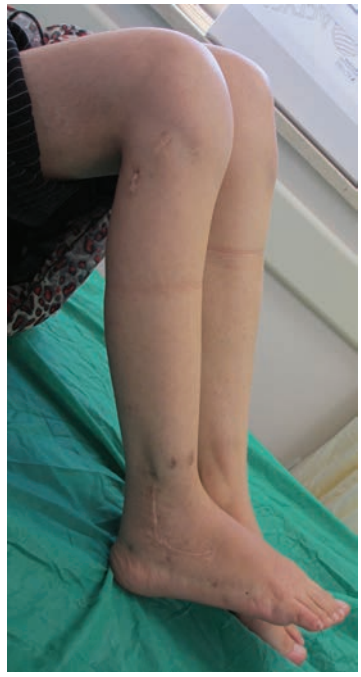

Figure 11.5 Galleazzi's test performed on patient in Figure 11.4 with polio disease right leg. Shortened right femur with lengthened right tibia.

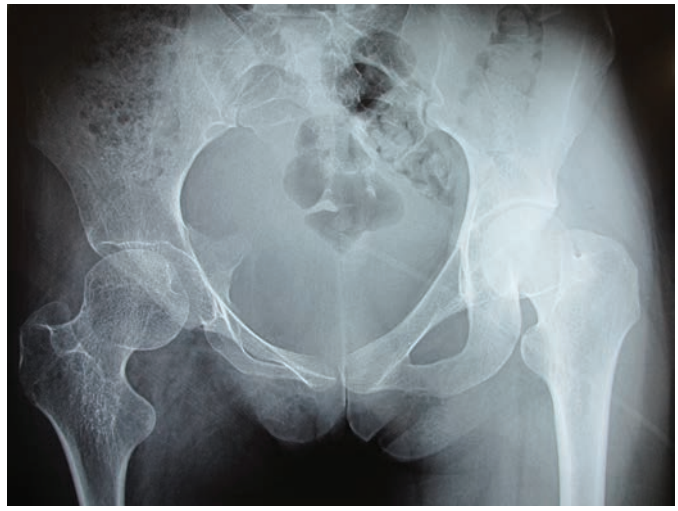

Figure 11.6 AP pelvis radiograph of patient in Figure 11.4 with polio disease right leg. Underdeveloped femur, pubis and ischium.

Galeazzi's test is confusing as the femur is shorter on the normal side and the tibia longer than the involved limb

Look for the scars from the epiphysiodesis surgery, these are not always obvious and you can end up concentrating on the involved hypoplastic leg missing the epiphysiodesis scar on the normal leg

A plantigrade foot with mild heel varus. The arch of the foot was well maintained

The other common scenario is the polio leg, which is equivalent/similar to clubfoot in all of the various combinations

Level 5

Both lower limbs are hypoplastic and this involves both the femur and tibia but there is usually a more affected leg and all combinations of lengthening of the short leg and shortening of the longer leg may have been attempted

COMMENT: Some very good candidates seem to intuitively be able to work out all these possible clinical combinations and do well. Borderline candidates may not be aware of all these potential scenarios and may struggle to work out what is going on from first principles. It is sometimes difficult to think fast on your feet working out leg length combinations from scratch in the stress of the exam. Better to have worked through these assorted scenarios beforehand.

Ankylosing spondylitis

Introduction

AS is a seronegative inflammatory disease of unknown aetiology primarily affecting younger men. Peripheral joint involvement is less common than spinal disease. The hip joint is involved in 30–50% patients and is usually bilateral

(50–90%). The typical age of onset is between 15 and 25 years. The younger the age at onset the more severe the disease is likely to be and the more likely the need for THA.

History

As for any inflammatory joint disease. The onset often is insidious with low back pain and stiffness, chest wall pain, enthesopathy involving the Achilles' tendon and plantar tendons. Occasionally may present with no pain but increasing stiffness in the hips and spine. Ask about ocular pain and problems with chest expansion.

Clinical features

The characteristic spinal deformity is a combination of a thoracic hyperkyphosis and a flattening of the lumbar lordosis, causing the patient's head and neck to thrust forward. Over time, the kyphotic deformity causes a downward and forward shift of the patient's trunk,

A functionally disabling advanced stoop develops with limitation of forward vision (question mark posture). The chin brow angle, occiput to wall distance and gaze angle are used to evaluate functional deformity involving the cervical spine. There is severe loss of motion at the hip joint, a fixed flexion deformity or ankylosis.

Chest expansion should be at least 5 cm, but is often limited in ankylosing spondylitis due to costochondral arthrosis. Not a reliable sign in the elderly or COPD.

Wall test: Heels, buttock and scapulae all should be able to touch the wall, but if decreased extension unable to do this

Schober's test: Mark two points, one 10 cm above and one 5 cm below the level of the posterior superior iliac spines and forward flex the spine, the distance should lengthen 5 cm

Radiographs

Radiographs show ossification of the ligamentous origins and insertions about the trochanters, iliac crest and ischial tuberosities. Later on radiographs become similar to end-stage primary OA.

Management options

If there is any uncertainty whether pain is arising from the hip joint or spine then a local anaesthetic injection into the hip joint may be useful. Hip involvement ranges from flexion contractures to complete ankylosis, often in a disabling flexed position. Total hip arthroplasty (THA) may be considered before spinal osteotomy because improvement in hip's range of movement and pain relief may obviate the need for spinal osteotomy in patients with severe hip flexion deformity. Others take the opposite view and recent evidence has shown higher dislocation rates when spinal osteotomy is done after total hip arthroplasty (THA)[1]. In addition, hyperextension of immobile spine during THA could lead to intraoperative thoracic vertebral body extension fractures with resultant acute traumatic paraplegia.

Indications for THA

Indications include severe disabling debilitating pain and correction of severe hip flexion deformities.

The aims of THA are pain relief, eradication of flexion contractures, increased range of hip joint, movement, improved mobility and correction of posture[2].

Consider bilateral surgery for severe bilateral fixed flexion deformities. The patient will not be able to stand up straight until both hips have been operated upon and if more than a few months is left between the two sides the deformity will reoccur. Another relative indication for bilateral surgery is if there are risks and difficulties associated with the anaesthetic, e.g. need for fibre optic awake intubation.

Technical difficulties

Many surgeons consider THA in AS patients to be a particularly demanding procedure. In the exam it is reasonable to mention that you would seek the advice of a senior hip surgeon and ensure adequate preoperative anaesthetic assessment before surgery.

Spinal and epidural anaesthetic may be difficult due to ossified ligaments (bamboo spine). Restricted chest expansion, ITU bed back-up and echocardiography for any valvular heart disease.

Difficulties relating to positioning of patients on the operating table, the correction of longstanding contractures, accurate placements of the acetabular component in the presence of pelvic obliquity or tilting and delicate skin and soft tissues have all served as deterrents.

AS patients with a fixed kyphotic spine tend to hyperextend their hips once they stand upright, in an attempt to look forward. If the cup is inserted according to the anatomy of the acetabulum, it becomes abnormal when the patient resumes an upright position.

The pelvic hyperextension brings the cup to a more open position with an exaggerated anteversion and may lead to anterior instability and dislocation. Exaggerated anteversion may lead to intraoperative difficulties including impingement of the prosthetic neck or the greater trochanter posteriorly or difficulties in reducing the hip.

There is a higher incidence of ectopic bone formation after THA leading to a reduction in the postoperative range of movement[b]. This is more common when trochanteric osteotomy is performed. There are concerns regarding the young age of patients, they may place greater demands on the prosthesis that results in increased rates of wear and loosening. In addition, AS tends to spare upper limbs resulting in higher demands on hip prostheses because overall function is better.

[b] Controversial. Recent reviews of the literature suggest that heterotopic ossification (HO) rates may not be that dramatically higher compared to age- and sex-matched counterparts.

Examination corner

Intermediate case 1: Ankylosing spondylitis

Patient's age, activity levels and expectations from THA surgery

Document spinal involvement, pelvic obliquity and LLD, status of the contralateral hip, bilateral knees and integrity of sciatic nerve

Loss of normal lumber lordosis

Stiff spine with gross restriction of all spinal movement

Abnormal Schober's sign: Lack of at least 5 cm increase in distance from a midline point 5 cm below and 10 cm above the posterior superior iliac spine

Some flexion at the knee to maintain erect posture

Thomas' test to demonstrate fixed flexion deformity both hips. Candidates will be quizzed on the principles of Thomas' test and various steps such as keeping your hand under the spine. Ensure you know how to assess patients which ispilateral knee and hip flexion deformity. A patient can be moved to the edge of the bed and the knee with a flexion deformity is dropped down to unmask any hip deformity. Keep the knee outside the edge of the couch

Methotrexate is generally not withheld in the perioperative period but the risk of infection with anti-TNF is well recognised and, therefore, usually stopped

Preoperative templating is important to estimate component size and positioning

Operating surgeon should be present to position the patient to familiarize themselves with deformities of the pelvis and spine and reduce risk of component mal-positioning

THA survival rates in AS patients are similar to that in other young patients with THA. There has been a move towards using uncemented components in young active patients with AS

Hip resurfacing in generally contraindicated in the presence of an inflammatory arthropathy. Li et al.[3] in a small study from China recommended their use in AS as ROM was significantly better than THA, with the same pain relief and a low dislocation risk. Hip resurfacing has fallen out of favour due to concerns with adverse reactions to metal debris (ARMD) pseudotumours etc. If a candidate decides to mention resurfacing arthroplasty in the exam they must follow through with referral on to a specialised hip resurfacing surgeon performing reasonable numbers each year. This may lead on to "Getting in right first time" (GIRFT) Tim Briggs recommendations and increased complications with low volume high risk procedures.

Intermediate case 2: Complicated case of 72-year-old male with AS

'Presented with bilateral hip pain. Difficulty walking with a walking distance of 200 yards. Had right hip fused several years previously for AS and established AS left hip. The history of the pain suggested referred pain from the spine into both hips especially as it radiated down past his knee into both feet. There was, however, groin pain bilaterally.'

'The discussion was tricky and seemed to focus on taking down the fused right hip to relieve the patient's right hip pain. I was surprised with this approach, as I had suggested a diagnostic steroid injection into the left hip, investigation for possible referred pain from the spine with an MRI scan, rheumatology review. I mentioned

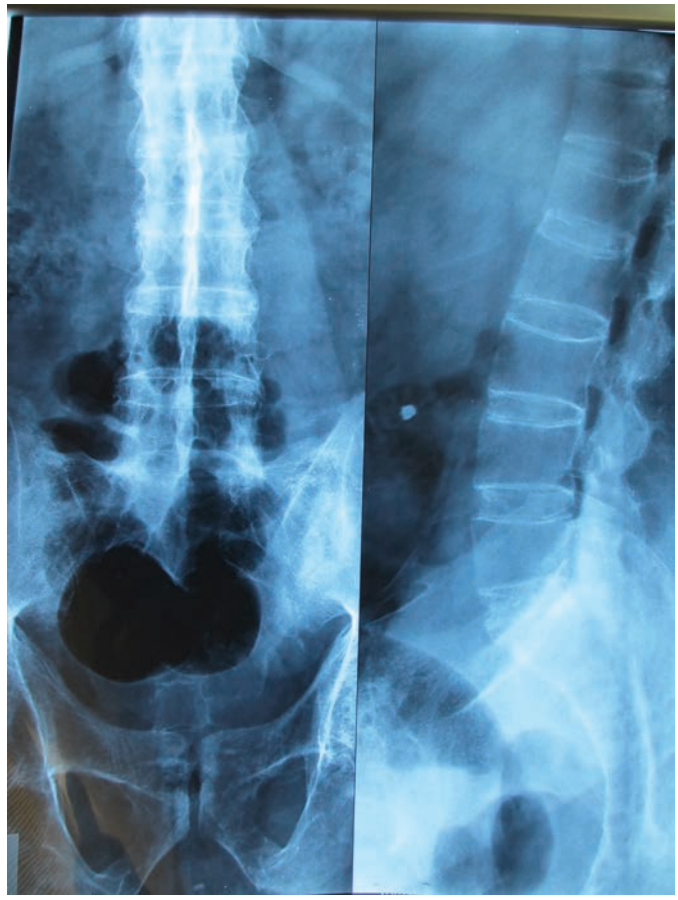

Figure 11.7 AP and lateral radiograph bamboo AS spine

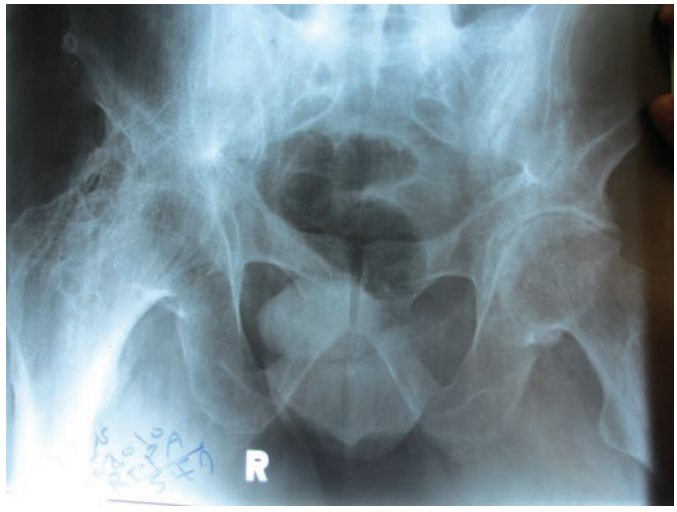

Figure 11.8 Fused right hip in AS

considering THA on the left side if the patient had experienced a good response to his steroid injection.' (Figures 11.7 and 11.8)

COMMENT: 'I don't think the candidate was necessarily wrong, but perhaps the emphasis should have been the fusion causing LBP and secondary left hip pain rather than the AS per se. Common indications for the conversion of an arthrodesed hip to a THA include pain arising from the lower back, ipsilateral knee or contralateral hip, a painful non-union or mal-positioned arthrodesis and planned ipsilateral knee replacement. It is important that the origin of pain is accurately defined.'

Developmental dysplasia of the hip (DDH)

History

Indications for surgery include persistent pain affecting routine daily activities. Women are more affected than men, family history and birth history are important. DDH may affect hip movements, problems with sexual intercourse and personal hygiene. Hip symptoms must be defined in detail. Buttock pain, back pain and an abnormal gait are usually the primary symptoms unlike typical groin pain of OA.

Unilateral hip disease may cause secondary problems with leg length discrepancy, ipsilateral knee pain, pelvic obliquity, scoliosis, limp, muscle weakness and LBP.

Examinations findings

'On examination the left leg appears shortened. The muscle bulk of the left thigh is markedly reduced. There is a compensatory pelvic obliquity/scoliosis/lordosis because of the leg length discrepancy/fixed flexion deformity. The attitude of the left leg appears externally rotated compared to the opposite side. The patella is not facing forward suggesting that the rotation is occurring in the femur. The pelvic obliquity can only be partially corrected with wooden blocks. Examination of gait demonstrates an obvious limp with a short stride, toe walking and increased lumbar lordosis. When sitting on the bed the scoliosis of the spine only partially corrects, which means that there is an element of both a flexible and a fixed deformity of the spine. The peripheral pulses are palpable with good capillary refill of the toes.'

If the scoliosis is not fully correctable, full correction of a leg length disparity can lead to persistent problems. When examining the patient supine on the couch attempt to square the pelvis if possible. Comment to the examiners if you can or cannot do this. If the patient has a fixed pelvic obliquity then apparent leg lengths (measured from the xiphisternum to the medial malleolus) should be calculated. Galeazzi's test and Bryant's triangle should be performed if there is any suggestion of true shortening (previous femoral osteotomy). It is important to differentiate between true and apparent shortening and to be able to explain this to the examiners if asked. Check for a difference in rotation of the hip when flexed or extended due to discrepancy in the shape of the femoral head in the acetabulum. Abductor muscle weaknesses may be present secondary to proximal femoral migration or reduced femoral offset. Functional abductor strength is evaluated with a Trendelenburg's test and side-lying abduction strength testing.

Examination of gait reveals a waddling gait. There is increased lordosis of the spine and the body is swaying from side to side on a wide base. The patient lurches on both sides whilst walking and is Trendelenburg positive.

Technical considerations

The aim of THA in DDH is to restore the centre of hip rotation and proximal femoral anatomy to allow optimal abductor function. Implant options must cover the need for small acetabular components and small/short femoral stem

Adequate preoperative planning such as clinical assessment, CT scan, preoperative templating and arrangements for bone grafts are required. The patient should be forewarned that the operation might be abandoned if either abductor musculature is poor or the bone of the pelvis is inadequate.

Decide on anatomical (true acetabulum) or non-anatomical (high hip centre) acetabular cup positioning. The proximal femur is typically smaller, narrower, straighter, weaker and more anteverted than in normal hips. Diffuse osteoporosis and thin cortices increase the risk of intraoperative femoral fracture when reducing the THA, especially if reduction is tight because of leg lengthening. The level of the true acetabulum needs to be defined for placement of the cup (the surgical landmark is the obturator foramen). A drill hole may be used to perforate the medial part of the acetabulum and a depth gauge may be used to decide how far to ream the acetabulum. Be aware of the possibility of sciatic nerve injury from excessive lengthening. Avoid >4 cm lengthening.

Discussion

Surgery is only indicated if there is disabling pain and all conservative management options have been exhausted. Pre-arthritic patients may be candidates for hip arthroscopy or periacetabular osteotomy.

Investigations include full-length standing bilateral lower extremity radiographs and CT scannogram if large LLD exists. CT scan of the pelvis is valuable in assessing acetabular position, version, bone stock, femoral anteversion and femoral canal diameter. MRI is seldom useful in the presence of established OA changes about a dysplastic hip. Subluxation or significant adduction deformity can cause functional shortening of the leg and secondary long leg arthritis in the opposite knee (owing to walking on a flexed knee to compensate for a LLD). A valgus OA of the ipsilateral knee caused by severe adduction deformity of the ipsilateral hip may develop. Knee arthroplasty in these patients will accentuate their scissoring gait and lead to early failure of the TKA if the adduction deformity at the hip is not corrected first

Hip reconstruction becomes more technically challenging as the grade and extent of dysplasia increases.

Subtrochanteric femoral shortening osteotomy (Figures 11.9 and 11.10)

Indications

Crowe type IV and selected Crowe type III hips with DDH and secondary arthritis that would lead to unacceptable lengthening of the limb if managed with THA without femoral shortening and concern with sciatic nerve injury.

Advantages

Allows simultaneous shortening and correction of rotational abnormalities, protects the sciatic nerve and preserves the proximal femoral metaphysis. The preservation of the proximal femoral metaphysis facilitates the placement of an uncemented femoral component by providing increased torsional stability for the implant; this helps to maintain more normal proximal femoral anatomy and avoids the need for a greater trochanteric osteotomy, which may not unite. It avoids the need for sequential proximal resection, which results in a small straight femoral tube with a small metaphyseal flare, which is usually unsuitable for an uncemented femoral implant.

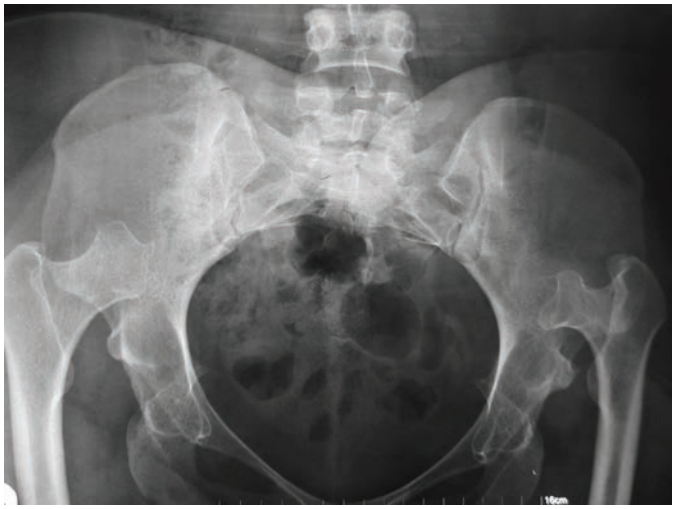

Figure 11.9 Preoperative AP pelvis radiograph bilateral Crowe IV DDH hip

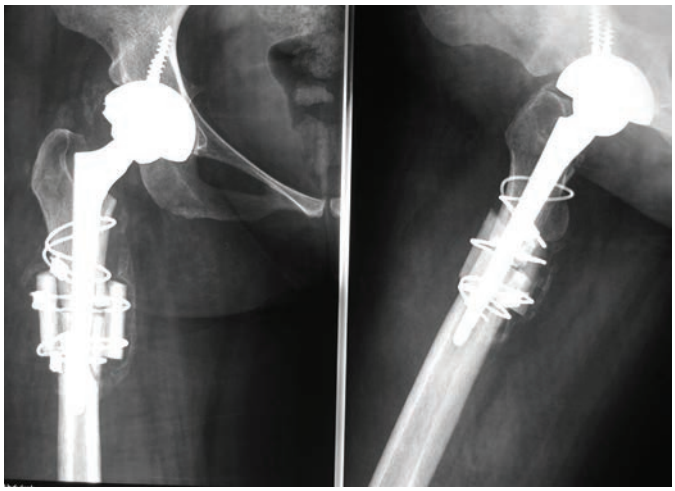

Figure 11.10 Postoperative right THA with subtrochanteric shortening. The opposite left side will require a similar procedure

Examination corner

Intermediate case 1: Developmental dysplasia of the hip (DDH)

Discussion

EXAMINER: What is your preferred placement of the acetabular cup for a hip congenital dislocation of the hip (Hartofilakidis grade 3 hip)?

CANDIDATE: At the anatomical position.

EXAMINER: What's wrong with placing the cup in a high hip centre?

CANDIDATE: At the level of the false acetabulum the lever arm for body weight is much longer than normal, which results in excessive load on the hip joint and at the non-anatomical level the shearing forces on the acetabulum may lead to early loosening.

A high hip centre compounds abductor insufficiency, limping and leg length discrepancy. Bone stock is better at the level of the true acetabulum.

EXAMINER: So why do surgeons sometimes place the cup high?

CANDIDATE: It may be difficult to fully cover the acetabular cup at the anatomical level. A femoral head allograft can be used to augment the superolateral aspect of the acetabular rim or a cotyloplasty performed medially but these are technically difficult. Cotyloplasty involves creating a comminuted fracture of the entire medial wall, autogenous bone graft and a cemented acetabular cup.

EXAMINER: What about cup size?

CANDIDATE: Poor bone stock at the anatomical position usually requires the use of a small acetabular cup. It is sometimes not possible to use a ceramic bearing surface and there has been concerns with inadequate thickness of polyethylene (PE), especially in young patients.

EXAMINER: Would you use a cemented or uncemented cup?

CANDIDATE: In recent years cemented acetabular cups have fallen out of favour because of reported high revision rates. Porous tantalum implants provide excellent initial stability with osteoconductive and osteoinductive properties and this would be my preferred choice of implant.

EXAMINER: These are expensive implants. Would you not want to choose a more cost-effective implant?

CANDIDATE: I would be slightly concerned about the possibility of poorer or unpredictable results for younger patients with compromised bone stock in the acetabulum if using standard shell liners

Intermediate case 2: Young female with unilateral left DDH

Main symptoms of left hip pain, low back pain and shortening

On examination

The pelvis is not level, there is pelvic obliquity
With the pelvis straight with blocks there is a leg length discrepancy of 4 cm shortening of the left leg
There is a compensatory scoliosis with convexity towards the left; it is a combination of both structural and postural elements
The patient has a short-leg gait
Trendelenburg's test is positive
Sit the patient to check if the scoliosis deformity corrects
Ask the patient to lie supine
Carry out the Thomas' test to test for any fixed flexion deformity of the hips
Check hip range of movement
Measure apparent and real leg lengths
Galeazzi's sign
Bryant's triangle test for supratrochanteric shortening

Intermediate case 3: Middle-aged lady with bilateral DDH

- Take a detailed history of the DDH
- Demonstrate various signs: Thomas' test, Trendelenburg's test, hip range of movement (ROM)
- Describe the technical difficulties in performing THA in DDH, explaining the role of the CT scan in planning the operation
- Describe the effect of anteversion on THA (component mal-alignment, dislocation proximal femoral fracture, internal rotational contracture hip)
- Describe correction of leg length inequality in DDH (decide preoperatively, how much LLD to correct what method to use). Do not over correct as sciatic nerve dysfunction may occur. Explain the use of bone grafts when performing THA
- Describe the long-term results of THA in patients with prior DDH. When properly performed, THA for DDH can result in good long-term results. McKenzie et al. reported 85% survival at 15 years[4]
- Revision of patients who have undergone THA for DDH is extremely difficult, particularly when the acetabulum has been placed high and revision has been delayed. There is often no anterior wall, little posterior wall and only the remnants of a medial plate. Femoral revision can be difficult, as the prosthesis may have been inserted with an uncorrected deformity. If a trochanteric osteotomy has gone on to develop a non-union, trochanteric drift is difficult to correct. Soft-tissue balance in these patients is extremely difficult and, therefore, dislocation rates are high

Mild hip dysplasia

Background

There is a changing pattern of hip dysplasia presentation. We are seeing less and less Crowe 3/4 hips and more younger patients presenting with mild hip dysplasia and hip pain. These latter cases are more likely to end in the clinicals.

Not all acetabular dysplasia is caused by DDH. A concave acetabulum cannot develop without a concentric force being exerted by a reduced femoral head. Other causes of dysplasia include polio, cerebral palsy, hyperlaxity and Perthes' disease.

Mechanical disorders of the hip can be divided into two major categories: Structural instability (dysplasia) and femoroacetabular impingement, or combinations of the two.

Osteoarthritis commonly occurs secondary to repetitive and/or chronic shear stress at the acetabular rim. Acetabular dysplasia and femoroacetabular impingement are the two most common causes of excessive shear stress and acetabular rim syndrome.

In DDH, inadequate osseous coverage of the femoral head results in mechanical overload of the anterolateral acetabular rim and labrum. As a result, patients with DDH commonly have anterolateral labral tears, anterolateral acetabular chondromalacia, acetabular rim fractures, and synovial cysts. This acetabular rim overload syndrome progresses to arthrosis with time unless the hip joint pathomechanics are corrected.

Femoroacetabular impingement is characterized by decreased clearance and abnormal contact between the femoral head–neck junction and the acetabular rim. These disorders are due to proximal femoral and/or acetabular rim deformity and are now recognised as common causes of pre-arthritic hip pain and secondary OA. Abnormal femoroacetabular abutment, particularly in positions of hip flexion and internal rotation, predispose affected patients to labral tears, articular cartilage damage and premature OA. Impingement abnormalities can be divided into two major categories: Cam-type and pincer-type impingement disorders and hip instability symptoms secondary to dysplasia.

History

Sharp activity-related groin pain increasing affecting lifestyle activities. The onset of pain may be insidious. Alternatively, it may start acutely after a period of increased activity, such as sports training or following an activity holiday (ski-ing, climbing). Pregnancy and weight gain may also cause a dysplastic hip to deteriorate.

Initially the pain may only affect running and sporting activities, but as symptoms progress the pain intrudes on everyday activities.

Symptoms may be worsened by rising from a seated position, getting in or out of a car, going downstairs or sudden rotational movements. These symptoms arise from the anterior labral tear and adjacent articular cartilage damage and, therefore, are similar to those of femoroacetabular impingement (FAI).

Additional features may include instability, weakness and the feeling of a 'dead leg'. Trochanteric symptoms may also be present because of abductor dysfunction and patients may also

describe clicking at the front of the hip, commonly originating from the psoas tendon.

Clinical examination

Sometimes there can be very little to find on clinical examination.

Candidates would be expected to demonstrate and explain the impingement tests (anterior and posterior impingement tests and FABER test).

There may be a mild antalgic gait or a delayed Trendelenburg's positive sign. There may also be some mild shortening, but generally this should not be excessive or else the case is most likely adult DDH (Crowe 2 or 3) with developing arthritis.

Hip range of movement of the hip is often normal, although internal rotation in flexion may be painful. A painful, reduced ROM suggests developing OA.

Instability may be demonstrated by apprehension on external rotation of the extended abducted hip. There may be additional signs of previous hip surgery (scars), hypermobility or an underlying neurological disorder.

Discussion

Imaging

- Standing AP pelvic radiograph. Lateral centre edge (LCE) angle >25° normal, below 20° dysplastic
- CT scan
- MRI arthrogram occasionally indicated for the diagnosis of labral tears and in assessing the condition of the articular cartilage

Management

- Hip arthroscopy. Occasionally indicated to treat labral pathology; however, results can be unpredictable and may lead to worsening of symptoms, as the stabilising effect of the labrum may be lost. Concern also exists regarding accelerating arthritis
- Femoral osteotomy. Seldom used in isolation where periacetabular osteotomy (PAO) is available as it is less effective, does not address the main deformity and complicates subsequent THA
- PAO. The surgical goal is correction of the acetabular insufficiency by repositioning the weight-bearing surface laterally and anteriorly to improve femoral head coverage. The hip joint center is medialised
- THA. For advanced end-stage painful dysplastic hip

Perthes' disease with moderate/severe secondary OA

History

It is important when taking the history to go into as much detail as possible about how the Perthes' disease was treated as a child. However, be quick and focused about it, be careful with your time and know the questions to ask beforehand. Did they have surgery; were they admitted to hospital at any time; did they use an ambulation abduction brace, etc. Alternatively the examiner may want you to jump over this part of the history for lack of available time and also so that you can go on to examine the hip, but it is important to cover these questions unless they indicate otherwise.

Examination

Standing

Carefully look for mild/moderate thigh or gluteal muscle wastage and comment on this finding to the examiners. Do not miss obvious muscle wasting. The thigh musculature may be normal if the individual is bulky with minimal disease. It is unlikely that a significantly LLD will be present; at worst possibly some mild shortening of the affected leg by 1–2 cm. If by chance the shortening is >2 cm look for a flexed attitude of the uninvolved limb or equinus posture of the involved foot. Possible mild external rotation deformity of the affected leg. Mild or moderate antalgic gait with a short leg component – If shortening present and clinically significant. A Trendelenburg's positive or delayed Trendelenburg's positive test.

Supine

Comment on any additional features not apparent on initial inspection of the leg when standing. Mention any feature even if already mentioned, particularly if it is more apparent supine. Comment on the attitude of the leg, especially if it lies in external rotation. Do Thomas' test.

Look at the relative position of heels/medial malleolus as a rough guide to shortening. (Make sure the pelvis is square.) Measure leg lengths and if shortening is present continue on and perform Galleazzi's test and digital Bryant's test. Flex the hips to 45° and the knees up to 90° and place the heels together. When one knee **projects farther forwards** than the other, either that femur is longer or more usually the contra-lateral femur is shorter. When one knee is **higher than the other**, either the tibia of that side is longer or the contra-lateral tibia is shorter.

Although not always performed in a focused hip examination ask the examiners if they would like you to palpate the hip for any areas of tenderness, any lumps or swellings. One difficulty is that the hip joint is too deep to assess for the presence of an effusion or synovial thickening.

Measure ROM of the affected hip and compare it to the opposite normal side. Comment if it is painful and be careful not to hurt the patient.

> 'There is a mild/moderate/gross painful restriction of all ranges of movement in the hip.'

Go on to perform a neurovascular examination of the lower leg.

> 'Examination of the spine was normal with good forward flexion, extension and lateral flexion demonstrated. Similarly, examination

of both knees was unremarkable Peripheral pulses were palpable with good capillary refill.'

Radiographs

AP radiographs may demonstrate old features of Perthes' disease: An elongated, deformed and flattened femoral head (coxa plana) with subluxation, a sagging rope sign (a classic radiographic feature of Perthes' disease, edge of the flattened femoral head) and coxa vara (femoral neck angle $<120°$). Mention the radiographic differential diagnosis for Perthes' disease and, if possible, Stulberg's radiographical classification of residual deformity and degenerative joint disease (but be sensible – If you are only vaguely familiar with it, don't go there).

Management options

The examiners may ask about treatment options of Perthes' disease during childhood as part of an intermediate case discussion[c]. With patients in their early 40s, the choice is between continuing conservative management and THA.

Technical difficulties with THA in Perthes' disease

An anteverted femoral neck may mislead the surgeon during stem insertion and lead to component malpositioning and increased risk of dislocation or proximal femoral fracture. A previous femoral osteotomy may cause difficulties in reaming the femoral canal. If prior hip surgery has been performed there is an increased risk of HO, infection, scarring, distorted anatomy, contracted musculature, etc. If significant hip shortening exists pre-operatively aim to equalize leg lengths. Avoid over-lengthening as generally not well tolerated. Pre-operative templating, intra-operative assessment (leg length calipers, ruler).

Pre-arthritic and early arthritic hip disease in a young adult with Perthes' disease

History

In Perthes' disease the head heals with coax magna and/or asphericity. In time, the acetabulum remodels and appears dysplastic. Young adults with Perthes' disease may present with anterior impingement pain and hip instability symptoms, which occurs as a result of secondary acetabular remodelling in response to the development of coax magna.

Get a detailed description of pain characteristics, activity level, associated co-morbidities and any previous hip surgery.

Are symptoms mainly associated with weight-bearing activities or hip flexion positions such as sitting? Hip pain exacerbated by sitting is commonly associated with femoroacetabular impingement. A history of true locking or catching suggests an intra-articular mechanical problem, such as an acetabular labral tear or chondral flap.

Clinical examination

- Gait
- Limb lengths
- Hip range of movements
- Patients with classic hip dysplasia usually have good flexion and internal rotation in flexion whilst patients with femoral acetabular impingement have restricted hip flexion and reduced internal rotation in flexion. With more advanced OA there will be a FFD and limitation of flexion, and the leg tending to go into external rotation (ER) with flexion
- Anterior impingement test. Passively flex, adduct and internally rotate the hip. If reproduction of groin pain considered a positive test. A positive test may be accompanied by a crepitus, clicking or a popping sensation. The test compresses the anterior surface of the labrum. It is a sensitive screening test for patients with acetabular labral disease and impingement. It can also be used as a non-specific screening tool for intra-articular disease and hip joint irritability

Management

Proximal femoral deformity may be corrected with a proximal femoral valgus osteotomy using a lateral approach and osteochondroplasty of the head–neck junction via a separate anterior arthrotomy. Alternatively, a transtrochanteric surgical hip dislocation provides good exposure for full surgical correction of the proximal femoral deformity.

Patients with pre-existing acetabular dysplasia may develop notable instability, necessitating surgical correction with PAO. The procedures may be staged, with surgical hip dislocation performed first to address FAI and with PAO done later to restore hip joint stability. Secondary hip instability may be apparent intraoperatively following surgical hip dislocation and osteochondroplasty. In this situation, it may be preferable to perform both procedures at the same setting.

Examination corner

Intermediate case 1: Perthes' disease

History

EXAMINER: Can you briefly summarize your history?

CANDIDATE: In summary, we have a 43-year-old man with Perthes' disease of his right hip. His hip has become increasingly painful in the last 3 years with a walking distance of 1 mile, interfering with his work as a railway engineer and also activities of daily living.

STOP STOP STOP[d]

[c] Please see website for additional information www.postgraduateorthopaedics.com.

[d] Summarize in two to three sentences maximum. The examiners wants this to act as an end to the history and focusing point for the beginning of the clinical examination. Do not get over excited or nervous and start repeating the full history you have obtained.

He is able to put his shoes and socks on, get in and out of a bath and car. He sometimes has difficulty getting up and downstairs at home. He is taking analgesia intermittently when his hip is particularly painful. When he needs analgesics he usually takes paracetamol but occasionally needs dihydrocodeine. He does not use any walking aids.

EXAMINER: I have asked you to summarize the history not repeat it!

Examination

EXAMINER: We would like you to examine his hip and talk us through it as you go along.

CANDIDATE: On examination we have a patient of muscular build, average height. Turning towards his right hip he has mild right proximal thigh muscle wasting, but from the back his gluteal muscles seem reasonably well preserved[e].

EXAMINER: If you look very closely there is in fact a small amount of gluteal wasting, which is apparent when you compare it to the opposite side.

CANDIDATE: (*I didn't look closely enough and missed this subtle clinical finding.*) Examining his gait he walks reasonably comfortable without any obvious abnormality present.

EXAMINER: I don't think that's quite the case. Could you just walk for us again, sir, away from us and then towards us. He demonstrates a mild antalgic right gait (not particularly obvious).

CANDIDATE: I will go on now and perform Trendelenburg's test. He is Trendelenburg positive on the right; I can feel his right hand push down on my left hand and his pelvis descends down to the right, indicating abductor muscle dysfunction.

EXAMINER: That's a good demonstration of the Trendelenburg's test except that you have tested the wrong leg[f,g].

CANDIDATE: I would like now to examine the hip supine. Could you lie down on the couch for me now, sir? His pelvis is level, the anterior superior iliac spines are at the same level and the legs are square with the pelvis and straight. The right leg is shorter than the left. I'd like to confirm this by measuring leg lengths formally. I'm measuring from the anterior superior iliac spine to the medial malleolus – On the right side the leg measures 91 cm; the left is 92 cm. Thomas' test reveals no fixed flexion deformity of either hip. There is a restricted range of movement of the right hip compared to the left. Flexion 70°, abduction 20°, adduction 10°, almost no internal or external rotation in flexion. Movements of the hip are painful, especially at the extremes of movement.

His spinal movements are unrestricted and pain free.

EXAMINER: Are there any other causes of referred pain to the hip?

CANDIDATE: The pain is arising from the hip joint. I have checked his spinal movements and these are normal. Forward flexion, extension, lateral rotation and lateral flexion are all full and pain free and straight leg raising was normal. These tests would seem to exclude the spine as a source of referred pain. The limb was neurovascularly intact with good peripheral pulses palpated and normal capillary refill. There are no clinical signs to suggest peripheral vascular disease

EXAMINER: Is there anything else that could be causing his pain?

CANDIDATE: Examination of the right knee was unremarkable. I can't think of anything else in particular that could be a cause of his pain.

EXAMINER: You have excluded the main causes of referred pain to the hip. Can you think of anything else?

CANDIDATE: No, sir.

EXAMINER: Have you heard of the piriformis syndrome?

CANDIDATE: No sir, sorry, I have not.

Piriformis test

The piriformis test is performed with the patient in the lateral decubitus position with the side to be examined facing up. The patient's hip is flexed 45° with the knee flexed about 90°. The examiner stabilises the patient's pelvis with one hand to prevent rocking. The other hand then pushes the flexed hip towards the floor. This manoeuvre stretches the piriformis muscle and elicits pain when the muscle is tight or involved with tendonitis. If the pain is not localized to the piriformis tendon but radiates in a manner suggestive of sciatica, a piriformis syndrome should be suspected. The piriformis syndrome is an uncommon cause of sciatica in which the radiation of pain along the course of the sciatic nerve is caused by entrapment within the piriformis muscle instead of lumbar disc disease.

This is not your everyday test performed in clinic. As such candidates may be under-rehearsed and find it difficult to perform smoothly in the exam setting.

Overview of clinical case:

- Detailed history of the treatment of Perthes' disease as child
- Demonstration of full hip examination
- Discussion about causes of referred pain to the hip (*Did you check his spinal movements?*)
- Piriform syndrome and how to test for it (*irritation of the sciatic nerve by the edge of the piriformis muscle*)

Discussion

- General discussion about the radiographs of the right hip: Moderate OA, features of old Perthes' disease, sagging rope sign. Shown arthrogram pictures and asked to comment on them, which led on to general discussion about the principles of arthrogram

[e] Do not miss obvious wasting!

[f] I got left and right mixed up but luckily the examiners let me off.

[g] This happens with alarming frequency in the exam. If your performance is otherwise good it isn't a big issue, but if you are ropey this really doesn't go down well at all with the examiners.

- Full discussion on the classification of Perthes' disease (Catterall, Salter and Herring). 'I went through each one in turn and then mentioned which one I preferred and why.' Discussion of Stulberg's[5] rating system of the femoral head at maturity and Herring treatment guidelines for Perthes[6]
- Management: The patient's hip arthritis was too good for THA so continue with conservative treatment for the time being. Is there a possible role for periacetabular osteotomy in the arthritic hip[h]? Discuss the figures for survival of THA in the young arthritic male (results from Wrightington[7] and Swedish Hip Register). In the Swedish Registry, the 15-year survival rate following THA was 77% for patients aged <45 years and 92% for patients aged over 65 years. THA in young patients remains a procedure associated with high risks of conventional polyethylene wear and osteolysis

EXAMINER: What type of THA would you perform and why?

CANDIDATE: I discussed the choice of cemented vs uncemented THA and ceramic on ceramic-bearing surface vs metal on polyethylene (MoP). The advantages of ceramic on ceramic (CoC) articulations include reduced wear, scratch resistance, reduced osteolysis, improved maintenance of lubrication with the wettability of ceramics producing excellent fluid film lubrication and excellent biocompatibility. CoC articulation is a very attractive option for young active patients in whom there are concerns with the long-term consequences of wear and osteolysis with MoP articulations.

The main disadvantages of ceramic-bearing surface relate to catastrophic head and liner fracture, squeaking (stripe line) and cost. I didn't go into specifics, but if you are potentially a score 7 or 8 you would need much more detailed knowledge about these complications.

'After everything we discussed I still think the examiners wanted me to say I would use a cemented MoPTHA.'

- Revision rate for cemented THA in young patients

Numerous studies have shown a revision rate significantly correlated with high activity level, unilateral disease, the number of preoperative hip operations, weight (>60 kg) and aetiology (DDH, trauma, AVN, juvenile idiopathic arthritis). All series have highlighted the greater risk of revision compared with that in older and/or less active populations

Intermediate case 2: A 44-year-old man with OA hip secondary to childhood Perthes' disease

Standard history

(See above)

Clinical examination

- Trendelenburg's – Positive. Quizzed about causes and significance
- Thomas' test. Fixed flexion deformity 10°. Scrutinized and quizzed during this test
- Demonstration of ROM hip. Reduced internal and external rotation in flexion. (Don't get the movements mixed up with each other!) Abduction and adduction. (Don't forget to stabilise the pelvis)
- Examination of LLD (2-cm shortening). Questioned about difference between real and apparent shortening
- Galeazzi's test

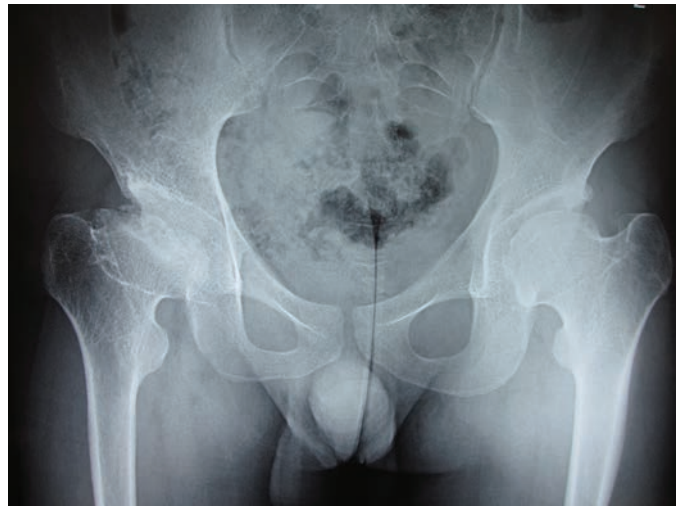

Figure 11.11 AP radiograph Perthes' disease right hip

Discussion

Radiographic features (Figure 11.11):

- Management options: THA required
- Consenting issues (DVT, PE, sciatic nerve injury/foot drop, LLD, dislocation, aseptic loosening, neurovascular injury, infection)
- Preoperative radiographic templating: Why do we bother with this? Select implants of appropriate size, recreate correct offset, and equalize leg lengths
- How to correct a LLD. Correct a real leg discrepancy but not an apparent one

The hip needing revision surgery

This is usually intermediate case material. Have a practised focused history relating to a painful THA prepared, as time is tight[i]. It is very easy to end up jumping about with the history without succinctly nailing down what the specific complaints are.

History

History of the implant[j]

When was the initial surgery performed?
How has the hip been since the operation?
Detail history of all prior hip operations

[h] Know the prerequisites for a periacetabular osteotomy as the examiners could easily lead into this with follow-up questions.

[i] This will score you a 7 rather than a 6, or a comfortable 6 rather than a borderline 6.

[j] The history of the implant is the most obvious lead in question at the beginning of the history. Focus then on taking a good pain hip history as that is probably the principle complaint.

Any historic factors to suggest infection[k].

Any complications in the postoperative period?

Enquire about any delays in wound healing, haematoma evacuation, excessive or persistent wound drainage and antibiotic usage at the time of primary surgery. Is there any history of urinary catheterization following surgery?

Has the hip always been painful or just in the last year or so? Any recent chest or urine infections or generalized systemic upset?

The infective hip questions are important and failure to ask them will suggest that a candidate hasn't considered infection as a possible cause of hip pain.

Pain. Most patients complain of as aching type of pain, which is mechanical in nature. It is typically provoked by activity and relieved by rest. The intensity is usually variable although not often great. Groin and/or buttock pain is typical of acetabular component loosening. Femoral stem loosening more often causes thigh pain. Aseptic loosening is often associated with an initial marked exacerbation of discomfort when the patient first stands up (start up pain) which reaches a steady state over the next few minutes and thereafter the pain may reduce. It is characterized by a pain-free interval following the initially successful arthroplasty surgery.

Patients with septic loosening may give a history of pain, which has persisted since the time of the original operation. Alternatively, there may be a sudden onset of pain following spread of infection from some distant septic focus. The pain itself is typically insidious in onset and both gradually and relentlessly progressive.

Ambulation capacity: How far can you walk? Patients may complain of a limp and a progressive reduction in walking distance. They have difficulty in climbing stairs

Shortening: Do you feel short or that your leg lengths are equal? Progressive shortening may be noticed by the patient. This may be caused by proximal and medial migration of the acetabular component with or without subsidence of the femoral component

Stiffness: Difficulty in donning shoes, putting on socks, cutting toenails bending down to pick objects off the floor, etc

Instability: Recurrent episodes of subluxation or dislocation. Instability can cause pain from capsular stretch and soft-tissue impingement. Sympoms can usually be reproduced by placing the limb in a certain position and usually recur each time that position is re-created

Sepsis: Easier if this is dealt with during the history of the implant

Referred pain: Lumbar spondylosis, spinal stenosis and sciatica, peripheral vascular disease may all provoke discomfort, which resembles hip pain

Co-morbidity factors: Patients requiring revision are often elderly with medical co-morbidity factors

Clinical examination

Look, feel, move.

Carry out a general inspection to include general stature, height and weight. Examine the quality of skin overlying the joint. Note any previous skin incisions, signs of infection. Mention any obvious muscle wasting, clinical deformity/shortening.

Gait should be carefully observed to look for antalgic gait, limb-length discrepancy or abductor deficiency. A marked Trendelenburg gait suggests non-functioning abductors that may be due to paralysis or loss of continuity.

Trendlenberg's test could be positive.

Wound inspection is important to help plan operative incision relative to previous incisions. It is not advisable to make a second, parallel incision, especially if the previous incision was posterior.

The onset of limb length inequality should be related to the time of the operation, as progressive shortening and muscle weakness may indicate subsidence of one of the components. If leg length shortening is present ensure you do an apparent and true measurement. Galleazzi's sign and Bryant's triangle to identify the area of shortening.

Assess movements both active and passive and comment on range and if any pain is present (do not hurt the patient any further if he/she has pain). Be careful not to dislocate the hip.

> 'I will go on now to test for hip range of movement but I will be careful not to hurt the patient or be too forceful in my movements to avoid any risk of hip dislocation[l].'

Specific restrictions in ranges of movement may be related to impingement, contracture or heterotopic bone formation. Pain at the extremes of movement may indicate impingement or loosening of prosthetic components. Pain with the leg jerking into internal or external rotation is suggestive of femoral component loosening. Pain in the groin with resisted straight leg raising is suggestive of acetabular loosening. Examination should be completed with assessment of neurovascular status in particular assessment of sciatic nerve function.

Radiographs

While describing the radiographs (AP/lateral) look for dates, type of prosthesis (primary/revision, cemented/uncemented), evidence of femoral loosening (Gruen zones), acetabular

[k] The infective hip questions are important to ask. If a candidate doesn't ask them it suggests that a candidate hasn't considered infection as a possible cause of hip pain and is likely to lose them a mark.

[l] It is probably safer to say this comment in the exam as it covers you to a certain extent and reassures the examiners that you are aware of possible dislocation. What you want to avoid is being a bit rough with the patient forcing hip movements, perhaps causing pain and appearing not to appreciate that you may dislocate the hip. This scores a 4 or 5 at best.

loosening (De Lee and Charnley zones) and for any cement extrusion. Compare with old radiographs.

Any medial migration of the component or placement of the cup medial to the Kohler's line should be mentioned. Further investigations such as a CT scan or angiogram may be required. Areas of cortical thinning, perforation or scalloping should be mentioned. Assess the bow of the femur (lateral view), which may have implications on using a long femoral stem. Look for any hetrotrophic ossification. It is useful to know of the classifications for femoral and acetabular bone loss (Praposky classification, AAOS classification).

Discussion

Mechanisms of failure

The principle causes of failure may be related to patient specific features, implant design features or variations in surgical technique.

Patient-related factors. Variables such as young age at primary procedure, increased physical activity, male gender, obesity, primary joint replacement following pelvic or femoral fractures all result in higher revision rates. Rheumatoid arthritis (RA) or OA secondary to childhood disorders such as slipped capital femoral epiphysis (SCFE), Perthes' or DDH may similarly compromise the long-term results of primary arthroplasty.

Implant-related factors. Faulty design, inferior material implant characteristics, implant fracture, periprosthetic fracture, delamination of the porous coating.

Surgeon-related factors. Inadequate preoperative aseptic precautions, prolonged operating time, mal-positioning of components, inadequate cementing techniques surgeon and hospital volume.

Indications and contraindications for revision hip surgery

Indications include aseptic loosening, deep sepsis, periprosthetic fracture, catastrophic implant failure, osteolysis, PE wear and recurrent dislocation. Contraindications include medically unfit patient, compromised bone and soft tissues, etc.

Differentiation between aseptic loosening and infection

Blood tests, hip aspiration, bone scan, intra-operative frozen section histology, intraoperative tissue culture etc.

Preoperative planning[m]

Essential for the successful outcome of surgery and to avoid complications:

1. Good quality serial radiographs of the hip and pelvis including lateral views of the femur to allow longitudinal comparison and evaluation of component migration, cement column fractures and the development of progressive radiolucencies
2. Array of full surgical equipment should be available
3. Position of previous skin incisions, check old notes for approach used
4. Decide on the choice of surgical approach beforehand (personal preference of surgeon, nature of osseous defects, type of implant, previous skin incision)
5. Order bone graft if needed
6. Large prosthetic inventory needed
7. Ensure access to the original operation note if possible and information about the prosthesis to be removed, e.g. head size, bearing surface etc
8. If intrapelvic cement is present or the acetabulum markedly protruding may require an intravenous pyelogram (IVP) and angiography
9. Complex acetabular defects may require accurate assessment with three-dimensional CT scan reconstruction of defects
10. Implant company representative to be present at time of surgery
11. Possible need for ITU bed

Surgical approaches used and equipment necessary

Equipment issues to consider would be:

1. Stem extraction instruments
2. Screws, pelvic reconstruction rings, porous tantalum revision shell with augments
3. Allograft bone (fresh frozen femoral head and/or freeze dried bone chips)
4. Trochanteric fixation devices and circlage wires
5. Hand or motorized cement removal instrumentation
6. Flexible medullary reamers
7. Fibreoptic lighting may be especially useful for visualisation of the distal part of the femoral canal
8. Pneumatic drills and burrs
9. Cement chisels and splitters
10. Flexible thin osteotomes
11. Canal plug removal instruments
12. Cell saver equipment
13. Bone graft mincer

Revision considerations

- Cement in cement revisions

  If the cement mantle is well fixed with no apparent defects it may be possible to cement a new stem into the existing cement mantle, downsizing the stem. Meticulous surgical technique is required to ensure a clean, dry femoral canal as a thin layer of blood or marrow may cause up to an 85% reduction in shear strength and 80% reduction in tensile strength of the cement–cement interface

[m] Similar to the core hip topics section. There will be slight variations on a theme with the individual case that is being discussed.

Examination corner

Intermediate case 1: A 77-year-old male, uncemented loose THA at 18 months

History

Uneventful postoperative recovery
Walking distance restricted to 200 yards
Mid-thigh pain, dullish pain[n] at rest, reaching peak in the start-up phase, diminishing over movement

Clinical examination

As per hip examination
Little to find
Trendelenburg's test, Thomas' test, ROM

Differential diagnosis

Infection, radicular pain, trochanteric or iliopsoas bursitis

Discussion[9]

Failed early osseo-integration of an uncemented THA
Superficial discussion without any probing in detail
Failure to appreciate the possibility of infection was a significant omission and a negative marking point
Radiographs of the hip were essentially normal without any features of frank loosening (subsidence, pedestal formation, cortical hypertrophy, and increasing radiolucencies)
Bone scan was hot but this is non-specific for loosening and did not differentiate from infection
A CT scan was shown which demonstrated failure of osseo-intergration. Discussion about the metal artefact from the femoral stem making interpretation difficult. The possibility of the CT being used to diagnose infection–periosteal reaction would be typically seen
PET scan is another new imaging modality used to diagnose an infected THA

Discussion topics for score 8 candidates[o,p]

Aetiological models-tip micromotions and tip overload
Rigidity mismatch
Engh's biological fixation classification into bone ingrowth fixation, stable fibrous fixation or unstable[10]
Extensively coated porous stems
Factors associated with Stress shielding. Main factor is stem stiffness affected by stem diameter, metallurgy, stem geometry.
Characteristic features of an uncemented stem
How do you revise an uncemented THA[q]?
Removal of well-fixed uncemented femoral stem is difficult requiring specialized equipment, time and patience along with experience and knowledge of the implant (promimally or extensively coated). An extended trochanteric osteotomy (ETO) is usually required
The Explant™ system allows safe penetration at the metallic cup bone interface of the acetabulum. Clear bone in path of femoral implant with flexible osteotomes, extraction kit.
Gigli saw on medial border stems. Do not remove femoral implant until all ingrowth interfaces are divided. Be careful with osteotomes. They tend to fracture cortical bone rather than cut it

[n] In typically cases the thigh pain is absent at rest.
[o] Score 8 candidates by definition will fly through the initial material to discuss progressing onto more advanced topics that will stretch both candidates and examiners.
[p] Some of these topics may be more at home with a basic science viva. The clinicals should be more biased towards testing candidates on clinical relevant material.
[q] This is a 1-minute answer in either the vivas or clinicals in which candidates just need to keep talking. Much better if they have some practical knowledge of the potential difficulties.

Paget's disease

Hip pain associated with Paget's disease may cause diagnostic problems. It can be difficult to distinguish whether the pain is due to active Paget's disease or to degenerative hip arthritis. Both can give a dull, aching pain that may worsen with weight-bearing. Relief of discomfort with intra-articular local anaesthetic hip injection suggests coxarthrosis as the source of pain. A therapeutic trial of calcitonin may also be helpful to differentiate between the two causes.

Exclude other sources of pain such as referred pain from spinal stenosis or radiculopathy, stress fracture and other causes of musculoskeletal pain. If the character of the pain changes consider the possibility of sarcomatous change.

Clinical examination

'On general inspection there is enlargement of the skull. There is also bowing of both the femur and tibia in both legs in both an AP and lateral direction. The sharp anterior edges of both tibias are thickened and curved, making them very prominent and giving an almost sabre tibia appearance to them.'

'On palpation of both lower legs there was no suggestion of increased warmth present (due to increased vascularity).'

'The spine has a uniform even kyphosis present (vertebral involvement leads to loss of height and kyphosis from disc degeneration and vertebral collapse).'

'The shoulders are rounded and the head and neck protrude anteriorly. The skull enlargement occurs in the vault and the enlarged frontal bones make the forehead bulge forwards. His arms appear to be disproportionately long (because of the kyphosis).'

'Examination of his left hip revealed gluteal and thigh muscle wastage. He had a marked stoop present, attenuated by bowing of both his femurs. Examination of gait revealed that it was antalgic in nature. Trendelenburg's test was markedly positive on the right side, delayed positive on the left side. Examination supine revealed equal leg lengths. On palpation of the hip there were no obvious areas of tenderness. Thomas' test revealed a fixed flexion deformity of 30°. Flexion of the hip was painful and reduced to 70° actively and could not be increased appreciably passively. Internal rotation in flexion was zero whilst external rotation in flexion was grossly reduced to a jog of movement only. Similarly, adduction was limited to 20° and abduction 30° passively, and was also painful. Distal pulses were palpable with good capillary refill, and neurological examination of the lower

legs was normal. There was marked restriction of all spinal movements, in particular forward flexion – He was only able to touch his knees. Straight leg raising reproduced low back pain but no true sciatic nerve root irritation.'

Discussion

Paget's disease is a chronic deforming metabolic bone disease characterized by increased osteoclastic bone resorption and compensatory increases in bone formation. In the later stages of the disease the involved bone becomes enlarged, dense, sclerotic with an irregular trabeculae pattern, obliterated medullary canal and thickened cortices. The poor structural integrity of the bone renders it prone to either pathological fracture or repetitive stress fractures. Progressive deformity and secondary OA of the hip affects between 30% and 50% of patients. For Paget's disease that involves the hip with secondary degenerative changes, surgery is indicated to manage significant pain, joint stiffness, deformity or a pathological fracture.

Preoperative treatment with bisphosphonates or calcitonin is recommended to reduce the incidence of intraoperative bleeding, heterotrophic ossification and loosening although no randomised control trial exists. There is a potential for significant intraoperative bleeding from hypervascular and osteoporotic bone, technical difficulties prolonging the length of surgery or the possible need for concomitant procedures (osteotomy). Excessive bleeding may require additional cross-matching of blood. Consider using tranexamic acid.

Proper preoperative templating and planning is necessary to size an enlarged medullary canal and determine the correct component size and the amount of cement to be used.

A broad spectrum of deformities of the proximal femur or acetabulum may hamper dislocation of the hip, exposure of bone or component alignment. Trochanteric osteotomy may be required for adequate exposure and beware the sciatic nerve is nearer the joint than normal. In the presence of protrusio acetabulum combined with coxa vara dislocation of the hip can be extremely difficult and the neck may need to be cut in situ. Coxa vara predisposes to a varus femoral stem position.

A marked deformity of the proximal femur with coax vara or anterolateral bowing of the femoral shaft may require a corrective osteotomy prior to THA. This will allow correct alignment of the femoral component at the time of THA. The presence of dense sclerotic bone may make reaming and bone preparation difficult. Sharp reamers will be necessary to shape the femoral canal.

If protrusio acetabulum exists ream to expand the periphery without deepening the socket to avoid causing added protrusio. Consider what method of cup fixation to use either uncemented acetabular fixation with supplementary screws to prevent cup migration and allow for boy ingrowth or a cemented cup. Inability to produce a dry acetabular bed may compromise cement interdigitation with bone. Reconstruct the acetabulum with the hip centre in the anatomical location with medial acetabular bone grafting or the use of an oversized hemispherical cup as well as the availability of offset liners to compensate for a medialized cup position in a protrusio defect. Cement fixation in combination with a cage or an uncemented cup-cage construct with a cement liner, may be required if extremely poor bone quality or significant bone loss is found.

A widened femoral canal can either be dealt with by using extra cement or by primary impaction grafting with cancellous allograft chips. Obliteration of the femoral canal with bone as opposed to cement is thought to provide a much more durable anchorage of the stem but is time consuming and technically difficult. A large cement restrictor or bone plug may be required. Use of a long cemented stem, an extensively coated uncemented stem or a modular tapered stem may be required to bypass mechanically insufficient proximal bone and achieve diaphyseal fixation.

There is an increase incidence of heterotrophic ossification; therefore, consider prophylatic measures.

Concern exists with the use of uncemented implants in Paget's disease due to possibility altered bony ingrowth. Wegrzyn et al.[11] reported 84% excellent or good medium-term outcome in 39 uncemented hips performed for Paget's disease of bone (PDG) at an average of 7 years follow up. No revisions performed.

Radiographs

Sclerotic appearance of bone with cortical thickening; trabeculae are coarse and widely separated.

Differential diagnosis: Osteitis fibrosa cystica, fibrous dysplasia, osteoblastic secondaries, osteopetrosis and lymphoma.

Examination corner

Intermediate case 1

EXAMINER: What are the indications for therapy in Paget's disease?

CANDIDATE:

- Bone pain
- Deformity
- Fracture
- Osteolytic lesions in weight-bearing bones
- Immobilization hypercalcaemia
- Markedly increased alkaline phosphatase
- Nerve compression
- Young age, especially if disease very active
- Before orthopaedic surgery

Ineffective for:

- Deafness
- Fissure fracture
- Sarcoma

EXAMINER: What are the causes of a bowed tibia?

CANDIDATE: True bowing caused by softening of bone occurs in Paget's disease and rickets. Apparent bowing owing to thickening of the anterior surface of the tibia secondary to periostitis occurs in congenital syphilis and yaws.

EXAMINER: What are the complications of Paget's disease?

CANDIDATE: Progressive closure of skull foramina may lead to:
- Headaches
- Deafness
- Blindness (optic atrophy)
- Tinnitus
- Vertigo

Other complications include:
- High output cardiac failure
- Pathological fractures
- Sarcoma change in bone
- Urolithiasis
- Spinal stenosis
- Hypercalcaemia

A variable pattern of presentation. Only a minority of patients become symptomatic.

EXAMINER: How common is sarcomatous change in bone?

CANDIDATE: It is very rare, probably <1%, but it carries a very poor prognosis. The patient presents with increase in pain and swelling.

EXAMINER: What biochemical abnormalities occur in Paget's disease?

CANDIDATE: Serum alkaline phosphatase and urinary hydroxyproline are elevated except sometimes in very early disease. Serum calcium and phosphate are usually normal in mobilized patients but can occasionally be increased or decreased. Urinary calcium rises in immobilized patients.

Intermediate case 2: Paget's disease

Discussion

A radiograph is shown of Paget's hips. (Figure 11.12).

EXAMINER: These are the radiographs of a 78-year-old female who is complaining of predominantly left-sided hip pain.

- Discussion of Paget's hip
- Radiographic features
- How to manage
- Bisphosphonates – How do they work?
- Precautions prior to THA

The control of Paget bone activity in the preoperative period reduces the risk of implant loosening and the abnormal intraoperative blood loss.

- Technical difficulties of THA

Short case 1: Paget's tibia

EXAMINER: Examine this man's leg. What is the diagnosis?

CANDIDATE: Bowed, enlarged leg, no other deformities, only other clue was hearing aid *(look around for any clues which may point you in the right direction)*. The leg is bowed because new bone is deposited beneath the periosteum on one aspect giving the illusion of being bent.

CANDIDATE: The examiner wanted me to mention 'sabre tibia'. In the tibia the forward bowing confirmed in lateral radiographs may be referred to as a sabre tibia (since the front of the tibia is blunt rather than sharp this is hardly appropriate). Also, this is not strictly correct, as sabre tibia can also be associated with syphilitic disease of bone. Syphilitic osteoperiostitis occurs late in the disease, on average 6 years after untreated syphilis. Usually one bone becomes painful and tender. Often the bone gives an illusion of being bent, because new bone is deposited beneath the periosteum on one aspect.

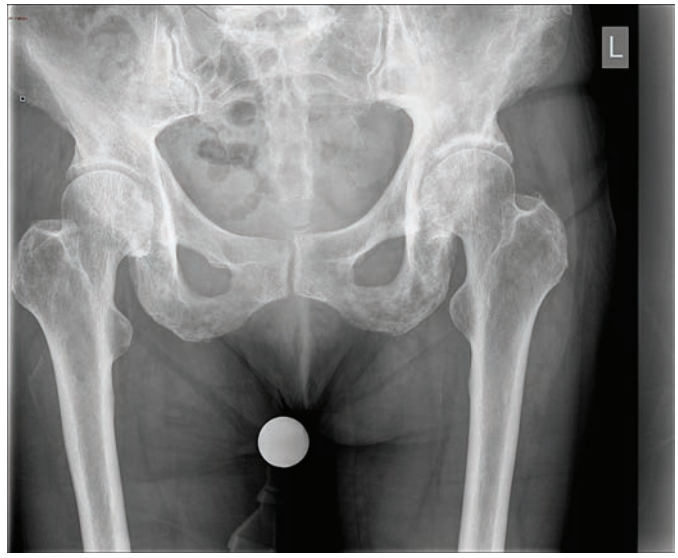

Figure 11.12 Paget's hip Cortical thickening and coarse trabeculations

Intermediate case 3: Paget's disease

- Pathogenesis of Paget's disease
- Complications
- Radiographic features
- Cause of bone bowing
- Management of pathological fractures
- Problems encountered in joint replacement – How do you control bleeding?

Post-traumatic OA hip

Accidents involving high-energy trauma may cause fractures of the acetabulum and fractures/dislocation of the hip joint. The treatment of choice for post-traumatic OA in patients over 50 is THA, but a younger patient is more likely to place higher demands on any implant.

History

- Age
- Occupation
- A full history should be obtained starting with the presenting complaint and any symptoms or disability experienced by the patient
- Full details of the original accident including the mechanism, all injuries sustained and the time course of subsequent treatment

- The timing and nature of all operative procedures and the development of any complications should be documented with specific enquiry about any systemic or wound infections or thromboembolism

Progress since the injury

- The onset, nature, progression and aggravating and relieving factors or symptoms should be established
- Social (smoking, alcohol) and general history
- Systematic enquiry
- Expectation and ambitions of the patient

Examination

- Comment on the patient's posture, stance and gait patterns. Scars inspected for site as this may interfere with subsequent surgery, any evidence of infection. Trendelenburg's test for abductor function
- True and apparent leg lengths for adduction contracture. The mechanical axis of the legs and true leg lengths should be compared for the effects of the hip pathology or any associated injury to either lower leg
- Thomas' test is used to detect a fixed flexion deformity.
- Examine ROM of the hip and the presence of pain or fixed deformity
- Muscle power, tone and distal neurovascular status should be checked for evidence of impairment due to nerve palsy or vascular injury
- Knees, the contra-lateral hip and the lumbar spine should be thoroughly examined especially if arthrodesis is being considered

Examination corner

Intermediate case 1: Post-traumatic AVN hip

A 16-year-old girl who had been involved in a RTA 1 year previously and had developed AVN with secondary OA of her left hip.

She had sustained a closed fracture of the right femoral shaft, which was treated with skeletal traction, and a traumatic posterior dislocation of her left hip. The left hip had been reduced under GA fairly promptly after admission. However, she continued to complain of left hip pain following relocation and 4 days later a further radiograph was taken, which showed a displaced fracture of the femoral head. This was fixed with a cannulated hip screw the following day.

Essentially her presenting complaint was severe constant pain in her left hip, interfering with every aspect of her life. Her walking distance was reduced to a few hundred yards, sleep was severely affected and she was taking regular analgesia with minimal benefit.

CANDIDATE: On general inspection we have a young girl of average height and build. There is an old longitudinal lateral scar present over the right proximal thigh. There are other numerous scars, both surgical and non-surgical present over the left and right lower legs. Her pelvis is not level when standing: The ASIS is lower on the left side. She appears to have a leg length discrepancy: The left leg appears to be shorter than the right. I didn't appear very confident when asking the patient to level up her pelvis as I was getting confused trying to formulate a diagnosis . I also omitted to mention using blocks to level up her pelvis

CANDIDATE: She walks with an antalgic gait. 'Can you lie down for us now so that we can continue with the examination?' 'On inspection supine . . .'

EXAMINER: What about Trendelenburg's test. I completely forgot about Trendelenbug's test and then made a hash of it .

The patient had a severe adduction contracture of her left leg, which gave the erroneous impression of severe shortening on the left side. However, most of this was apparent shortening. True shortening of the left leg was minimal and probably not significant.

EXAMINER: How do you know the left leg is short? There was a femoral fracture on the right side. Could the left leg not be a normal length and the problem be a longer right leg secondary to the right femoral fracture? In fact, the right leg could be shorter than normal but the left leg could be even shorter because of the hip condition.

EXAMINER: Could you demonstrate Thomas' test for me?

EXAMINER: You must put your hand properly behind the lumbar spine. Let me show you: This is where your hand should be. It should go all the way behind the small of the patient's back. The patient has a fixed flexion deformity of 20°.

EXAMINER: Can you measure movements of the hip?

CANDIDATE: She flexes the hip from 20° to 100°.

EXAMINER: You must stabilise the pelvis when testing for flexion of the hip as the pelvis moves a lot sooner than you realize.

Discussion

Discussion centered on the management of this patient. She was too young for a THA and unlikely to be happy with a fusion. Secondary OA was too far advanced for an osteotomy and the condition was too painful to do nothing. No definite management plan was agreed upon.

Radiographs of the initial dislocation were shown. The day 4 radiographs showed a transepiphyseal fracture of the femoral head through the proximal femoral physis. The examiner described it as a type of SUFE. Delbert's classification of hip fractures in children was briefly mentioned. Discussion then followed of the postoperative radiograph, which showed the fracture adequately fixed with a single cannulated hip screw. The candidate was asked about the entry point for cannulated screw fixation for SUFE (it is not the dynamic hip screw (DHS) entry point for a proximal femoral fracture, which is much more anterior).

Transepiphyseal fractures represent about 8% of all hip fractures in children. They may occur with or without dislocation of the femoral head, and results are generally poor owing

to a combination of AVN and premature closure of the physis. The diagnosis is often delayed because of concomitant injuries. The patient had developed AVN of the left hip but we didn't discuss this.

Fail: Most of the intermediate case can go very well but the differentiating feature is the three or four mistakes that are made with a candidate sometimes not appreciating their full significance: (1) there was a silly mix up in the history summary – She was a pedestrian not a passenger in a car; (2) a poorly performed Thomas' test (this is a very common mistake); (3) Failure to stabilise the pelvis when testing for flexion; (4) Forgetting Trendelenburg's test and then not properly explaining the test to the patient. The candidate was at the end of the day so the patient intuitively knew what to do, but if the candidate had been first one in – A waiting disaster!

Arthrodesed hip[r]

This is classic material for the hip intermediate case, in which there are good clinical signs to demonstrate and enough to talk about in the discussion afterwards. At least one arthrodesed hip is usually present in the short case examination hall.

Memorandum

'On general inspection the patient has a walking stick visible in the corner. He also has a shoe raise in the right foot. Looking at his right leg he has gross muscle wasting of the thigh and gluteal muscles. There is a well-healed extended longitudinal scar over the right proximal thigh. He has an obviously shortened flexed right leg. His pelvis is not level with stance. The ASIS is hitched up on the left side and he has a compensatory scoliotic curve of his spine apex to the right. He demonstrates a short leg walk with a slow asymmetrical and arrhythmic gait. He is Trendelenburg positive on the right side. There is increased motion in the normal (sound) hip and increased flexion of the knee throughout the stance phase on the fused side.

'Sitting down on the bed the pelvic obliquity does not fully correct and he still demonstrates a scoliotic curve of his spine suggesting an element of fixed deformity to the spine.'

'On inspection supine we can see the quite obvious leg length inequality on the right side. On measuring leg lengths there is a 5 cm true difference. Thomas' test reveals a fixed flexion deformity of his right leg of 30°. On attempting to flex the hip the pelvis moves immediately, which is very suggestive of a fused hip. In addition, there is no adduction/abduction possible at the hip joint'.

Salient clinical features

- Scar
- Fixed flexion deformity
- Shortening
- No movement
- Check nerve function

Pitfalls

Whatever method you use to measure hip movement you must stabilise the pelvis to detect its movement. It is absolutely vital to keep one hand over the ASIS when measuring flexion so as to detect tilting of the pelvis. It is possible to 'flex' a completely fused hip by 30–40° – the movement actually occurring at the spine. One will not pick up the diagnosis of a fused hip in this situation.[s]

The patient can sometimes have a pseudo-arthrosis of the hip, which allows some movement to occur at the hip, and this may create confusion. If the good leg is flexed up and the patient holds their knee, use one hand to palpate the lesser trochanter and iliopsoas and then with the other hand smartly abduct the arthrodesed leg. If there is protective contraction of the muscle group then the arthrodesis is not sound – the unsound arthrodesis (spasm of muscles).

An arthrodesed hip is often seen with tuberculosis and a fixed contracture of the ipsilateral knee. Be careful when measuring leg lengths to place the opposite leg in the same position. This may require the use of pillows to flex up the opposite leg. It may even require you to measure the component parts of the legs separately. Anterior superior iliac spine to medial joint line, medial joint line to ankle, etc. With fixed knee flexion when checking hip flexion move the patient to the end of the couch to eliminate the effect of a fixed flexion deformity at the knee.

Often the ipsilateral knee is limited in motion and is painful on weight-bearing in a strained valgus position.

When performing Thomas' test in a patient with an arthrodesed hip lift the ipsilateral hip until the lumbar curve flattens. At this point this measures the fixed deformity of the hip.

Discussion

Indication

May be indicated in patients with severe femoral or pelvic deformity that precludes THA, in neurological cases where the risk of dislocation is high or with an increased risk of THA failure in a young highly active patient with monoarthrosis of the hip. The ideal candidate is an adolescent or young adult with a history of multiple hip surgeries, post-traumatic arthritis, and/or post-infectious hip disease. Should be minimal pre-existing arthritis of the lumbar spine, ipsilateral knee or contra-lateral hip

[r] Please see website for additional information: www.postgraduateorthopaedics.com

[s] This is regarded as an absolutely classic examination error. If one mentions that there is 30–40° of flexion in the hip when it is fused one will definitely fail a short case, whilst in the intermediate case you will be on the back foot and have your work cut out to recover.

Not a popular procedure because patients are familiar with the impressive results from arthroplasty and often find it difficult to accept hip arthrodesis.

Arthrodesis may be mentioned as a possible management option in the young patient with severe osteoarthritis affecting the joint.

> 'I would at least discuss the option of hip fusion with the patient even if they were likely to refuse it'

Position of fusion

- 20–30° flexion
- Neutral or slight adduction (0–5°)
- 5–10° external rotation
- Avoid abduction and internal rotation
- LLD <2 cm
- Abduction creates pelvis obliquity

This position is design to minimize excessive lumbar spine motion and opposite knee motion which helps minimize pain in these regions. Excessive adduction in women will cause difficulties with sexual intercourse and wetting the inside of the thigh during urination. Insufficient flexion makes sitting extremely difficult and excessive flexion accentuates LLD. Increased hip flexion best suited for a patient with a desk job, less flexion if the job is manual labour.

Methods of arthrodesis

- AO Cobra plate. Involves osteotomy of the greater trochanter. Stable fixation but disrupts hip abductors which may be problematic if subsequent THA
- Trans-articular sliding hip screw. The lag screw is inserted across the joint and just superior to the dome of the acetabulum. Poor fixation achieved due to large lever arm and the resulting torque on the lever arm
- Combined intra-articular and extra-articular fusion. Some form of bone graft is required

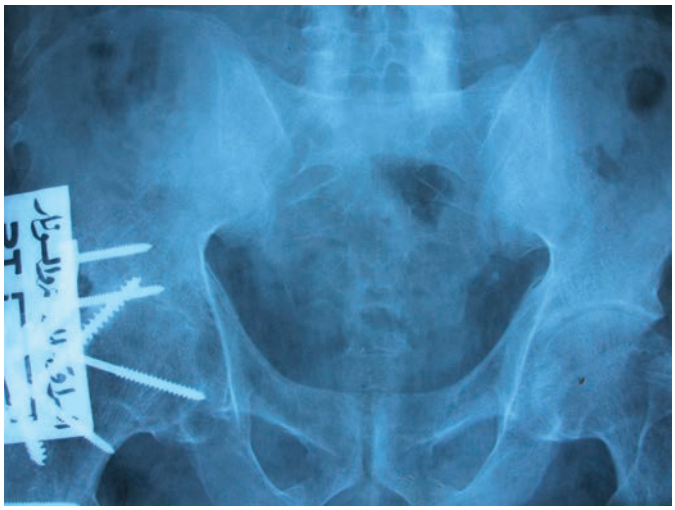

Figure 11.13 Fused right hip

Complications

- Neurovascular injury
- Femoral fracture in the first year following surgery
- Failure of internal fixation
- Non-union (pseudoarthrosis rate 15–25%)
- Mal-union
- OA contralateral hip, both knees, spine
- Instability ipsilateral knee

Revision arthrodesis to THA (taking down the hip)

The older patient requesting conversion to THA following previous arthrodesis is a much more likely intermediate case than a young patient in whom a hip fusion is being considered.

- Conversion for severe persistent low back pain
- Painful pseudo-arthrodesis
- Mal-positioned arthrodesis
- Ipsilateral knee pain
- Contralateral hip pain
- Limitation of walking distance
- Difficulty in performing ADL mainly involving hip flexion such as bending, putting shoes and socks on and tying shoe laces
- Sitting can be uncomfortable for prolonged periods or in cramped places

The origin of pain should be accurately defined, and the functional demands and expectations of the patient explored. It is important to make sure the back or knee pain is not caused by other pathology, which would not be improved by THA. The original reason why the arthrodesis was performed should be sought. If the arthrodesis was performed following infection make sure active infection has been excluded. Ensure that the patient has a good indication for conversion. In patients with long-standing ankylosis from childhood infections, there is extensive scar formation, limb shortening and decreased size of the hemipelvis and proximal femur. This will necessitate the use of smaller components.

Examine whether the hip is soundly fused, the amount of limb shortening (this can be difficult to assess). Palpate the hip abductors for bulk and defects and test for hip abductor strength. Good quality radiographs to identify bone stock, hardware, status of the greater trochanter. A CT scan can sometimes be helpful for identifying bone stock, the proximity of heterotopic bone to neurovascular structures and the abductor muscle mass. The abductors may be inadequate. The sciatic nerve is closure than normal during surgery and one may need to release psoas with or without adductor tenotomy if abduction is <15°. Surgery is technically challenging and associated with a higher infection rate. Meticulous preoperative planning for acetabular position abductor moment arm restoration and leg length restoration.

Prophylatic measures for HO (NSAIDs). Preoperative CT can be helpful to determine adequacy of bone stock and the presence of a pseudoarthrosis.

Surgery[t]

Principles of surgical technique involve[12]:

- Identify and preserve hip abductor muscles
- Accurately identify the hip rotation center of the acetabulum
- Perform concentric reaming of the acetabulum to achieve medialisation and sizing of the component
- Avoidance cup placement in an excessive cephalic position
- Optimize leg length
- Restore ideal femoral offset to avoid impingement and instability

Patient positioning may be difficult, bone often osteoporotic, difficulty with locating native acetabulum (obturator foramen useful landmark). Exposure difficult because prior incisions, distortion anatomic planes, medialisation hip centre and soft-tissue contractures. Sciatic nerve often embedded in scar tissue. Line of neck resection identified, avoid cutting into greater trochanter or dividing the posterior acetabular wall. Identification orientation acetabulum can be difficult. Careful acetabular reaming to preserve anterior and posterior columns. Adductor tenotomy, iliopsoas muscle release and anterior capsulectomy ofter required to correct severe contractures.

Complications following conversion

Patients must be made aware of the higher rate of complications compared to primary THA:

- Deep infection 1.9–15.3% (higher in conversion of surgical fusion)
- Dislocation 1.7–6.25%
- Sciatic nerve palsy 1.8–13.4%. Leg length correction has to be limited (max. 4 cm)
- Femoral nerve palsy 3.6%

The gluteal muscles are atrophied and usually require the use of crutches for 3–6 months until the abductor function is strengthened. It may take 2 years to gain the full benefit of surgery. Normal abductor power may not be regained. The knee has a tendency for a valgus deformity if the hip is fused. If the hip is fused in a poor position consider corrective osteotomy first before arthrodesis. Patients whose hips were fused before puberty had less improvement in hip muscle function following THA because of underdevelopment of the greater trochanter. Leg length is improved in most patients but accurate restoration of leg length can prove difficult due to anatomic deformity and bone loss.

Results

Correction of LLD was an important element in overall patient satisfaction. Relief of back symptoms more pronounced than ipsilateral knee, fused hip or contralateral knee and hip. ROM is slightly less than after primary THA.

Risk factors for early failure include surgically fused hips, age <45 years, patients with two or more operations before surgery.

Hamadouche et al.[13] reviewed 45 consecutive conversion THA in 45 patients with ankylosed hips. Mean duration from initial hip ankylosis 35.7 years. The mean functional hip score of Merle d'Aubigné significantly improved from 11.3 points preoperatively to 16.5 points at last follow up.

Examination corner

Short case 1: Fused hip

Examining this hip:

- Scar over a stiff hip (surgical arthrodesis)
- Stiff leg gait
- Tredelenburg's: False positive, able to maintain abduction with no abduction function
- Measurement of real and apparent leg length discrepancy with tape measure
- Shortened leg with no hip movements
- Ensure you stabilise the pelvis while checking hip movements. Failure to do this will lead to incorrect diagnosis

Short case 2: Hip arthrodesis

'Examine this man's gait.'

- Difficult to describe as not the classic gait described of shortened stance phase and prolonged swing phase: A gait dysrhythmia due to a slower gait velocity with a shortened stride length, a greater than normal anterior pelvic tilt and lumbar lordosis. The increase in lumbar lordosis and change in pelvic tilt resulted in the mobile hip having a greater flexion/extension excursion than normal. Also real inequality in limb length (fused hip) and apparent leg length discrepancy (hip position in the frontal plane caused by adduction 0–5°) adversely affected walking performance. There was irregular forward progression with lateral motion of the head and trunk and a tendency to walk slower

'This was all above my head and I remember saying that he was walking with some difficulty with a gait suggestive of a stiff hip.'

[t] As a candidate you may not have seen this surgery. It is sensible to let the examiners know this, but you should know the principles of the operation. 'I haven't personally seen this type of surgery performed and have limited clinical experience of this complex operation, but some of the difficulties may include . . .'

Intermediate case 1: arthritic knee below a fused hip

Discussion usually centres on the pros and cons of whether to take down the fused hip first and perform a THA and then afterwards perform a TKA vs going ahead and performing a TKA above a fused hip.

TKA in patients with an ipsilateral hip fusion leads to a reduced ROM and the frequent need for manipulation under anaesthetic (MUA) because of stiffness. More over these artificial joints function under abnormal overstress leading to early failure.

Studies on the results of TKA in the presence of a fused hip have reported high complication rates with unpredictable outcome. Thus, the only exception to performing a TKA before converting the fused hip would be a patient with a satisfactorily positioned hip in whom abductor muscle function was questionable. In these patients, the results of THA are known to be inferior, with poor gait patterns and a decreased likelihood of adequate knee pain relief. If the hip is fused in a poor position and the patient has significant knee pain, the conversion THA is preferable because of the notably inferior results of a TKA in that setting.

Intermediate case 2: arthrodesis left hip (post-SUFE fixation with LLD), left THA and then periprosthetic fracture

Discussion

- Position of arthrodesis
- Work up of the infected hip
- Classification of periprosthetic fractures
- Management of periprosthetic fractures
- Taking down the arthrodesis
- TKA with hip arthrodesis
- Risk of low back pain
- Other joint arthrosis

Intermediate case 3: elderly patient in good health

History

- Index aetiology
- Indications for hip arthrodesis
- Type of ankylosis (spontaneous or surgical)
- Age since the ankylosis
- Previous complications (infection, venous thromboembolism (VTE), non-union, sciatic nerve injury)
- Indication for conversion THA
- Low back and ipsilateral knee pain
- Right hip arthrodesis post tuberculosis aged 15
- Intra-articular arthrodesis

Examination

- Location of previous incisions – postoperative scars over the anterolateral aspect hip and right iliac crest
- Measurement of leg lengths
- Demonstration of gait
- Demonstration of Trendelenburg's test (false negative)
- Perform Thomas' test
- No pain on attempted hip movement
- Position of fusion: 35° flexion, 5° adduction and neutral rotation
- In addition, femoral and sciatic nerve function, vascular status, pelvic obliquity, kyphoscoliosis lumbosacral spine and ROM and stability of the contralateral hip and knee and ipsilateral knee. Functional integrity and strength of hip abductor muscles

Discussion

- Why convert the arthrodesis to arthroplasty?
- Is hyperlordosis the cause of low back pain?
- Should antituberculosis treatment be used preoperatively? If so for how long?
- What are the side effects of antituberculosis drug treatment?
- What consent issues are there preoperatively?
- Neurovascular problems, in particular the need to expose the sciatic nerve or not
- Hip instability
- Infection
- Results of conversion
- Preoperative planning of the arthroplasty
- Plain films, CT scan
- Implant considerations relevant to a stable hip

Intermediate case 4: Older patient with fibrous ankylosis of the hip following SUFE and previous proximal femoral osteotomy

Discussion on:

- SUFE
- Osteotomies
- Surgical approaches to the hip
- Demonstration of the flexor contracture of the hip
- THA
- Surgical approaches
- Management of difficulties in this case

Intermediate case 5: young female who had presented with DDH aged 4

The patient had open reduction, Salter's osteotomy and a femoral osteotomy. She developed AVN and growth arrest of capital femoral epiphysis. She had an arthrodesis aged 14 complicated by sciatic nerve palsy.

Discussion points

- Diagnosis?
- What operations has she had?
- Measurement of the centre-edge angle?
- How would you do an arthrodesis of the hip?
- What do you think of the position of this patient's arthrodesis?
- Why did she develop a sciatic nerve palsy
- Options for further management
- Outcome following revision to THA (taking down the hip)

Short case 3: arthrodesis of the hip

EXAMINER: Examine this man's hip.

CANDIDATE: I mentioned that I would start by examining the patient's gait. The examiner said ignore the gait and, therefore,

I said I would like to perform a Trendelenburg's test. Again, the examiners said ignore Trendelenburg's testing. They wanted me to go straight on to hip movements and deformities. There were absolutely no movements in the hip and there was a healed scar on the lateral aspect. I told the examiners the diagnosis could be an arthrodesis of the hip. They agreed with this and showed me the x-ray to confirm it.

The examiners asked me what I would do about management. I said I would ask the patient if he had any pain with the hip. The patient said that he had no symptoms at the moment so I said nothing should be done with the arthrodesis. The examiners were happy with that. They gave me a scenario: If the patient came to see me with pain, what management would I offer? I said that we could convert an arthrodesis of the hip into THA but that the patient should be warned about failure and also weak abductors and neurovascular injury. The examiners were again happy with this answer. By that time 5 minutes were up and the bell went.

Before answering the scenario question I should have perhaps clarified with the examiners that the pain was definitely arising from the hip and not referred from elsewhere.

Primary OA hip

Introduction

Most likely an intermediate case but equally a candidate could be asked to demonstrate the Trendelenburg's test, Thomas' test or ROM in a short case. Examiners view a straightforward intermediate case of primary hip OA as an easy case. Be professional and thorough in your presentation. The expectations from examining a primary hip OA case are high and it is very easy to loose marks. It is a condition that candidates will see on a day-to-day basis in clinic and they would be expected to know it inside out for an exit exam.

The examiners have a very low threshold for any minor mistakes or errors made. This is a high stakes exit exam and if you miss subtle details out of the history, do not demonstrate Trendelenburg's test particularly well, botch-up the Thomas' test or say something 'not quite correct' in the discussion you well end up scoring poorly[u]. In comparison, a very difficult intermediate case may mean that the examiners are more likely to be forgiving if you should make the odd mistake during the history and examination.

History

Pain: The predominant and most important symptom. How long have you had pain? Where is the pain felt – pain from an OA hip is classically felt in the groin, around the greater trochanter and occasionally in the buttock (suspect either the lumbar spine or sacroiliac (SI) joints). Is the pain getting worse or staying the same? Does the pain radiate anywhere – radiation to the front of the thigh and knee commonly occurs and at times pain may present solely in the knee. Was the pain insidious or sudden in onset – OA or AVN. Aggravating and relieving factors, etc.

Quality of life/limitation: How the symptoms are affecting the patient such as daily activitites (dressing up, putting on shoes shoes, cutting toenails).

Decreased walking distance: How far can you walk before you have to stop – sometimes patients do not have a restricted walking distance but get pain after 10 minutes or so. Does the pain stop you from walking any further or is it shortness of breath or chest pain – concerns about fitness for surgery.

Sleep disturbance: Does the pain stop you sleeping at night? How many times do you have to get up in the night because of the pain? Night pain can be particularly distressing to patients and is an important and strong indication for surgery.

Analgesia: What painkillers are you taking and how often are you taking them. When was the last time you took a painkiller?

Limp: Limp may be noticed early, but more often than not comes on later than pain or stiffness. It can be due to a variety of cause including pain, muscle weakness and stiffness (capsular contractions).

Stiffness: Inability to put shoes, socks or stockings on, inability to cut toenails, get in and out of a bath, in and out of a car. Stiffness and limp are relative indications for surgery and should not be regarded as the sole indication for surgery in the absence of pain.

Drug history and past medical history: Fitness for surgery. Need for anaesthetic assessment.

Miscellaneous and social history: Do you use a walking stick – for how long – which hand? Previous treatments tried and success. Smoking and alcohol. Do not forget the social history – do you have stairs in your house, home help, meals on wheels, etc. Look for any walking aids, etc, next to the patient.

Examination

Make sure the pain is arising from the hip joint – NOT back pain; – NOT knee pain. Moving knee in the plane of knee (pain = knee). Move knee as pendulum (pain = hip). Be suspicious if back movements reproduce pain. Occasionally patients are referred for THA with arthritis of the hip but examination and observation of gait will show that the patient is more limited by peripheral vascular disease, peripheral neuropathy or Parkinson's disease than by the arthritic hip. Under these circumstances THA is relatively contraindicated.

[u] This could be a one gaff mistake in the discussion (analgesia pain ladder) or more subtle, such as the candidate giving the impression they have read about the subject in a book but haven't come across it much practically in the OP clinic.

Discussion

Discussion would usually start with a description of the patient's radiographs. Be able to describe typical features of hip OA.

This would lead onto a discussion of conservative management of hip OA. What painkillers to use, the role of physiotherapy, when to list for an intra-articular steroid injection. Be fairly neutral with this and don't say anything too controversial[v].

EXAMINER: How would you consent the patient? What would you tell them about the risks of the procedure?

This may lead on to what approach 'YOU YOURSELF' would use for a primary THA possibly followed by the advantages and disadvantages of two of three other common surgical approaches used. National Joint Registry (NJR) data has shown a significant increase in the use of the posterior approach in THA along with use of larger diameter heads and a lower dislocation rate[14].

EXAMINER: What type of THA would you use and why?[w]

This may lead to potential discussion of cement vs uncemented hip designs. There has been a recent trend towards performing more cemented THA as NJR data suggests a lower early revision rate using cement and lower cost.

The viva may then move on to long-term published results of THA in the literature and end with survival analysis curves or how to set up a study to assess the long-term outcome of a particular THA.

Three main factors influence the outcome and survival time of a primary THA:

1. The surgeon's skill and experience
2. The implant design and method of fixation
3. Patient characteristics such as sex, age, weight, underlying disease and activity level

It is important to know current NICE guidelines regarding primary THA and have read the British Hip Society hip replacement booklet[15].

Candidates should know about the Orthopaedic Data Evaluation Panel (ODEP) ratings. These are the criteria for product categorisation of prostheses for primary THA against NICE benchmarks:

- Pre-entry benchmark (products commercially available that are involved in post-market clinical follow-up studies but have failed to meet NICE's 3-year entry benchmark)
- Entry benchmark (after 3, 5 and 7 years: Level A – Acceptable evidence; level B – Weak evidence)
- Full benchmark (10 years: Level A – Strong evidence; level B – Reasonable evidence; level C – Weak evidence)

For each year, there is a level for unacceptable evidence, where products should only be used as part of a clinical trial.

- Discussion of hip registers

  Discussion may cover funding and maintenance of the register, performance of implants, revision rates, stratification of variables (age, indications, gender, etc)

Examination corner

Intermediate case 1: A 56-year-old woman with a painful right hip

History

As per hip OA

'The patient wasn't the best historian. I was first candidate to examine in the morning.'

Clinical examination

Shoulders level, and symmetrical stance. No pelvic tilt with ASIS level and no LLD.
Mild antalgic gait not marked, positive Trendelenburgs test. Thomas' test. No FFD hip.
Marked restriction of internal and external rotation in flexion with only a jog of movement present.
Abduction restricted to 30°, adduction similarly restricted to 20°; both were painful.
Mentioned neurovascular and spine examination.
I was then asked to measure for true leg length, which as expected was normal. I am not sure if the examiners were finding their feet being the first case and hadn't quizzed me in enough detail on the earlier positive finding and had to find something for me to do to use up the remaining time.

Discussion

The approach I would use (posterior) and asked to describe in detail.
Implant – I played safe and suggested using a cemented Exeter hip replacement. I was lucky, as the examiners didn't push me on this choice as I had thought about an uncemented THA with ceramic on ceramic bearing surface.
Consent – quite detailed grilling of potential complications and incidence compared to the previous question.
Benefits of the NJR.

[v] 'Physiotherapy is generally a waste of time with advanced OA hip.' Avoid any sweeping generalized controversial statements. About 1 in every 10 candidates, whether through nerves or whatever, will say something that in hindsight is really not very sensible or helpful for them.

[w] CANDIDATE: I use the Exeter prosthesis because I am most familiar with this design from my training. The instrumentation is relatively straightforward; the neck cut is not critical. Most importantly it has successful long-term peer-reviewed published results. The collarless, highly polished, double taper stem allows controlled insertion. . . etc. The Exeter femoral component has a 94–98% 10-year survival rate and a 10A Orthopaedic Data Evaluation Panel (ODEP) rating.

Intermediate case 2: Primary OA of the hip

'The history and examination were relatively straightforward. In the discussion section I was asked virtually everything possible there was to know about THA in great detail.'

Slipped upper femoral epiphysis

History

'Sixty-five-year-old man. He had a severe left SUFE in-situ fixation as a 14-year-old boy.'

'His main complaint now is of left hip stiffness. Limited activities of daily living. He has difficulty putting his shoes and socks on and getting in and out of the bath. His walking distance is not restricted but he needs to take his time getting from place to place. Pain is not a significant feature and there is no sleep disturbance and he is on no regular medication. He is otherwise fit and healthy.'

Clinical examination

'On general examination the patient is of average height and build. He looks well for his years. On examination standing there is a well-healed left lateral hip scar present from his previous hip surgery. He stands up straight with a flexion attitude of his right leg, which on straightening up the pelvis revealed a left LLD. A mild external rotation deformity of the affected leg was present. There is marked left thigh muscle wastage. He walked with an antalgic short left leg gait. He was Trendelberg's positive on the left side.'

'Suggest to the examiners using a block test to assess LLD prior to supine examination.'

Supine

'Thomas' test demostrated a FFD of 15°. Measure apparent leg length. Square pelvis to measure true length. The ASIS on the left side was elevated, squaring the pelvis (bringing the ASIS to same level unmasked a fixed adduction contracture of the left hip). Measurement of leg lengths using a tape measure confirmed 2 cm of real shortening. Galleazzi's test revealed this to be in the femur. Digital Bryant's test suggested the shortening was above the trochanter. If there is fixed flexion deformity , do not mention extension at the hip as this is absent. Flexion was from 15° to 90°, with the leg tending to go into fixed external rotation as it was flexed up (axis deviation). There was virtually no internal rotation (IR) or external rotation (ER) at 90° flexion. Abduction was limited to 30° and similarly adduction limited to 20°. Check active and then follow on with passive movement. Pain was present at the extremes of movement. The leg was neurovascularly intact and examination of the spine, opposite hip and both knees were unremarkable.'

Discussion

- AP radiographs (Figure 11.14)
- Aetiological factors of slipped upper femoral epiphysis (SUFE)

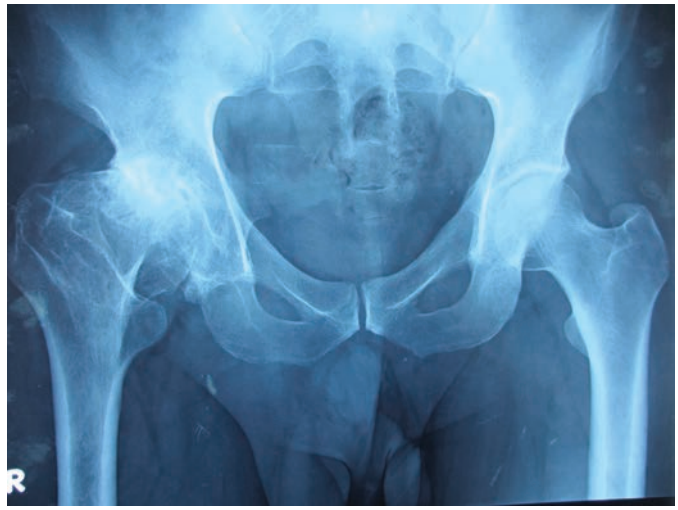

Figure 11.14 Severe SCFE disease right hip with coexisting OA

- Dunn and Loder classification of SUFE
- Radiological finding in SUFE
- Treatment options of severe slips
- Various osteotomies

Management of this particular case

In the absence of significant pain this patient is not a candidate for THR, as ROM is rarely significantly improved. Advanced changes of OA are too severe for osteotomy. Therefore, continue with conservative treatment at present.

Technical difficulties of performing a THA in SUFE

An anteverted femoral neck may mislead the surgeon during stem insertion and lead to component mal-positioning. There could be problems with metalwork removal – Have special instruments available. Consider a 2-stage procedure – Metalwork removal first followed by THA 3 months later.

Young patient with painful hip post SCFE[16]

Slipped capital femoral epiphysis (SCFE) deformity often results in significant femoroacetabular impingement (FAI), which may lead to the development of OA at an early age. Posterior and inferior slippage of the epiphysis results in a metaphyseal bump – A Cam lesion that leads to FAI. Post-SCFE cartilage and/or labral damage develops in patients with symptomatic mild to moderate SCFE deformity.

History

Pain often with activities that force the hip into flexion
Usually groin pain (83%) but also can present with lateral hip, thigh, buttock and low back pain
Sitting may be difficult, flexing only with marked external rotation

Pain is usually activity related-running, pivoting and walking
Mechanical symptoms such as popping, snapping, catching, locking and/or instability present in about two-thirds of patients

Clinical examination

Gait: Patients often walk with out-toeing of the affected leg because the slip results in external rotation of that leg
Trendelenburg's test: Often positive
Evaluation of ROM: Internal rotation, flexion and abduction is often limited because of the position of the epiphysis. External rotation is often increased with SCFE. Flexion is always reduced whilst abduction is usually reduced
Provocative hip tests: Usually positive anterior impingement sign (pain on forced flexion, adduction and internal rotation). The metaphyseal bump impinges within the acetabulum or against the rim of the acetabulum
Leg lengths: Often the affected leg is shortened
Test for hip abductor muscle strength: Usually reduced. Test side lying

Discussion

Imaging:

AP pelvis and frog-leg lateral. This assesses external rotation, metaphyseal bump, prominence impingement on the acetabulum and varus or valgus deformity
False positive view to obtain better view of metaphyseal bump and anterior coverage acetabulum
CT to visualise SCFE deformity and better plan surgical management
MRI. Metal artefact if screw still in situ

Management

Arthroscopic femoral neck osteochondroplasty
Limited open osteochondroplasty
Moderate or severe slips may require redirectional flexion intertrochanteric osteotomy
THA

Examination corner

Short case 1: SCFE

A middle-aged man in his 40s. Huge protuberant abdomen who struggled to do anything much during the examination.
Asked to examine his hip.
I commented that he had an external rotatory deformity of the leg with shortening, muscle wasting and a lateral scar. He had an antalgic gait.
I was put off by the fact he was overweight and in a lot of pain struggling when walking.
I performed Trendelenburg's test but wasn't particularly slick with it.
I examined hip ROM which was painful and limited.'
I didn't comment on the leg going into external rotation when flexed and he was in quite a lot of pain with any hip movement.
They asked me whether I would normally perform Thomas' test when examining a hip. Instead of saying that the patient was in a lot of pain and it wasn't appropriate, I said yes I would and then went on to perform the test. However, the examiners stopped me after I started flexing up the opposite hip as even this seemed to cause him severe pain.
I was asked about the possible diagnosis and I mentioned old slipped SCFE.
I really didn't do very well with a straightforward case, it didn't seem to flow very well at all.

(5 – fail)

Rheumatoid patient with hip disease

Background

Despite numerous medical advances in the treatment of RA, severe involvement of the hips is common. Fortunately, THA provides excellent reliable relief of pain and functional improvement. Between 6% and 15% of THA are carried out for RA. The average age of onset of rheumatoid disease is 55 and the average patient has had hip symptoms for 4 years.

In general, forefoot deformity should be the first corrected to ensure they are capable of comfortable weight-bearing and reduce sources of infection at a later date.

Thereafter, the hip should take priority over the knee and hindfoot. Knee pain referred from the hip is abolished and the restoration of knee anatomy and ligament balance at subsequent TKA is easier and more reliable when the hip above is mobile. Rehabilitation of TKA is difficult with a stiff, painful or deformed hip. The hindfoot should be corrected last as correction of hip and knee deformities may alter the dynamic position of the hindfoot.

There are concerns regarding:

- Polysystemic/multiple joint nature of the disease
- Polypharmacy as patients often take a variety of medication that can affect surgery
- Immunosupression either from the disease or by their treatments
- Difficulties with rehabilitation
- Check for neck symptoms (cervical instability)
- Enquire if the patient is on any biological therapy that may have to be stopped prior to surgery

Memorandum

History

Insidious onset groin, buttock or thigh pain. Subject to acute flare-ups'

Examination

Shake hands and see skin condition – Dry, eczema, psoriasis scars, etc.

> 'On general inspection the patient has features of extensive rheumatoid disease affecting many joints. The buttock and thigh are markedly wasted with the limb held in external rotation and fixed flexion. The skin overlying the joint may appear shiny, thin and atrophic with evidence of spontaneous bruising. All movements of the hip joint are restricted and painful. Hip flexion and adduction contractures of various degrees may be present. It is important to examine the upper extremities to evaluate the patient's ability to use walking aids. The knees, ankles and feet should be examined for arthritic involvement. Look for any walking aids, footwear and any braces.'

Beware of the patient in a wheel chair.

Some patients with severe multiple lower extremity involvement may be confined to a wheelchair.

- Can they walk?
- How?
- Do they use a stick?
- When did they last walk?
- Make the patient walk if possible

Radiographs

Radiographic findings in the rheumatoid hip can often be subtle. Osteopenia is seen in most cases. Typically there is concentric joint space narrowing due to generalized loss of articular cartilage without evidence of osteophytes or cyst formation. With progression of the disease, medial and/or superior migration of the femoral head with protrusio deformity occurs especially if steroids are used.

Management

Often rheumatoid patients are generally disabled having various degrees of osteopenia, skin fragility, vasculitis and poor musculature. It is important to ensure the patient is in as fit a state as possible for surgery. Synovitis should be as well controlled as possible and no chest, urinary, dental or skin sepsis should be present. Anaesthetic risks such as neck instabilty should be excluded. Medical management of RA may be discussed. Biological medications generally have to be stopped. Methotrexate should be continued as it has been shown not to increase the rate of infection in THA. Stopping methotrexate can increase the risk of associated disease flare up and associated complications.

Grennan et al.[17] in 2001 in the *Annals of Rheumatoid Disease*, in a prospective randomised control trial of 388 patients with RA, demonstrated no increased infection risk from elective orthopaedic surgery. They recommended that methotrexate therapy should not be stopped before surgery[x,y,18].

In general, it is recommended that biological disease modifying anti-rheumatoid drugs (DMARDs) should be withheld before and after surgery.

A get-out clause, if the examiners are cornering you, would be mentioning a multidisciplinary approach to the rheumatoid patient. Medication management requires a risk–benefit discussion between patient, surgeon and rheumatologist. A local hospital protocol may have been developed for guidance and it would be worth mentioning this as well[z].

Management options include:

- Intra-articular injection of steroid/local anaesthetic
- Open synovectomy – Not popular, risk of AVN femoral head
- Arthroscopic partial synovectomy – Becoming more widely practiced, may have a role in the young rheumatoid patient
- Osteotomy – contraindicated as the disease is generalized throughout the joint
- Arthrodesis – contraindicated, requires good functioning not diseased neighbouring joints
- THA

Operative issues

Careful position on the operating table is vital due to poor skin and other painful joints, padding of all pressure areas. Care must be taken with the neck during patient transfer and positioning. Temporomandibular disease may make intubation difficult. Bone stock is often poor being soft and osteoporotic, so great care is required during dislocation and relocation of the hip to avoid fracture of the femur. Dislocation may be difficult if protrusio is present. Care is needed not to bruise the skin and soft tissues of the leg. The femoral head and neck may be partially absent. Due to the reported high rates of

[x] We suggest when quoting papers a so called 'four-corner approach' should be used. Author, institution, year of publication and journal. If you remember all four corners this is an excellent solid use of evidence. Three corners are still good whilst two corners are OK-ish and one corner is not so good.

[y] There was a 10-year follow up of the Grennan paper in which they adhere to their original advice. A lot of patients, however, were lost to follow up – See reference 17.

[z] There are several local hospital policy guidelines that usually have been developed. These could include antibiotic use in open fractures or elective orthopaedic surgery, massive blood transfusion or the septic joint. 'We have a hospital protocol in place based on NICE guidelines. I would refer to this protocol to guide to my treatment decision.' These are guidelines only to help with treatment decisions and perhaps not to be absolutely rigidly adhered to. Have an answer preplanned in case the examiner is dubious about their usefulness and starts challenging you about using them or starts having a rant about the over importance placed on NICE guidelines.

non-union in RA the transtrochanteric approach should generally be avoided. Reaming can compromise the edges of the acetabulum and the acetabular bone is soft and easily penetrated. If protrusio acetabuli present (20–40%) bone grafting of the medial wall (with a bulk autograft or packed reamings from the femoral head) and in severe cases mesh and shell re-inforcement may be required.

The rate of deep infection is approximately double (2.6) that of osteoarthritis. Late infection is more common in rheumatoid disease, possibly related to immune suppression or other sources of infection. Fitzgerald et al. reported a deep infection after hip replacement in 3.1% of 223 RA patients[19].

There is some debate as to whether the incidence of thromboembolism is reduced in rheumatoid disease due to a mild coagulopathy. Evidence in the literature is sparse. HO is less common in RA. The dislocation rate has been reported as more common but there is little evidence for this.

Implants should in general be cemented as poor bone stock may not support cementless fixation. Uncemented components have not been widely used in the treatment of RA. Orthopaedic surgeons have been concerned that the osteopenia, contractures and bony deformity often seen in RA would make it difficult to obtain safely and reliably the initial stability necessary for bony ingrowth. In addition, there has been concerns relating to the how much bony ingrowth will occur in the presence of systemic inflammatory disease and the effects of antirheumatic medications. Cemented implants may be a wiser move but there is little evidence in the literature to support this opinion.

Mixed results have been reported for the survival of primary THA compared to OA. In the Swedish Hip Registry[aa] an increased rate of revision of acetabular cups in rheumatoid patients both young and old was noted. Other studies have found no difference in survival between RA and OA.

AVN of the hip

AVN of the hip is an ideal intermediate case. There would be emphasis on history taking, differential diagnosis and treatment planning. The patient typically would be young or middle aged and present with early AVN and worsening hip pain. Another possible scenario would be progression of AVN with worsening hip pain following unsuccessful core decompression. In a short case you may be asked to demonstrate fairly specific hard clinical signs such as Trendelenburg test, Thomas' test or a restricted painful range of hip movement rather than go through a complete hip examination.

History

As with any diagnosis, the history is critical. A high index of suspicion is essential especially if the patient has one of the atraumatic conditions associated with AVN.

The standard hip questions should be asked. In addition, the history should include a search for possible aetiological factors. Inquire about steroid use, alcohol intake and history of previous hip trauma, deep sea diving and any blood disorders.

The clinical presentation is of gradual onset of intermittent groin and/or thigh pain. The pain is typically deep seated; throbbing, felt at night and is unremitting and similar to bone tumour pain. Pain is usually worse with ambulation, although some patients progress to have pain at rest. Very occasionally the pain presents abruptly. A few patients may remain relatively asymptomatic despite radiographic evidence of advanced progression of the disease.

Examination

The clinical findings on examination can be unremarkable or can include pain on internal rotation of the hip, a decreased range of motion, an antalgic gait and clicking of the hip when the necrotic fragment has collapsed. Pain with internal rotation of the hip and a limited range of hip motion are often signs that the femoral head has already collapsed.

Memorandum

On general inspection the patient looks well for his years. He is standing up straight with his pelvis level and taking weight equally through both legs. Possible left gluteal and thigh muscle wasting.

Examination of gait revealed that he walked with an antalgic gait. Trendelenburg's testing of his lower limbs revealed a delayed positive response on the left side and negative Trendelenburg test on the right side[bb].

Formal measurement of leg lengths supine revealed 2 cm of true shortening of the left leg. Galleazzi's test confirmed that the shortening was arising from the femur. Bryant's triangle testing suggested that the shortening was above the trochanter. Movements of the left hip were painful and grossly restricted particularly abduction and internal rotation in flexion. The left hip demonstrated almost full internal rotation in extension but with the hip flexed it was grossly restricted[cc]. The left hip had a tendency to twist into fixed external rotation during passive hip flexion.

[aa] www.shpr.se/Libraries/Documents/AnnualReport_2013-04-1_1.sflb.ashx

[bb] Some surgeons doubt the significance of a delayed Trendelenburg's test, citing the fact that even a normal patient's pelvis will dip down after 30 seconds.

[cc] If internal rotation is full with the hip extended but restricted in flexion this suggests pathology in the anterosuperior portion of the femoral head, which is probably AVN – the so called 'sectorial sign'.

Examination corner

Intermediate case 1

Discussion

How do you assess the rheumatoid patient in general for THA surgery?
What about the cervical spine? When do you order new x-rays?
What about methotrexate? Would you stop it before surgery in order to decrease the risk of infection

Discussion

Discussion will probably begin by reviewing hip radiographs (Figure 11.15). A candidate would be expected to describe the typical radiographic findings of AVN (can be very subtle) and then stage the disease. In the current FRCS (Tr & Orth) exam classification systems are not asked for directly by the examiners. One may, however, benefit by knowing various radiological and MRI classifications and volunteering this additional knowledge to score extra points. Discuss classifications only if you are sure about them. The examiners may also have an MRI scan of both hips available and there are several possibly lines of questions which can emanate from this (Figure 11.16).

The aetiology of AVN will generally be discussed and, if you are doing well, the latest theories of pathogenesis for AVN. Management options of AVN is probably the pass/fail area. A candidate will need to decide whether to simply go through a list of possible options or be more specific in his/her management plan for his/her particular case[dd]. Recent advances such as stem cells and bisphosphonates could be brought into the conversation.

The discussion can lead to:

- The advantages and disadvantages of performing either a cemented or uncemented THA in a young patient
- How you would perform a THA? What approach would you use and why? What implant would you use and why? What are the long-term results of this implant? Do you know of any published results of this implant?
- Discuss the current NJR data on THA for AVN
- What are the results like for THA in AVN compared to a normal standard group with OA?

Short case 1: psoriatic arthropathy with AVN secondary to steroid use

EXAMINER: Examine this lady's hips:

- Bilateral AVN of the hips with scars on each side of the proximal thigh for core decompression

Shown AP pelvis radiograph of patient and asked to pass comment:

- Radiographic features of AVN
- Discussion on Ficat and Steinberg's classification system

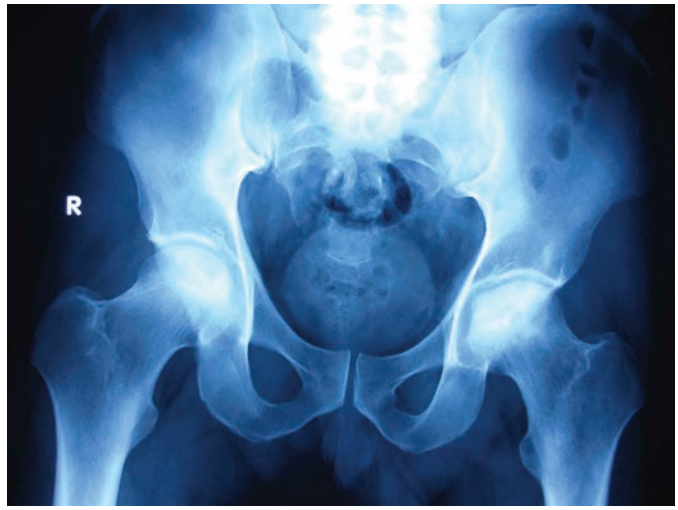

Figure 11.15 Bilateral osteonecrosis hips

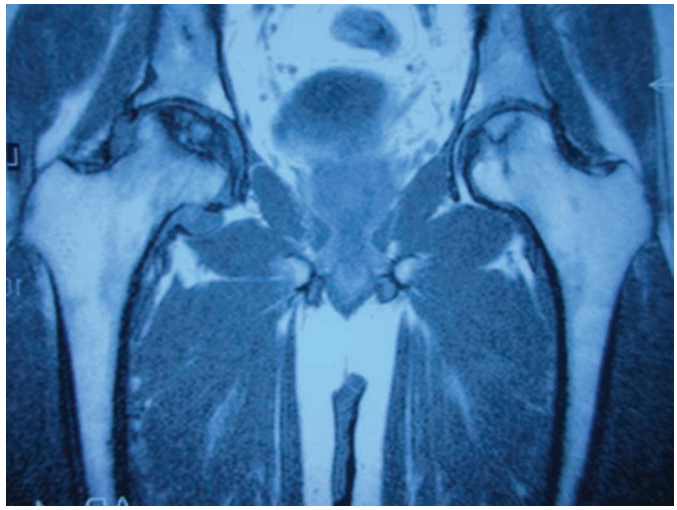

Figure 11.16 Coronal T1-weighted MRI image of bilateral ON of differing age. The low signal intensity in the superior weight-bearing area of the right femoral head is typical for osteonecrosis

Short case 2: Middle-age man with pain/limited hip movements

EXAMINER: This man was involved in a RTA; just examine the range of movements of the right hip.

CANDIDATE: I examined his hip movements and found a restricted and painful range of movement in his right hip,

[dd] If you are asked about your management plan it is preferable to discuss your own preferred treatment choice for the case rather than mentioning a whole list of management options.

[ee] Mechanism of action (MOA) probably from altered fat metabolism. Steroids cause osteoblastic stem cells to become fat cells (apidogenesis). Existing marrow fat cells undergo hyperplasia and hypertrophy. Capillary occlusion and intraosseous hypertension results.

particularly abduction (15°) and internal rotation in flexion (virtually nil). I mentioned the large proximal thigh scar present. For some reason, and I have no idea why , I asked the patient two or three questions about his hip pain. 'How severe was his hip pain? How far was his walking distance? Did it keep him awake at night?' The examiner then led me through his radiographs.

EXAMINER: These are his radiographs. He had a severe posterior pelvic fracture, which has been fixed with pelvic reconstruction plates. The diagnosis is obvious looking at the femoral head. He has developed AVN.

CANDIDATE: *(It wasn't that obvious and I may have struggled to get to it.)*

EXAMINER: What would you do for the patient?

CANDIDATE: His pain is not too severe at present and he seems to be coping reasonably well with things. He can walk up to 2 miles without too much difficulty. He isn't kept awake at night with this pain. I wouldn't do anything with him at the moment. I would review him regularly in the clinic, and if his symptoms deteriorated significantly I would offer him a total hip replacement.

EXAMINER: Yes, you are quite right. There is no need for any surgery at present as his symptoms are minimal.

CANDIDATE'S COMMENT: If your luck is with you it is with you in incredibly large amounts. I do not know why I started to ask the patient questions. The examiners certainly didn't ask me to but it gave me the information needed to answer the question about his current management plan correctly. The examiner assumed that I had picked up on the fact he had developed AVN.

Intermediate case 1: Male aged about 60 years with history of Caisson's disease

- Painful left hip – Moderate OA secondary to AVN
- Right THA
- Moderate bilateral varus OA knees
- Most of the clinical findings above were demonstrated to the examiners. In particular they were interested in the difference between true and apparent shortening of the right leg

Discussion

The discussion focused mainly on the differential diagnosis, pathology, aetiology, classification, grading and management of AVN (detailed discussion on core decompression).

Results of THA for AVN was also discussed.

Intermediate case 2: 14-year-old boy with AVN and collapse with ankylosis of the left hip following pinning of severe SUFE

Clinical findings included

- Gluteal and buttock muscle wastage
- Shortening of the left leg
- Short leg antalgic gait and stiff hip gait. There was a subtle increased motion of the pelvis on the lumbar spine during swing. The classic finding in a stiff hip gait of rotating the pelvis and swinging the leg in a circular fashion was markedly absent
- Trendelenburg's testing was false negative. The patient was able to maintain abduction with an ankylosed hip
- Demonstration of Thomas' test. There was a fixed flexion deformity of 20°
- 'Demonstration of ROM, but not done particularly well as I didn't stabilise the pelvis and significantly overestimated hip movement'
- 'Measurement of true and apparent leg lengths using tape measure, asked by the examiners to describe what I was doing as I went along. Grilled by the examiners to explain exactly what I meant by true and apparent shortening of the leg'

Discussion

Radiographic features of AVN: Cysts, sclerosis and crescent sign, etc.

General discussion about potential management options without really deciding anything at all.

Treatment of AVN following SCFE is difficult and often unrewarding. Articulated hip distraction (arthrodiastasis) used in adolescents with AVN may reduce pain and limitation in daily activities. However, it is less effective for AVN second to SCFE and is not the final solution to AVN.

Salvage procedures include proximal femoral osteotomy and shelf acetabuloplasty. Arthrodesis for advanced deformity with arthroplasty at a later date when the patient is older is another potential option.

Investigate by examination under anaesthesia and dynamic arthrography. Under anaesthesia fixed deformities can be assessed, the size and shape of the femoral head and joint congruity determined, presence of hinged abduction and most congruent position femoral head within the acetabulum evaluated.

Intermediate case 3: AVN

History

'A 62-year-old woman with a history of left hip pain. I was asked to take a history. I started off by asking her age, occupation and presenting complaint. She told me she has pain in the left hip and also difficulty walking for the past 6 months. I asked her in detail about her pain – the site, variation, character, aggravating and relieving factors, and also enquired about the limping. I also asked whether she had any significant past medical history and she mentioned she suffered from persistent lupus erythematosus and had been on long-term oral steroids for many years. I more or less got a diagnosis from the history itself. I went on to her medications, social and family history. At this point the

[ee] Mechanism of action (MOA) probably from altered fat metabolism. Steroids cause osteoblastic stem cells to become fat cells (apidogenesis). Existing marrow fat cells undergo hyperplasia and hypertrophy. Capillary occlusion and intraosseous hypertension results.

examiner stopped me and asked me to carry on with the examination.'

Examination

'I told the examiners that I would like to see her walking and they agreed with me. The patient took a few steps but it was very painful so I mentioned to the examiners that she had an antalgic gait. The examiners agreed with this. I continued on to Trendelenburg's test. The examiners helped me with this because the lady needed a lot of support as she was in a lot of pain. I carried on to inspection and LLD. The examiners cut me short here and advised me that there was no LLD and to proceed with the deformities. I proceeded to examine for a flexion deformity. The examiners also helped me out with the Thomas' test. I then began to look for an abduction and adduction deformity but they said not to bother and asked me to demonstrate hip rotations. I mentioned that i would also want to check and examine the knee and spine for any evidence of disease. The examiners asked me to ignore this.'

Discussion

'We then proceeded on to the discussion section. They asked me what the diagnosis was. I told them it could be AVN of the femoral head with secondary osteoarthritis and they asked me why I said this. I told them she has a past medical history of systemic lupus erythematosus and she has been on steroids. They agreed with this and asked me how AVN of the femoral head develops in steroid therapy.'

'I explained that steroids cause abnormally elevated lipid levels which lead to microemboli and endothelial cell changes resulting in venous stasis, increased intraosseous pressure and bone necrosis[ee]. I also told them that she has systemic lupus erythematosus so there was a possibility that she has minor coagulation defects in addition to the steroid intake. They were happy with that.'

'They asked me about additional sites that can be affected with AVN and I mentioned femoral condyles, tibial plateau, talus and humeral head.'

The infected THA

Introduction

This topic is generally not well-suited for use as a short case. As an intermediate case you will not get a acutely infected hip draining pus in the examination hall. More likely the hip will be painful due to either a chronic low-grade infection or have been revised because of previous infection and had complications postoperatively such as a dislocation or periprosthetic fracture. The best way to deal with this type of case is to go back to basics and keep things simple. Think in terms of how you would normally approach this type of case if you had seen it in the outpatient clinic.

History[ff]

History of the original index THA:

Where was the operation performed, who performed the surgery[gg] and how long was follow up for?

How long has the hip been painful?

Was the hip always painful after surgery (low grade infection) or was there a pain-free interval (aseptic loosening, late onset infection)?

Is the pain getting worse?

There is an increased risk of deep prosthetic infection as a result of delayed wound healing or large haematoma formation. Therefore, specific inquiry should be made about wound drainage, persistent fever, prolonged antibiotic administration or delayed hospital discharge. Has the patient had a recent urine or chest infection? Reduction in walking distance and/or are walking aids now necessary. Pain at night or at rest suggests infection (or tumour).

Mention that you would obtain old records to check for surgical approach, implants used and postoperative review records.

Examination

'On examination this elderly male gentleman has difficulty standing upright unaided. There is an old well-healed left lateral hip scar. The surrounding skin and soft tissues appear normal. There is no evidence of a discharging sinus in the wound. There is marked left thigh and gluteal muscle wastage.'

'Examining of gait revealed that it was antalgic with a marked Trendelenburg's positive test on the left side but negative on the right side. Formal measurement of leg lengths with a measuring tape revealed 0.5 cm true shortening in the left leg. Thomas' test failed to reveal any fixed flexion deformity of the left hip. Examination of the left hip movement demonstrated a global decreased range of movement, which was painful but not stiff.'

Investigations

Radiographs

Suggest AP and lateral radiographs and look for radiographic features suggestive of infection such as endosteal scalloping, multilamellar periosteal new bone formation in

[ee] Mechanism of action (MOA) probably from altered fat metabolism. Steroids cause osteoblastic stem cells to become fat cells (apidogenesis). Existing marrow fat cells undergo hyperplasia and hypertrophy. Capillary occlusion and intraosseous hypertension results.

[ff] History is quite similar to the painful hip requiring revision but don't forget to focus more heavily on the infection questions.

[gg] Perhaps relevant in real life much less so for the exam.

the femur and rapidly progressing llosening or osteolysis. It would be wise to compare with old radiographs.

Bloods

FBC, ESR, CRP and IL-6

Bone scan

In practical terms, a bone scan isn't particularly helpful in differentiating between aseptic and septic loosening. The value of bone scan is limited in the early postoperative years. A radioactive-labelled WBC scan (leucocyte scan) is more sensitive and specific but its value is still somewhat limited.

Aspiration hip

In theatre, sterile conditions, blood culture bottles ± arthrogram. It is important not to pick up skin flora during the aspiration. Any antibiotics must be stopped 2 weeks or so beforehand as they may affect the results of the aspirate giving negative values in the presence of ongoing infection.

Management

The FRCS Orth exam is much more than just presenting facts to the examiners. Just as important are the linking of words and sentences, which connect these facts.

> 'My management would be directed towards trying to identify a cause for this painful hip. There are numerous possibilities; the most common causes would be infection, aseptic loosening or referred pain from elsewhere. Other causes could include impingement, instability or fracture. Features suggestive of infection would include . . . It is potentially a very difficult problem to treat especially if deep infection is present in the medullary canal. There are several ways to treat deep prosthetic infection, which may include . . . I would perform a two-stage procedure, as that's what I can do safely in my hands.'

Discussion

A large topic with plenty to discuss. Keep the discussion simple, straightforward and non-controversial and avoid getting yourself into a corner. Examiners can sometimes focus in on fairly minor details. Five minutes discussing the sensitivity/specificity of ESR/CRP measurements in the diagnosis of infection can become very uncomfortable. Most candidates would dry up within a couple of minutes.

For an intermediate case discussion you may spend some time discussing various treatment option that may include:

- Antibiotic suppression therapy
- One-stage revision
- Two-stage revision
- Girdlestone excision arthroplasty

Evidence of the effectiveness of one- and two-stage surgical revision is mainly based on interpretation of longitudinal studies. Beswick et al.[20] in systematic review of published studies reported that there was no difference in re-infection rates between the two procedures. They concluded that randomised trials were needed to establish optimum management strategies.

Most surgeons prefer a two-stage revision as it gives the surgeon the opportunity for repeat debridement and exchange of PROSTALAC® spacer if the infection is still present. The results from most single-stage revisions have been from specialized hip centres dealing with a lot of periprosthetic infections, which may not be applicable to an average hip surgeons practice.

Know the principles and operative technique for a two-stage revision[21]. Several recent studies have called into question the use of laminar flow theatres and body exhaust suits in reducing the incidence of periprosthetic joint infection (PJI)[22]. Hooper et al. reviewed 10-year results of the New Zealand Joint Registry and found no benefit with using laminar flow theatres on the rate of revision for early deep infection in total joint replacement [23].

Several new strategies for the prevention of PJI have emerged including the beneficial affects of UV light ,prosthetic antibiotic and antibiofilm coatings and biofilm eradication[24].

The painful THA

History

- The hip is painful
- The patients walking distance has become less and/or walking aids are necessary
- The hip is stiff or does not move at all
- Duration: how long has the hip been painful
- Progression: is the pain getting worse
- Site of pain. Pain localized to the trochanter region suggests bursitis, irritation secondary to underlying wires or sutures, osteolysis or fracture. Pain felt in the buttock or groin suggests vascular or neurogenic claudication, acetabular loosening or osteolysis. Less frequently it may indicate iliopsoas impingement or tendinitus secondary to acetabular cup retroversion, hernia or gynaecological cause. Thigh pain may be secondary to a loose femoral implant or modulus mismatch between the stem and bone
- Pain felt at rest or during the night raises the possibility of infection or malignancy
- Any problems with the wound postoperatively. A history of persistent wound drainage, haematoma, prolonged course of antibiotics following the operation or return to theatre for wound washout should increase the index of suspicion for infection as a cause of the pain
- Any recent bacterial infection or possible bacteremia: urine or chest infection, dental procedure, etc
- Has pain been present since the original index operation: subclinical infection

- Pain-free interval following the initial successful THA: aseptic loosening, late-onset infection
- Is the pain new or similar to preoperative symptoms. Pain similar to the preoperative symptoms suggests the original problem may not have been addressed with the THA and alternative diagnoses should be considered
- With aseptic loosening a triphasic pattern is classic. Pain is sharp with the first few steps of ambulation, is reduced after the patient has walked a moderate distance and then gradually increases after the patient has walked a still greater distance
- Pain that is constant suggests inflammation caused by infection whilst activity mechanical pain suggests implant loosening or impingement

The possible causes for pain are divided into extrinsic or intrinsic aetiologies. Additionally, whether the source of pain is emanating from the soft tissues, bone, implant or a combination.

Extrinsic

This can be further subdivided into local extrinsic-relating to the hip region (but not the implant) and remote extrinsic – unrelated to the hip area but the source of the pathological condition may cause pain to radiate to the hip region.

Remote extrinsic

- Referred pain from elsewhere. A history of back pain with radicular symptoms radiating down the lower extremity well past the knee into the foot more likely relates to spinal pathology. Spinal stenosis, facet arthropathy and radiculopathy can all cause pain in and around the hip and thigh

Local extrinsic

- Trochanteric bursitis, abductor tear, suture irritation, broken trochanteric wires, herniation of the vastus lateralis and HO

Intrinsic

- Aseptic loosening: one or both components
- Infection is present: subclinical, acute, delayed, etc
- Soft tissue or bony impingement
- Modulus mismatch

Examination

- Look at the hip wound, skin and soft tissues noting any inflammation, healed sinus tracks, ulcers, scars or discolouration. Note if there is marked thigh atrophy from disuse
- **Gait**: Is there any asymmetry or abnormality of gait? Is there an antalgic, LLD or abductor deficiency pattern?
- Is the patient Trendelenburg test positive?
- **Palpation**: Are there any hernias or defects in the deep fascia? Is there tenderness on palpation? This may suggest a neuroma or an area of osteolysis. Tenderness over the pubic rami may suggest a stress fracture. Pain at the extremes of hip motion suggests aseptic loosening. Extreme pain with any hip range of motion suggests active synovitis and raises the concern of infection. Pain over the greater trochanter is suggestive of bursitis or trochanteric pain syndrome. Medial pain over the lesser trochanter could be secondary to ilio psoas impingement
- Pelvic obliquity and leg length discrepancy must be determined
- Straight leg raising causing groin pain raises the possibility of psoas impingement
- Check both lower limbs for neurovascular status. Peripheral vascular disease may occasionally present as discomfort in the hip or thigh area. The spine must be evaluated for areas of tenderness or deformity and range of movement

Investigations

Radiographs

Obtain up to date good-quality AP pelvis and true lateral radiographs of the relevant hip and if possible compare these to previous radiographs as this may document migration of either the acetabular of femoral component, which is pathognomonic for loosening.

Look for radiographic signs of aseptic loosening[hh]. Look again for any radiographic features suggestive of infection[ii].

Bloods

'I would then want to perform some routine bloods – ESR/CRP, FBC and WCC[jj].'

Bone scan

Reasonable option to suggest but be careful to follow through and mention that a bone scan may not always be particularly helpful in differentiating between aseptic and septic loosening[kk]. Consider a leucocyte scan or radioactive-labelled white cell scan if infection is suspected.

[hh] If you want to spin out the discussion talk about the Harris classification of loosening (definite, probable or possible), but do not make it too obvious as it will irritate the examiners.

[ii] A general comment to the examiners that radiographs are not particularly helpful in diagnosing infection but features suggestive of infection would include periostitis, osteopenia, endosteal reaction and rapidly progressing loosening or osteolysis but in this particular case none of these features are present.

[jj] It is not unreasonable to explain why you are performing these tests and what you are looking for. Be prepared to discuss sensitivity, specificity, etc. Throw a couple of recent references in if you can but be sensible – It may not be particularly appropriate do so.

[kk] Easy for the examiners to back you into a corner with this one, especially if you don't get your phrasing just right. Again, be prepared to talk about sensitivities, specificities, etc. 'What is the sensitivity of a test? What is specificity? What do we mean by

Hip arthrography and aspiration

In theatre, sterile conditions, ensure skin flora is not picked up – Blood culture bottles ± hip arthrogram[ll]. Arthrography of the hip can demonstrate pocketing of the radiopaque medium in the area of the pseudocapsule, which suggests infection. Unfortunately this finding is uncommon. Culture and cell count obtained.

Differential diagnosis

Divided into intrinsic and extrinsic causes (Table 11.1). An extrinsic aetiology is defined as pain emanating from outside the hip joint. It is further sub-classified into local extrinsic – relating to the hip region (but not the implants) – and remote extrinsic – unrelated to the hip area, but the source of the pathological condition may cause pain to radiate to the hip. An intrinsic aetiology is defined as emanating from the hip joint itself.

Remote extrinsic causes

Spinal stenosis and nerve root irritation can cause pain in the buttock, thigh and sometimes the groin. Vascular disease commonly causes buttock or thigh pain. Metabolic disorders such as Paget disease can occasionally cause symptoms on its own that may persist after THA.

Local extrinsic causes

The psoas tendon may become irritated by a prominent anterior flange of an insufficiently anteverted acetabular component.

Intrinsic causes

Instability can result in pain from capsular stretch and from soft-tissue impingement.

Nothing wrong with the hip

Salient feature was that the pain was never relieved by the THA. Examination may show features suggestive of a problem with the THA, such as a limp and some limitation of motion, but nevertheless none of these signs are likely to be gross. Review of case notes and plain radiographs of the original hip may reveal that the hip before replacement was minimally, if at all arthritic, so that with the retrospectoscope it is clear that the original symptoms did not come from the hip.

Management

Obviously dependent on the cause. The examiners can now choose a number of paths to go down:

- Preoperative planning for revision hip surgery
- Surgical approaches used

Table 11.1 Differential diagnosis of pain following THA

Intrinsic causes	Remote extrinsic causes
Aseptic loosening	Lumbar spine disease: Stenosis, spondylolysis/spondylolisthesis herniated nucleus pulposus (HNP)
Infection	Pelvic disease (PVD)
Wear debris synovitis	Stress/insufficiency fracture
Instability	Metabolic disease
Component impingement	Complex regional pain syndrome (CRPS)
Tip of stem pain (modulus mismatch)	Metabolic (Paget's, osteomalacia)
Stess/periprosthetic fracture	Malignancy, metastases
	Local extrinsic causes
	Heterotopic ossification
	Trochanteric bursistis
	Iliopsoas tendinitis

- Choice of implant
- Complications of revision surgery

Metal on metal (MoM) hips

This is very much intermediate case material. There are enough patients around with painful MoM hips (either resurfacing or total) to bring in for the exam. It is a good quality case with plenty to discuss[mm].

History

Similar to the painful THA
Be slick in your history extraction
Time interval since primary surgery
Initial diagnosis
Make of MoM hip/resurfacing and head size (<46mm higher failure rate)
Pre-operative level of activity and expectations from surgery

Examination

Scar
Anatalgic gait
Trendelenburg positive test
Restricted painful ROM

accuracy? What are the typical values quoted in the literature for the various scans, etc.?'

[ll] All cases or just selectively – Have an opinion – You will have to decide yourself in 2 years or so if you become a consultant.

[mm] It doesn't suit the 'shorts cases' that well unless there are strong positive clinical findings or there is a shortage of patients for the shorts.

Discussion

All symptomatic patients should have either a metal artifact reduction sequence (MARS) MRI performed. MRI is able to diagnose periprosthetic collections, abductor muscle detachment, pseudotumours and the extent of osteolysis.

The Medicines and Healthcare products Regulatory Agency (MHRA) recommend measurement of whole blood cobalt–chromium–metal ion levels. If levels on the initial blood test are >7 parts per billion then blood tests should be repeated at 3 months. This equates to 199 nmol/l cobalt or 134.5 nmol/l chromium. If levels are rising on a second sample and the hip is painful then revision is recommended. There has been some indecision and controversy on the setting of normal threshold values.

Remember to mention non-hip-related causes of pain such as low back pain (LBP), PVD etc.

Indications for revision surgery following MoM resurfacing include fracture, loosening/lysis, pseudotumour, metal hypersensitivity, pain, avascular necrosis, infection and instability.

Examination corner

Intermediate case 1: Painful MoM hip resurfacing implant

History

- When was the index procedure performed?
- Were there any postoperative complications such as wound infection or washout or need for antibiotics?
- What is the pain like: Is it sharp, dull or like a knife?
- How far can you walk?
- Is there sleep disturbance at night?
- Is the pain the same as it was before the index surgery?
- Do you have low back pain?
- What makes the pain better?
- What brings on the pain?

Examination

- Demonstration of gait
- Trendelenburg test

Discussion

EXAMINER: How do you investigate a painful MoM hip resurfacing?

CANDIDATE: I would take a full history and perform a detailed clinical examination. As per MHRA guidelines I would order a MARS MRI scan of the hip and blood cobalt–chromium level measurements

EXAMINER: What are the causes of pain in an MoM hip resurfacing?

CANDIDATE: Excluding extrinsic referred causes of pain such referred pain from the spine intrinsic causes include fracture, osteolysis, AVN, loosening, adverse reactions to metal debris (ARMD), hip impingement, iliopsoas tendinopathy and pseudotumours.

EXAMINER: These are the radiographs. What do you see?

CANDIDATE: I would assess for cup abduction and anteversion angles, radiolucencies about the metaphyseal stem, narrowing of the neck (compare with postoperative radiographs) and radiolucent zones suggestive of osteolysis. (Figure 11.17)

COMMENT: Attempt to mention to the examiners (subtly or otherwise) three angles: The neck shaft angle (NSA), the stem-neck angle (SNA) and the stem-shaft angle (SSA). (Figure 11.18) A higher NSA of the femur reduces the risk of fractured femoral neck. Look for evidence of repetitive impingement demonstrated by radiographic signs of repetitive bone-to-component abutments such as a depression in the neck or a contour just below the junction with the component (divot sign) often associated with a reactive exostosis.

EXAMINER: What was the mode of failure of the ASR®?

CANDIDATE: The mode of failure was closely linked to design features. The implant has a subhemispherical acetabular component and a lower diametrical clearance between components. The low clearance caused lubricant fluid lockout greatly increasing metal wear and the subhemispherical design increased the risk of edge loading of the acetabular component, especially in mal-positioned, small components. There was a reduced arc of cover compared to other MoM implants. Therefore, in components matched for size and inclination, articular contact takes place closer to the rim of an ASR® component, resulting in edge loading, which is strongly

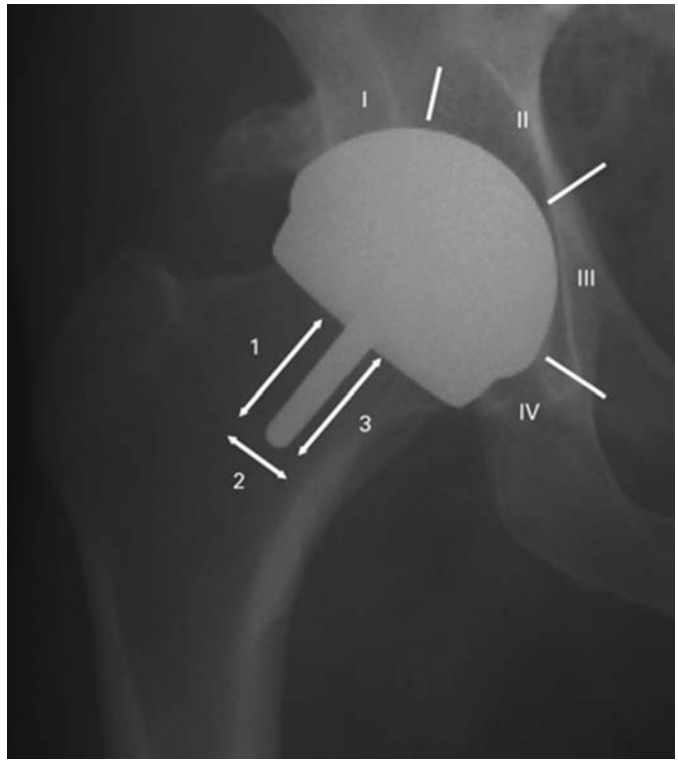

Figure 11.17 AP radiograph hip demonstrating zones 1, 2 and 3 around the peg of a hip resurfacing arthroplasty implant as described by Amstutz et al.[25]

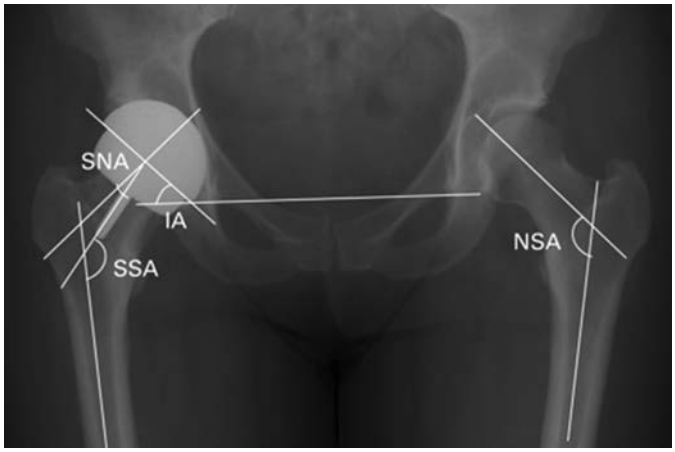

Figure 11.18 AP radiograph hip demonstrating MoM hip angles. Neck shaft angle (NSA), Stem neck angle (SNA) and stem shaft angle (SSA)

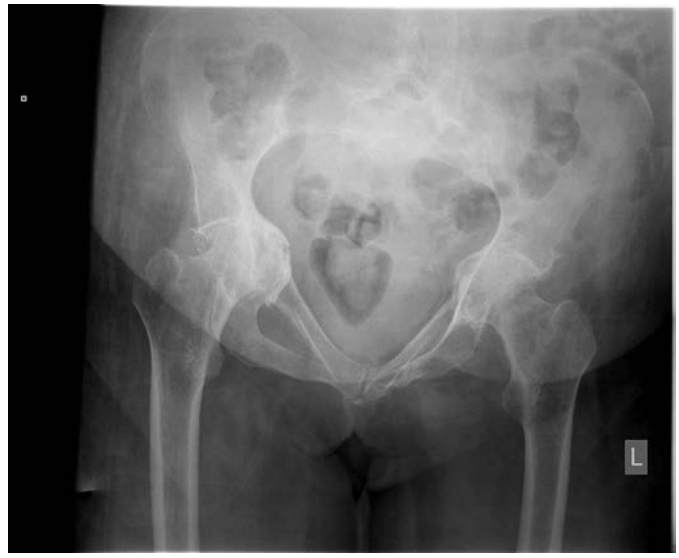

Figure 11.19 AP radiograph of bilateral severe protrusio hip

associated with articular wear. In simple terms the design of the cup was too shallow. The ASR® implant had higher-than-expected revision rates in the UK NJR and Australian registry. The revision rate at 5 years in the NJR was 12% for ASR® resurfacing and 13% for stemmed ASR® components. This compares with a 5% cumulative revision rate at 8 years for the Birmingham hip from the Australian registry. This has led to its withdrawal.

EXAMINER: Is a resurfaced MoM hip a better functioning hip than a conventional hip?

CANDIDATE: Current data suggests that there is very little clinical difference in outcome between resurfacing hip arthroplasty and conventional THA. However NJR data does suggest resurfacing hip arthroplasty may perform slightly better in males aged below 55 years.

Protrusio acetabuli (Figure 11.19)

History

'Middle-age female patient with progressively worsening bilateral hip pain. Pain worse on the right side. Pain dull in nature worse with activity.'

'Typical complaints are of increased stiffness and lack of hip flexibility rather than pain (anatomical abnormality-deepened socket).'

'As secondary OA develops pain and limp become more pronounced.'

Enquire about any family history of hip disease.

Symptoms due to causative disease – Rheumatoid, osteomalacia, Paget's, etc.

Clinical examination

'There is increased lumbar lordosis due to the flexion deformity at the hips. Gait was analgic. Trendelenburg's test was strongly positive bilaterally. Thomas' test revealed a fixed flexion deformity on the right of 20° and 10° on the left.'

Square up the pelvis with the bed and make sure the legs are parallel with the pelvis. Measurement of leg length revealed true right leg shortening of 2 cm. Galeazzi's sign suggested the shortening was in the femur. Digital Bryant's triangle palpation suggested that the shortening was above the trochanter. There was a reduced distance between the thumb and tip of the index finger on the right side compared to the left[nn].

Right hip movements were grossly restricted and painful in particular abduction. Active flexion was limited to 60° with only 70° obtained passively. There was no internal or external rotation in flexion. Passive abduction was markedly limited to only 10° whilst adduction was greater at 20°.

On the left side movements were again restricted and painful but to a lesser degree. Active flexion was only possible to 90°. No internal or external rotation in flexion was possible. Passive abduction was again markedly limited to 10° with adduction restricted to 20°.

All hip movements become progressively painful and limited, especially abduction as the trochanter starts impinging on the superior acetabular margin. Often a better arc of flexion is preserved. The hip may even become ankylosed in severe cases with PR revealing a globular mass on the lateral rectal wall.

Discussion

- Aetiology of the condition: Idiopathic or secondary (rheumatoid, Paget's, osteomalacia, osteoporosis, AS, trauma, Marfan's, etc)

[nn] Talk to the examiners during your clinical case. Do not examine in silence. In this case the candidate is indirectly letting the examiners know that he understands Bryant's triangle.

- Radiological classification – Hirst: The ilioischial line is often used to measure the amount of protrusion. The ilioischial (Kohler's line) line represents the quadrilateral plate of the acetabulum that projects over the medial acetabular wall due to the tangency of the x-ray beam
- Preoperative planning including investigations, implant choice and bone grafts

  If bilateral leave the index leg longer with a plan to equalize leg lengths at the second procedure. Discuss LLD preoperatively with patient to avoid dissatisfaction. Avoid offset and significant LLD issues. CT scan evaluates defects in the posterior and medial wall prior to surgery. Preferred option is to restore hip center of rotation using an uncemented cup with or without bone graft. Template to determine size and location cup and femoral offset. Avoid excessive femoral lateralisation as this may cause excessive tension on the iliotibial band/abductors, resulting in an abduction contraction and a significant functional leg lengthening
- Principles of THA reconstruction

  Restoration of the hip center of rotation to optimize ROM
  Maximize efficiency of hip musculature
  Minimize adverse loading conditions across the articulation
- Surgical technique

  Femoral neck may need to be osteotomised in situ before dislocation. Mobilization of the femur may be difficult due to inward migration femoral head and stiffness. Do not forcefully attempt to dislocate hip or else a fracture of the posterior wall acetabulum, fracture proximal femur or major ligamentous injury knee can occur. The acetabulum does not need to be deepened, but a bleeding cancellous bony bed for graft placement is preferred to encourage healing of the graft to host bone. Sciatic nerve is often nearer the operative field due to medial migration of the femur associated with the deformity. A femoral stem with increased offset may be required to reduce likelihood of bony or component impingement but avoid excessive offset (see above)
- Results of THA

  Baghdadi et al.[26] in a restrospective review of 162 hips with protrusio reported 89% survival rate for uncemented cups at 15 years compared to 85% for cemented cups. There was a 24% increase in the risk of aseptic cup revision for every 1 mm medial or lateral distance away from the native hip centre to the prosthetic head centre. Lateralized hips were equally at risk of failure, so bone graft when not needed should be avoided
- Complications of THA

  Loosening, medial migration of the acetabular component, dislocation, infection and LLD

Tuberculosis hip

A case of old tuberculosis of the hip enrolled for the examination.

Memorandum

'On examination we have an elderly man of average height and build. There are several well-healed scars over lateral aspect of the left hip There is a discharging sinus present in the groin/greater trochanter There are puckered scars suggestive of healed sinuses. The left leg is shortened with gross generalized wasting particularly of the thigh and buttock. There is a pelvic obliquity with the ASIS lower on the left side and a compensatory scoliosis which does not fully correct when sitting down on the couch suggestive of a fixed element to the scoliosis.'

'Inspection of gait revealed a short leg gait. There was also a suggestion of lack of movement of the left hip with the trunk being thrown forwards to aid walking.'

'There is a gross left leg length shortening. The left leg has a flexed attitude and in order to measure leg lengths correctly both limbs need to be placed in equivalent positions; therefore, using a pillow to fix the right leg in the same position of flexion as the left. There is a combination of real and apparent shortening of the left leg. Galeazzi's test demonstrated that most of the shortening is in the femur, but there is, however, a suggestion of a small amount of tibial shortening. Bryant's triangle suggests most of the femoral shortening is above the trochanter. There is a definite decreased difference between my thumb over the ASIS and fingers over the trochanter on the left side compared to the right. The hip is flexed, adducted and medially rotated. All movements are grossly restricted by pain and spasm.'

Muscle spasm in the early stages can be elicited by rotating the extended hip when the muscles around the joint as well as the abdominal muscles exhibit spasmodic contraction (Gauvain's sign[oo]) If no hip movement occurs at all consider bony ankyloses. Usually a combination of real and apparent shortening exists. In the initial stages of the disease there is slight flexion, abduction and lateral rotation. Earliest clinical sign is a limp, which comes on after walking. Flexion is concealed by an exaggerated lumbar lordosis. Adduction is corrected by tilting pelvis upwards which results in a scoliosis of the lumbar spine with convexity towards normal side. If the hip has been fused the patient may develop back or hip pain several years later. In a young patient if there is mal-position of the fusion consider corrective osteotomy rather than arthroplasty. If a painful pseudoarthrosis exists consider fusion rather than arthroplasty.

[oo] Described by Sir Henry Gauvain in 1910. This test is of value in early doubtful cases of tuberculosis of the hip. In active tuberculosis of the hip, on initiating rotatory movements the muscles around the hip and lower abdomen go into spasm. The lower end of the thigh is rotated internally and externally. The movement is then checked and any further slight sharp rotation is followed by spasmodic contraction of the joint muscles as well as those of the lower abdomen.

Radiographs

The earliest sign is a general haziness of the bones as seen in a bad film but with a normal joint space and line with or without an area of rarefaction in Babcock's triangle (inferior aspect of the femoral neck). Increased joint space due to an effusion.

Later on there is gross enlargement of the acetabulum roof with the femoral head migrating into the dorso-ilium (**travelling or wandering acetabulum**). The combination of partially destroyed femoral head, destroyed acetabulum and muscle spasm can lead to a posterior dislocation of the hip. In some situations the femoral head is destroyed and becomes small and contained in an enlarge acetabulum giving rise to a **mortar and pestle** appearance. Softening and destruction of the medial wall of the acetabulum can lead to **protrusio**. With healing bony ankylosis may occur.

Clinical features

Presenting complaint: Disease insidious in onset and runs a chronic course. One of the first symptoms is stiffness of the hip. A child may be pale, apathetic with loss of appetite before definite symptoms pertaining to the hip appear. Pain may initially be absent or be referred to the knee. Pain occurs around the hip particularly with weight-bearing

Gait: Stiff hip gait. While walking the hip is kept stiff and forward – Backward at the lumbar spine is used for propulsion of the lower limb. Because of the flexion deformity of the hip the patient stands with a compensatory exaggerated lumbar lordosis Later on an anatalgic gait may develop to quickly take the weight off the affected side

Muscle wasting: The thigh and gluteal muscles are wasted

Swelling: There may be swelling around the hip because of a cold abscess

Discharging sinus: There may be discharging sinuses in the groin or around the greater trochanter. More likely there may be puckered scars from healed sinuses

Shortening: There is a true shortening of the hip in tuberculosis except in stage 1. There may be a combination of true and apparent shortening of the limb. Be able to measure true and apparent lengths of a limb and be quite clear on the difference between the two

Stage 1. Synovitis: The stage of apparent lengthening

Initially the clinical features are common to all diseases producing synovitis. There is a joint effusion, which demands the hip to be in a position of maximum capacity and comfort. This is obtained by a position of **flexion, abduction and external rotation of the hip**.

Since the flexion and abduction deformities are only slight and are compensated for by tilting of the pelvis, these do not become obvious. Flexion is concealed by a lumbar lordosis and by tilting of the pelvis forwards. Abduction is corrected by tilting the pelvis downwards and scoliosis of the lumbar spine with convexity towards the affected side.

As the pelvis tilts downwards to compensate for the abduction deformity, the affected limb looks longer (apparent lengthening) than the normal opposite hip, though on measuring true limb lengths the two limbs are found to be equal. This stage lasts only short length of time and it is rare to see a patient in such an early stage of the disease.

Stage 2. Arthritis: The stage of apparent shortening

The effusion subsides There is involvement of the articular cartilage. This leads to spasm of the powerful muscles around the hip to protect its movement. Since the flexors and adductors are stronger muscle groups than the extensors and abductors the attitude of the hip is one of **flexion, adduction and internal rotation**. The flexion and adduction may be concealed by the compensatory tilt of the pelvis but the internal rotation of the leg is obvious.

Adduction is corrected by tilting the pelvis upwards resulting in scoliosis of the lumbar spine with convexity towards the sound side.

As the pelvis tilts upwards to compensate for the adduction the affected limb appears shorter (apparent shortening) than the normal opposite hip, although on comparing the limb lengths in similar positions, true shortening is usually absent or not >1 cm.

Stage 3. Erosion: The stage of real shortening

In this stage, the cartilage is destroyed and there is erosion of the upper part of the acetabulum and the femoral head becomes dislocated by the by the spasm of the adductors. (Wandering acetabulum or pathological dislocation.) The attitude is similar to that seen in stage 2 but exaggerated. There is true shortening of the limb because of the actual destruction of bone. In addition, the apparent length of the limb is further reduced because of the adduction deformity.

Management

Total hip arthroplasty

Consider:

- Possibility of reactivation of infection following THA
- Shortening of the limb
- Deformed greater trochanter
- Distorted anatomy placing sciatic nerve and femoral artery at risk of injury
- Acetabular defect
- Antituberculosis treatment 3 months pre-surgery and 9–12 months post surgery
- Disease-free interval 10 years recommended
- Preop confirmation with biopsy/aspiration. Rule out other focci of TB such as chest
- Both uncemented and cemented THA can be used

Excision arthroplasty

This is an option for a patient who will not accept stiff joint. Culturally will allow them to sit cross-legged and also squat. It provides a mobile and painless hip joint but produces shortening and instability leading to tiredness and a need for a walking aid.

Arthrodesis

This is a possible option in a young patient with a deformed painful hip. The rate of pseudoarthrosis is high (up to 70%) because of poor bone stock. It produces a painless, stable and immobile joint with a slow asymmetrical and arrhythmic gait.

Examination corner

Intermediate case 1: Tuberculosis left hip as child

- Underwent arthrodesis left hip aged 20
- Deformity of spine
- Leg length discrepancy
- Valgus deformity left knee
- OA right knee

Intermediate case 2: Arthrodesis left hip with ispsilateral knee OA

Examination

Flattening of the buttock and gluteal fold secondary to muscle wasting of glutei and adductors.
Wasting thigh muscles.
Adduction deformity with pelvic tilt.
Long discussion about real/apparent shortening. I had to be slick with the tape measure whilst also answering the examiner's questions. Shortening was a mixture of true and apparent shortening owing to the adduction deformity.
Stiff hip with no movement.
Thomas' test.
Knee had a semiflexed deformity with posterior subluxation. It was stiff to move.
Knee effusion with synovitis. Asked to test for knee effusion[pp].
I had to be slick with the tape measure with the FFD when measuring true and apparent leg lengths.

Discussion

'TKA alone in a patient with an arthrodesed hip is unlikely to provide a satisfactory result. Patients with severe knee disease below a hip arthrodesis require THA followed by TKA.'

'There is a need for anti-tuberculosis treatment for at least 3 months before surgery and continued for a total of 18 months. Generally a long period of quiescence is recommended before TKA. There are concerns with reactivation of the infection, loosening of the TKA. As a minimum, posterior-stabilised (PS) knee should be used.'

'There was a recent paper from India[27] published in the Journal of Bone and Joint Surgery, which suggested that THA/TKA in the presence of active tuberculosis hip/knee is a safe procedure when preoperative chemotherapy is commenced and continued for an extended period after operation. I would, however, have severe reservations in adopting this policy for UK patients. I would adhere to standard UK protocols and guidelines if arthroplasty is indicated.'

Short case 1: Tuberculosis hip

'I got a case of tuberculosis of the hip in a 12-year-old boy with a painful limp. The examiners asked me to take a brief history from the parents, which I started off by asking about presenting complaints, which were of hip pain and limping.'

'They cut me short and asked me to examine the hip. I made the child walk. They asked me what type of gait it was. I mentioned it was an antalgic gait and then said I would like to go on and perform Trendelenburg testing but the examiners said not to bother with this. I proceeded on to movements and deformities. The child had a 40° FFD, and further flexion up to 90° was possible. All other movements, including adduction and abduction as well as rotation, were restricted. The examiners asked me for a differential diagnosis. I said that it could be old septic arthritis or even tuberculosis as there was still some hip movement preserved. The examiners showed me an x-ray that showed collapse of the femoral head and asked me what differential diagnosis I could think of. I said that it could be an AVN secondary to an old femoral neck fracture or Perthes' disease. The examiners were happy with that. Before they could go on to the management, the bell rang and the 5 minutes were up.'

Young patient with rheumatoid hip disease

This is classic intermediate case material with a lot to talk about and discuss with the examiners. In juvenile RA the problems relates to the onset of the disease. The younger the onset the more severe the growth retardation and deformity. These patients are often severely affected with multiple joint disease and severe osteoporosis. In the FRCS (Tr & Orth) examination a patient seen, as a intermediate case may be under consideration for surgery; therefore, a specific general physical examination should be undertaken.

Specific attention should be paid to the following.

Anaesthetic concerns:

- Cervical spine instability. It is important to access stability cervical spine preoperatively and exclude the presence of a cervical mylopathy[qq]. Evaluate for neck pain, neurological signs and symptoms and radiographic changes

[pp] Be careful with this, get it slick. The examiners weren't happy with my method. I am not sure why, perhaps not performing the test with the confidence that one should have with doing it everyday in clinic. Possibly they were being picky.

[qq] Intermediate case of rheumatoid disease mainly affecting the shoulder and elbow. A large part of the clinical examination was directed towards examining a cervical myelopathy present.

- Hypoplasia of the mandible and stiffness of the TMJ may make intubation difficult
- Pulmonary involvement
- Restriction in movement upper limb so to avoid injury whilst establishing IV access

Orthopaedic concerns:

- Medication (steroids, etc)
- Biological medications is usually stopped prior to surgery
- Rheumatoid disease distorting hip anatomy
- Implanting THA in a young patient

Steroid use:

- General debility
- Fragility skin
- Osteoporosis
- Poor musculature
- Increased risk of wound infection
- Wound healing takes longer

Distorted anatomy:

- Hypoplasia of the pelvis and femur
- Gross anteversion and valgus angulation of the femoral neck may lead to difficulty controlling alignment of a femoral component
- Generalized severe osteoporosis and marked soft-tissue contractures
- Marked anterior bowing upper femoral shaft
- Acetabular dysplasia – Small size but possible protrusio
- Coxa magna and/or subluxed femoral head
- Premature closure of the growth plate
- Fibrous ankylosis hip necessitating in-situ osteotomy of the neck

Implanting THA in a young patient:

Polyarthritic, often underweight, often put less stress on their components, wear less than expected

Indications:

Pain is the major indication for surgery. Loss of function and reduced ROM are secondary indications. Mobility may not be greatly improved post-THR because of limitations due to disease in other joints and an improvement in mobility may depend on replacement of other joints in the lower limb.

Technical concerns when performing THA:

Great care is needed when dislocating the hip to prevent a femoral shaft fracture or damage to the to ipsilateral knee. Reaming the femur is easy because the femoral canal is usually wide but the cortex is soft and easily penetrated or fractured. The femur is often underdeveloped and a smaller implant may be required. Intraoperative pelvic fracture can occur. If acetabular protrusio is present avoid penetration of the medial wall and be prepared to bone graft if necessary. Some authors suggest it is preferable to avoid trochanteric osteotomy since during re-wiring of an osteoporotic trochanter there is a risk the bone may become fragmented. Others would suggest trochanteric osteotomy decreases the risk of femoral shaft fracture or perforation from malposition of the femoral reamers. Increased risk of trochanteric displacement (15%) and non-union (10%)[28]. When the acetabular floor is extremely thin bone grafting with multiple morselized segments cut from the excised femoral head may help to preserve bone stock. If there is a large medial wall deficiency a single solid bone graft fashioned from the femoral head may be used for reconstruction.

If the roof of the acetabulum is deficient then it can be reconstructed by screwing on segments of the patient's femoral head or similar allograft bone to reconstruct the roof. Where the acetabular floor has become completely fragmented or destroyed but the rim of the acetabulum remains intact use of an acetabular ring may prove useful.

Results:

The survival of the prosthesis is less than that seem in elderly rheumatoid patients, and just under 50% are loose at 5 years and the 10-year survival as measured by revision is approximately 75%.

Recurrent dislocation THA

This is more likely to be a viva topic than a clinical case. The hip may dislocate during examination and this would be a disaster.

History and examination

The history should begin with details of the last episode of dislocation and any previous episodes of instability. When was the index hip procedure performed and what was the original diagnosis? What approach was most likely used (check scar) and which components were used (check operation note)? What was the direction of the dislocation (anterior, posterior, other)? What was the prior management of the dislocation?

Any patient-specific risk factors for late dislocation, such as younger age (greater wear), female gender (decline in muscle mass), AVN, preoperative diagnosis of fractured neck of femur, etc.

Clinical examination

This should include a full bilateral lower extremity examination with particular attention to scar, gait, hip contractures, ROM, strength of muscles (particularly abductors) and neurovascular examination. Look for clinical evidence of infection. Limb length discrepancy can be associated with component

malpositioning because intraoperative instability secondary to suboptimally positioned components such as a retroverted socket may have been addressed by lengthening of the femoral neck to increase soft-tissue tension.

Operative notes should be reviewed to determine surgical approach, type of soft-tissue repair, specific implants used and any technical difficulties encountered.

Imaging

AP view of the pelvis, AP view of the hip and a cross-table lateral view of the hip are required. Check for component mal-alignment, evidence of wear by eccentric seating of the femoral head inside the acetabular liner, osteophytes, bone quality and integrity, femoral offset and leg length, component geometry, trochanteric non-union, osteolysis and component loosening. Component identification must be carried out (must confirm).

CT is sometimes necessary to assess component position more accurately, especially acetabular version.

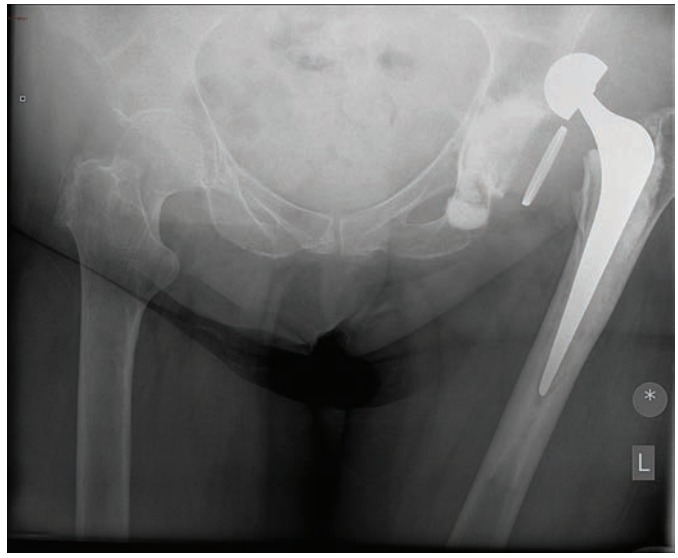

Figure 11.20 AP pelvis demonstration: Type III failure of the femoral head locking mechanism

Classification of dislocation

Early dislocation (weeks or months) suggests problems with soft-tissue tension, such as muscle weakness and inadequate capsular healing and scarring, component malposition, infection or patient non-compliance. Late dislocation (beyond 5 years) is usually multi-factorial and can include stretching of the soft tissues, polyethylene wear, neurological impairment and trochanteric avulsion fractures secondary to wear and osteolysis.

Dislocation occurring between 6 months and 5 years is classified as intermediate.

Discussion

Discussion would start with a review of the radiographs. This will lead on to the causes of recurrent dislocation. There may be an obvious cause but often the reason is complex and multi-factorial. Next would be management of recurrent dislocation in general and in this particular patient.

1. Incidence
2. Classification (early, intermediate, late, with discussion of aetiology for each category)
3. Mechanism of dislocation

   Type 1: Cup malposition (33%)
   Type 2: Stem malposition
   Type 3: Abductor insufficiency (37%)
   Type 4: Impingement
   Type 5: Wear and head penetration
   Type 6: Unclear aetiology
   Management specific to the mechanism is identified and consists of component revision (type 1 and 2), insertion of constrained liner (types 3 and 6), removal of the source of impingement, and insertion of a larger head (type 4) or liner exchange (type 5)
4. Predisposing factors for dislocation Divide these into:
   - Patient-specific risk factors (female sex, AVN, obesity, increased age, co-morbidities, femoral neck facture)
   - Variables under the surgeon's control (surgical approach, component position and orientation, femoral head size, restoration of offset, preservation of soft tissue integrity, leg length and prosthetic impingement)
   - Surgeon experience (risk of dislocation inversely related to the case volume of the operating surgeon)
5. Component alignment

   Unrecognised subtle component mal-alignment is common and difficult to detect on plain radiographs. Ascertain the direction of dislocation as a possible clue (excessive cup anteversion associated with anterior dislocation, etc). The role of CT is to more accurately assess component positioning, especially cup version
6. Early vs late dislocation

   Early dislocation is often successfully managed non-operatively. Late dislocation generally requires surgery
7. Management options

   These include closed reduction with or without bracing, THA component revision, exchange of modular parts, bipolar hemiarthroplasty, tripolar unconstrained acetabular component (dual motion), elevated rim liners, use of a large femoral head, use of a constrained acetabular liner, greater trochanter advancement and soft-tissue augmentation. The choice depends very much on the aetiology of the problem. Revision arthroplasty for recurrent dislocation is much more likely to be successful when a cause has been identified
8. Posterior vs anterolateral approach

For patients at high risk for posterior dislocation (elderly, mild cognitive impairment, etc), an anterolateral approach may reduce the risk. Woo and Morrey[29] demonstrated a significantly higher dislocation rate after a posterior approach 5.8% compared to 2.3% for an anterolateral approach. The safe acetabular zone concept of Lewinnek et al.[30] is worth knowing

9. How do you perform a posterior approach to the hip?

   Favourite surgical approach for examiners to ask. May lead on from previous question

10. Modular component exchange

    This involves exchanging the acetabular liner and the femoral head, with the main intention being to 'upsize' the femoral head and/or use an elevated liner. This is only successful if the patient has well-positioned and well-fixed acetabular and femoral components. The acetabular component in place must be sufficiently large to allow an adequate thickness of polyethylene to be used with the larger femoral head

11. Constrained acetabular liner

    This is used as a salvage procedure in a difficult subset of patients which includes the following: Management of recurrent dislocation secondary to soft-tissue (abductor) dysfunction, recurrent dislocation of unknown aetiology, patients with neurological impairment and elderly patients in whom components are well fixed. It is designed to hold the head captive within the acetabular component by means of a locking mechanism. Forces which would otherwise cause dislocation are transferred to the locking mechanism and the liner-shell and shell–bone interface. They can be either bipolar (single articulation) or tripolar (double articulation) and cemented or uncemented design. A constrained liner can be cemented into a well-fixed cementless acetabular shell. The retained acetabular component should be large enough to allow an adequate cement mantle around the constrained liner. It is important to make sure before using a constrained liner than components are well positioned and that subtle malpositioning is not the cause for the dislocation

12. What are the problems associated with the use of constrained liners?

    The problems are early wear and dislodgment of these liners. Cooke et al.[31] classified three types of early failure

    - Type I failures (of the bone-prosthesis interface)
    - Type II failures (of the liner locking mechanism)
    - Type III failure (of the femoral head locking mechanism) (Figure 11.20)

    In addition, a fourth failure mechanism – Complete dissociation of the pelvis (type IV) has been described when a constrained liner is used in combination with a cage

    Type I failures can be avoided by using supplemental screw fixation for the cementless shell before inserting the constrained acetabular component. Type II failures can be minimised when cementing the constrained liner into the cementless shell by seating the liner fully into the shell. Scoring or roughening the polyethylene lightly with a burr enhances the grouting bond of the cement and may minimize debonding of the cement–polyethylene interface. Type III failures can be minimised by ensuring that range of motion does not lead to component–component or component–bone impingement

13. Bipolar arthroplasty

    This is not a first-line choice as there is potential for medial or superior migration of the prosthesis with time. In addition, groin pain is not an infrequent problem

14. Dual motion (tripolar arthroplasty)

    This involves the use of a large bipolar head articulating with a large acetabular shell. The large femoral head and the potential for motion at two interfaces increases hip range of movement until impingement occurs and accounts for the high rate of success of this prosthesis in addressing recurrent instability

15. Jumbo heads

    The arc of motion required to dislocate a prosthetic head is directly related to the diameter of the head (jump or excusion distance). Reasonably successful early results have been reported but there is concern about the use of a thin PE liner and osteolysis[32]

16. Soft tissue reinforcement and advancement of the greater trochanter

    The main issue is variability in outcome of technically demanding surgery, which is likely to fail with component malposition. It is best suited for young high demand patients

Multiple epiphyseal displasia

This is one of the more common skeletal dysplasias. It is characterized by abnormal maturation of the epiphyses, affecting the hips, knees and ankles to a greater extent than the shoulders, elbows and wrists.

History

- There is deteriorating hip pain interfering with activities of daily living (ADL). Patients are characterized by mild asymmetric short stature, short limbs relative to their trunks, short stubby digits, early onset osteoarthritis and a waddling gait
- Hip arthritis is bilateral and symmetrical and requires THA at an early age
- Femoral abnormalities include an expanded metaphysis, a narrow isthmus and a varus femoral neck–shaft angle. The acetabulum is dysplastic with deficiency of the dome and

anterior wall predisposing to various degrees of proximal femoral displacement
- The femoral stem used has to take into account the metaphysis/diaphysis mismatch
- Patients are prone to heterotopic ossification

Radiological features of multiple epiphyseal dysplasia (MED)

This is an AP radiograph of the pelvic and both proximal femora. Both metaphysis are expanded, the isthmus is narrowed and there is a varus femoral neck shaft angle. Both acetabuli are dysplastic

Preoperative planning

This is mandatory to predict the implant size, position and orientation and alignment; to restore the centre of rotation of the arthroplasty; to equalize limb length; and to anticipate complications. Get a CT scan to look for femoral head and acetabular version

S-ROM® uncemented femoral stem

There are challenges performing THA in patients with MED. The S-ROM® Modular Hip System femoral stem is a good option as it will address the atypical femoral geometry seen in this type of patient[rr].

It is a proximally loading modular stem designed for maximal proximal fit without distal ingrowth in order to promote proximal stability and ingrowth.

It is a good option for a complex primary hip replacement and although it can be used in a revision setting, this is generally avoided. If there is poor proximal bone stock ingrowth is less likely to occur which may result in subsidence and loosening of the stem.

Rotational stability is achieved by using flutes. Coronal slots reduce bending stiffness and the risk of femoral fracture on insertion. The design feature significantly reduces thigh pain.

The modularity between the S-ROM® stem and the sleeve allows optimal engagement, both at the diaphysis and at the metaphysis of the femur, which are mismatched in patients with MED. One possible disadvantage of using an S-ROM® implant in patients with a small stature is the relative length, which may cause anterior impaction due to proximal femoral bowing. Thus, preoperative templating on both the anteroposterior and lateral radiographs is necessary to identify the appropriate entry point for the femoral component.

Other possible skeletal dysplasias include spondyloepiphyseal dysplasia and pseudoachondroplasia.

[rr] The examiners may quiz you about the specifics of this implant if you are a score 8 candidate.

Leg length discrepancy and THA

This is estimated at approximately 15% after primary THA and is the commonest cause of litigation in the USA. It forms a possible focal point of discussion for complications arising from THA. For an intermediate case there are more interesting causes of leg length discrepancy and perhaps not enough clinical signs to demonstrate unless it is part of a more complex case A stand-alone short case is possible, but patients are generally not happy with leg length discrepancy after THA and so are unlikely to volunteer for the exam unless awaiting revision surgery[ss].

History

- Take a complete surgical and medical history
- A history of previous fracture, infection, physeal arrest and various dysplasias may result in leg length discrepancy
- Abnormalities of the axial skeleton such as previous spinal fusion, scoliosis, neuromuscular disorders or soft-tissue contractures of the hip or knee may contribute to the subjective impression of leg length discrepancy and account for differences in true and apparent shortening

Examination

> 'On examination standing there is a pelvic obliquity present with the ASIS not level which is suggestive of a leg length discrepancy. An equinus position of the left foot levels the ASIS and squares the pelvis. The two dimples overlying the PSIS directly above the buttocks are not lying horizontal, which again is consistent with pelvic obliquity. The iliac crest is subcutaneous and easily felt; normally they are level in relationship to each other but if not this is usually due to pelvic obliquity. The posterior edge of the greater trochanter is easily felt and is usually level.'
>
> 'I would like to correct the leg length discrepancy with wooden blocks.'

True leg length is determined by measuring the leg from the ASIS to the tip of the medial malleolus. These landmarks may be difficult to feel in obese individuals. The apparent leg length is determined by adding the effects of pelvic obliquity and soft-tissue contractures.

A compensatory flexible mobile scoliosis may develop with a true leg length discrepancy. The scoliosis deformity will fully correct when a block is placed under the shorter extremity or when the patient sits down on an examination couch. A rigid coronal deformity remains unchanged.

[ss] Ten or so candidates telling the examiners the hip was put in short/long in front of a patient is not conducive to low medical defence fees.

When measuring leg lengths look as though you have used a tape measure before and not as though in the exam it is for the first time. Make sure the tape measure is easy to carry about, neat if the foldaway variety and make sure it is long enough[tt].

EXAMINER: What is happening with a partial correction of the scoliosis curve?

CANDIDATE: This suggests the scoliosis is a mixture of both a fixed and flexible deformity.

AP radiographs: A line is drawn at the most inferior portions of the ischia, providing the pelvic reference line. A perpendicular line is drawn bilaterally from the transischial line to the superior aspect of the lesser trochanter to determine length difference.

Accurate preoperative planning and templating of radiographs is critical to the proper selection and positioning of components. This should reduce the possibility of a leg length discrepancy following THA.

Intraoperatively, there should be a reproducible method for determining limb length. Palpation of the heels and patella is a relatively crude method. Other methods include measuring the distance between fixed pins into the ilium and greater trochanter, callipers and measuring from the lesser trochanter to the centre of the femoral head.

Examination corner

Intermediate case 1: Elderly male with cemented right THA and left hip resurfacing. Leg length discrepancy 5 cm with short right leg

History

The right hip replacement had been performed 15 years previously. The hip resurfaced 6 years previously.

The main compliant was of the leg length discrepancy. The patient mentioned that they had had a problem with the right hip in childhood but wasn't sure of any further details, and he explained that this is why the leg was short. There were no issues whatsoever with the left resurfaced hip, it was pain free and functioning well. Because of the significant LLD the left hip resurfacing procedure would have been relatively contra-indicated. I mentioned that perhaps there was a worry with dislocation risk and that's why it had been performed.

Examination

Huge shoe raise on the right side

Scars

Measurement of true and apparent leg length discrepancy. The shortening was in the femur

Galeazzi's test. Knees at different levels. The femora were parallel so the discrepancy was in the femur (not below the knee)

Bryant's triangular test. There was a difference in the perpendicular distance between ASIS and the greater trochanter (GT). This suggested the discrepancy was proximal to the GT on the right side

Discussed Nelaton's and Schoemaker's lines very briefly. 'The examiners just wanted the name rather than anything else'

Discussion

'I was shown an AP radiograph. The cup on the right side was cemented and had been put in very superiorly. The cement mantle was thin but didn't appear loose. The cemented femoral stem had been inserted very distally. The left hip resurfacing looked fine. I was asked what I thought of the radiograph. I mentioned possible DDH with the cup placed in a high hip centre. I was asked if I would revise the right hip. I said in the absence of pain I wouldn't because it would be a complex revision case that may end up with complications. The patient had a number of significant co-morbidity factors, which meant I didn't think revision would be the correct answer. My guess was correct.'

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[tt] Don't use a short tape that doesn't cover the distance between ASIS and medial malleolus or cheap and cheerful paper tape, which gets torn during use.

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Chapter 12

Knee clinical cases

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Clinical examination

General guidance:

- Introduce yourself to the patient
- Ask for permission to examine the knee
- Adequately expose both knees, thighs and feet
- Ask if the knee is painful
- Always ask the patient to walk
- Always remember to check spine, hip and pulses
- You must have a well-rehearsed system to examine the knee

Standing

- Look for clues (brace, sticks, callipers, rheumatoid arthritis (RA) hands and shoe raise)
- Inspection (front, side and behind)
- General posture (leg length, rotation, varus, valgus or windswept knees, recurvatum, fixed flexion, patella rotation, legs and feet disorders)
- Quadriceps wasting, especially vastus medialis obliquus (VMO) (ask patient to tense quads whilst standing)
- Skin changes (psoriasis, varicose veins)
- Scars are very helpful but sometimes they are not visible especially arthroscopic (get closer)
- Skin grafts or flaps and sinuses
- Swelling (position in relation to patella)
- Look at the popliteal fossa

Walking

- Gait pattern
- Is the knee kept in flexion or moving freely? If flexed posture – Cannot comment on 'fixed flexion' until full examination of knee and hip
- Varus/valgus thrust. Thrust = increased deformity in stance phase

Supine

Look

Check again for any skin discoloration, sinuses, scars, quads wasting, joint swelling and osteophytosis.

Feel

- Skin temperature
- Effusion test. For small effusion use the bulge, sweep or milk test

  Empty the suprapatellar pouch and displace the knee fluid laterally. Compress the lateral side and watch for a bulge medially. Use the patellar tap or cross-fluctuation test for large effusion. Pinch any synovial thickening to differentiate it from effusion
- Assess extension (particularly hyperextension)
- Fixed flexion deformity. Slide your hands behind the patient's knees and ask him/her to press the knees into the couch or lift up both legs. If present, ALWAYS assess hips for fixed flexion
- Ask patient to straight leg raise (SLR) (check for quadriceps lag)
- Look for posterior tibial sag (look from the side with the knee at 90˚ of flexion). The anterior tibial surface normally lies 1 cm anterior to the distal end of the femur. Double-check with the card test or palpate a step-off at the anteromedial joint line - the medial tibia should sit 1cm anterior to the medial femoral condyle.
- Palpation; move the knee to 70–80° flexion and check for tenderness on the joint line, collateral ligaments, femoral condyles, tibial tuberosity, patellar tendon, patella, retropatella, quadriceps insertion

Move

- Extensor mechanism integrity assessed by active SLR
- Check active and passive knee flexion/extension and compare to opposite side
- Check collateral ligaments in 20–30° flexion
- Anterior and posterior drawer tests

  Anterior drawer (always in comparison to opposite side)

  Grade I: 0–5 mm translation
  Grade II: 6–10 mm translation
  Grade III: 11–15 mm translation

  Posterior drawer (always in comparison to opposite side)

  Grade I: 0–5 mm in posterior translation (greater than the uninvolved side)

Grade II: Anterior surface of the tibia and femoral condyles are flush (>5–10 mm of relative posterior translation)
Grade III: Tibia can be translated posterior to the anterior femoral condyles (>1 cm)

- Lachman's test and pivot shift

  Lachman's test

  Grade I: 0–5 mm translation
  Grade II: 6–10 mm translation
  Grade III: 11–15 mm translation

  Pivot shift test

  Grade I: Pivot glide
  Grade II: Pivot shift
  Grade III: Clunk/explosive pivot (gross shift after momentary locking)

- McMurray test if meniscal injury is suspected

  Hip and knee flexed 90°. Apply axial compression while internally and externally rotating the tibia whilst extending knee. Reproduction of pain and/or clicking indicates meniscal tear
- Load the medial and lateral compartments and check for crepitus and pain
- Dial test in prone position if you suspect posterolateral corner/posterior cruciate ligament (PCL) injury

  Externally rotate the knee at 30° and 90° flexion: >10° difference in external rotation between sides is abnormal. Abnormal rotation at 30° suggests posterolateral corner injury. Abnormal rotation at 90° suggests combined posterolateral corner (PLC) and PCL injuries

Short case 1: Chronic patella instability

EXAMINER: This is a 15-year-old girl who sustained an injury to his right knee. Examine her knee for instability.

CANDIDATE: I examined patella tracking, Q angle, J tracking, tilt, translation. Brief discussion on potential anatomical abnormalities causing instability (high Q angle resulting in lateral vector of quads pull, deficient MPFL and trochlea and any rotational abnormalities in the limb).

I would normally start my examination by asking the patient to walk.

Short case 2: Patella hypoplasia with instability. Previous surgery to knee

EXAMINER: This young girl is complaining of left knee pain. Would you examine her knee?

CANDIDATE: There was an obvious surgical scar over the lateral aspect of her knee. I didn't initially comment on this as it seemed so obvious but the examiners had to prompt me making me look a bit stupid and I had to quickly recover my composure. I went straight for examining the patella femoral joint which was instinctively the right decision. The patella was dislocated laterally when the knee was flexed to 90°m, which I commented on. The patella reduced with knee extension. There was quite a big meal made of how I examined for a knee effusion. The examiners weren't happy with my technique but I didn't think it was that bad. I didn't argue with them but stood my ground a little.

The examiners asked me what surgery I thought she had had. I mentioned medial patellofemoral ligament (MPFL) reconstruction.

They asked if it had worked and I said no.

The examiners then asked what I would do next. I mumbled something about it being a difficult case and I would refer it to an experienced knee surgeon *(Pass – 6: I think I must have just scraped through.)*

Patellofemoral joint examination

You may be asked by examiner specifically to 'test the patella-femoral joint' during a short case, or else this should be at the end of your normal knee examination routine.

- Check for hypermobility syndrome (Beighton's score)
- Assess lower limb alignment and rotation
- Sit the patient on edge of bed and check patellar tracking through range of movement (ROM), J sign (abrupt lateral deviation of patella when nearing terminal extension when moving knee from flexion) and feel for crepitus
- In extension with leg relaxed
  - Feel and measure the quadriceps bulk (10 cm above superior pole of patella)
  - Assess for tenderness around the patella
  - Patellar tilt test – Holding patella between thumb and fingers, attempt to tilt patella by elevating lateral edge and lowering medial edge. Normal range is 0–20°. If you cannot get the patella to a horizontal position (<0°), this implies a tight lateral retinaculum
- In 30° flexion with muscles relaxed: Please note that the Q angle could be measured in many different ways. In supine, sitting or standing with muscles contacted or relaxed
  - Measure Q angle (quadriceps pull angle) – The angle between anterior superior iliac spine (ASIS), centre of patella and tibial tuberosity. <15° is normal. (Ask patient to place finger vertically on ASIS to aid calculation of angle)
  - Patellar glide – Divide patella into imaginary quarters, grasp patella between thumb and fingers and assess lateral mobility compared to the normal side (lateral shift of <25% width of patella is normal, >25% implies deficient medial restraints ie. medial patellofemoral ligament (MPFL))

- Patellar apprehension test (ask the examiners if they want you to proceed as this can be painful)
- Patellar grind test and Clarke's test[a] are best left to the end as they may be painful
- Ober's test to check for iliotibial band tightness

Knee examination: Quick reference

Introduction, ask about pain
Scan room for patient aids/accessories
Look at knee while standing (from front, side, back)
Comment on scars, alignment, posture, muscle wasting

Assess gait. Comment if obvious varus or valgus thrust
Lie on couch, look closer for scars/quadriceps wasting/other abnormalities
Assess for effusion and temperature change
Palpate joint (easiest at 90°) for tenderness
Assess range of motion of knee (active and passive)
Ensure to examine hip at same time and demonstrate painfree hip motion

Assess ligament stability (anterior cruciate ligament, ACL; posterior cruciate ligament, PCL; lateral collateral ligament, LCL; MCL, medial collateral ligament)
Special tests based on the above findings into either
1. Ligaments(dial, quads active)
2. Meniscal pathology(McMurray's)
3. Assess patella–femoral joint(patella grind test/patella apprehension test)

Offer examination of spine, ankles, neurovascular status of limb

Investigations (x-ray, MRI)

PCL–quadriceps active test

The patient is supine with knee flexed to 80–90° in the drawer test position. While the examiner holds down the foot the patient is asked to attempt to lift their foot off the table. Contraction of the quadriceps in a PCL-deficient knee results in an anterior shift of the proximal tibia[1].

ACL deficiency

ACL-deficient knees might be found in the short cases or possibly a combined ligament injury in the intermediate cases. In an exam in the ACL-deficient knee setting, the anterior drawer and Lachman's tests are usually glaringly obvious. There is no subtleness about these test signs unlike in everyday clinic where they are not always obvious.

[a] Controversial – Perhaps safer to mention to the examiners rather than perform as this will hurt the patient.

Memorandum

'This patient is a young, typically male, sporty type of person in shorts. *(They usually have obvious quadriceps wasting with possible medial and lateral arthroscopic portal scars.)* The patient has a normal gait and knee motion, possibly minor effusion and no specific areas of tenderness in the knee. The anterior drawer test and Lachman's test revealed an increase in translation of the tibia on the femur compared to the opposite side and no firm endpoint was felt. *(When there is an increase in translation always think ACL/PCL or both.)* The pivot shift test was positive for anterior cruciate deficiency *(describe what you are doing as you are doing it)*. A pivot shift demonstrates a non-functioning ACL.'

History

Important details to elicit from the patient:

- Nature of injury (~70% are non-contact, typically during sudden change of direction with the leg planted and valgus, external-rotation or hyperextension)
- Effusion/hemarthrosis (usually large, within a few hours of injury)
- Further instability episodes (with risk of further meniscal/chondral damage)
- Treatment so far
- Sporting activity level and plans to continue sports

Examination

Clinical examination **MAY** reveal:

- Antalgic gait
- Quadraceps wasting
- Effusion
- Joint line tenderness (consider associated meniscal pathology)
- Positive anterior drawer, Lachman's and pivot shift tests
- Assess MCL, LCL, PCL, posterolateral corner

Pivot shift analysis

This can be quite a painful test and at best can only be performed on one occasion, as the patient will thereafter tense up. It is best performed under general anaesthetic (GA). Suggest performing the test to the examiners but they may ask you to omit it for the above reasons.

With the leg extended there is an anterior subluxation of the tibia on the femur. Slight knee flexion with a valgus stress and axial load to the knee causes the anteriorly subluxed knee to reduce spontaneously into its normal position with respect to the femur with a sudden visible jump or shift at 20–30° flexion. The iliotibial band (ITB) plays an important role in the reduction of the tibia as its pull moves posterior to the axis of flexion. The medial collateral ligament, on the other hand, bears the axis of rotation of the pivot shift; therefore, it should be kept tight by applying valgus force.

Matsumoto, in cadaveric knees, showed that sectioning of the ITB in the ACL-deficient knee diminished pivot shift but

not internal rotation, and sectioning of the medial collateral ligament in the ACL-deficient knee diminished both pivot shift and the degree of internal rotation[2]. However, anterolateral ligament injury accentuates pivot shift.

Requirements for a pivot: Intact MCL to pivot around, ITB to reduce knee upon flexion and no fixed flexion deformity.

The pathological motion elicited by the pivot shift is graded as:

Grade I = Glide
Grade II = Clunk
Grade III = Gross clunk with locking. Explosive pivot

Discussion

Indications for ACL reconstruction

- Symptomatic instability following adequate rehabilitation. Frobell et al.[3] randomised patients to either reconstruction or rehab. and delayed reconstruction – No early difference in outcome but lower activity scores and higher meniscectomy rate in rehab. patients
- Consider early reconstruction in young patients who wish to continue participating in high level pivoting sports or high demand occupations. Sri-Ram et al.[4] identified that delay in reconstruction of >5 months in younger patients results in a higher incidence of medial meniscal tears and/or chondral damage
- Early reconstruction for ACL in association with repairable meniscus tear
- Early reconstruction when associated with high-grade other ligament injury

Technique – Hamstrings vs patella tendon

Currently there is no evidence to suggest one is better than the other with most studies reporting similar function and stability with either graft type.

- Patella tendon – Bone to bone healing is quicker. Higher incidence of kneeling pain
- Hamstrings – Possibly slower healing with risk of tunnel widening in early rehab protocols

Surgical complications

- Stiffness/arthrofibrosis – Often due to reconstruction before the knee has settled. Can be avoided by waiting until the swelling has settled and normal range of motion has been regained.
- Graft failure (3–5%) – May be due to fixation failure, poor tunnel position, graft impingement in femoral notch, poor rehabilitation
- Infection, deep vein thrombosis (DVT), numbness (injury to infrapatellar branch of saphenous nerve)

Be prepared to comment on postoperative radiographs of ACL reconstruction (Figure 12.1). Aim for posterior femoral tunnel placement >80% along Blumensaat's line with oblique, not vertical tunnel. Aim for tibial tunnel 40–50% from the front of the tibial plateau on a lateral x-ray and slightly medial on AP (also 40–50% from medial plateau margin)[5].

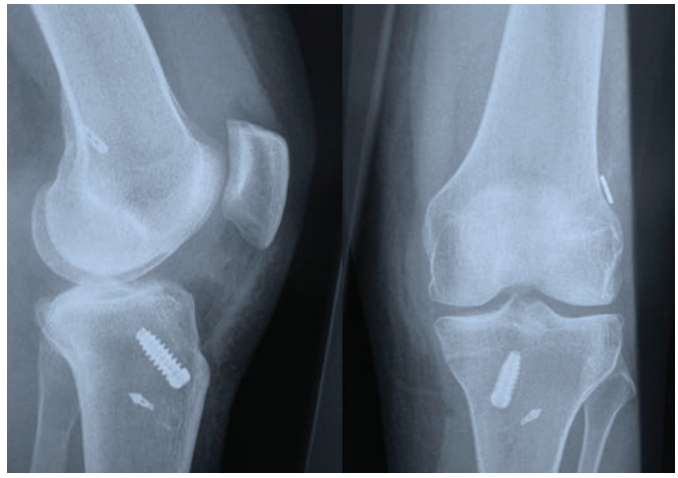

Figure 12.1 AP and lateral radiographs of ACL reconstruction. The graft is suspended in the femur using an endobutten (Smith & Nephew) and fixed with an RCI screw (Smith & Nephew) and PushLock® SP (Arthrex) in the tibia

BEWARE: A PCL injury may have a false positive anterior drawer and Lachman's (tibia sits in a subluxed position posterior to femur; these tests will reduce the tibia to a normal position, appearing to indicate ACL deficiency). ALWAYS listen to the history (PCL injury typically occurs with anterior blow to shin with planted foot), assess femoral–tibial step off to determine resting position of tibia and assess posterior drawer. MRI will also be helpful in these cases, but mixing up an ACL with a PCL injury means you generally fail that particular short case as you have been unable to correctly interprete your clinical exam findings.

Combined ACL and PCL injuries are rare – Often due to knee dislocation with higher energy injury and associated with LCL or MCL injury. Follow routine early management vascular monitoring, reduction and stabilisation. Most surgeons prefer early surgical treatment (within 3 weeks from injury) with ACL and PCL reconstruction and repair or reconstruction of LCL if present. With a combined ACL and PCL injury you are less likely to fail the case if you only diagnose the ACL injury but it doesn't look very clever missing the PCL injury!

Short case 1

EXAMINER: This is a 20-year-old male who sustained an injury to his right knee 1 year ago. Examine his knee for instability.

CANDIDATE: I would normally start my examination by asking the patient to walk.

EXAMINER: Don't bother, just examine him on the couch.

CANDIDATE: Mentioned quadriceps wasting. Knee flexed to 90°, negative sag, normal step-offs. The anterior drawer test was positive and there was a soft endpoint on Lachman's test. Varus and valgus stressing the knee at 0° and 30° of flexion was normal. The candidate continued by asking the patient to lie prone to

demonstrate dial test at 30° and 90° (external rotation was symmetrical).

EXAMINER: How do you manage an ACL-deficient knee?

It was agreed that the role of an MRI scan should not be routine but only used to confirm findings following an equivocal pivot shift test or to identify any associated meniscal injury. Noyes' 'rule of thirds'. Physiotherapy initially and, if this is unsuccessful, then consider ACL reconstruction.

Intermediate case 1: 38-year-old man sitting on a couch

EXAMINER: This patient is 38 years old injured his knee 9 months ago. Now he is complaining of pain and instability. Would you like to check his knee ligaments?

CANDIDATE: Asked the patient to walk and then examined him on the couch. Told the examiners that the patient has ACL and MCL laxity.

EXAMINER: Demonstrate Lachman's test and pivot shift.

CANDIDATE: Showed both tests.

EXAMINER: What else would you check for?

CANDIDATE: Medial joint line tenderness.

EXAMINER: What is it called when you have all thee injuries together?

CANDIDATE: O'Donoghue's triad.

Discussed further investigation with MRI (not obligatory but used to confirm findings).

Discussed treatment options – Noyes' 'rule of thirds' – One third of patients would compensate well with conservative treatment; one third would avoid symptoms of instability through 'modification or substitution' of activities; and one third would do poorly and require reconstructive surgery[12]. Some surgeons are now more aggressive with early reconstruction in active patients who are keen to continue in pivoting sports, particularly considering the risk of developing further chondral or meniscal damage if remaining active on an unstable knee.

Intermediate case 2: 21-year-old male very sporty with knee injury

History – 21-year-old sports man (not high level but keen to keep playing rugby/soccer/cricket). Injury 5 months ago, physio. Given way since (got the impression of poorly compliant patient).

Examination – Lateral joint line mildly tender, mild effusion, full ROM but pain deep flexion. Ant. drawer, Lachman's +ve. Demonstrate pivot (difficult and pt guarding/resistant). Examine other ligaments.

Discussion – MRI to determine if other pathology (high risk due to continued playing/giving way and lateral pain). Single slice T2 sagital MRI – I think lateral meniscus – Posterior horn tear. Symptomatic instability in active person – Discussion about ACL recon. Surgical options (hamstrings vs Pat tendon).

Asked – What would I do if patient presented with acute injury with swollen, stiff knee and locked bucket handle tear meniscsus/ACL tear? Two options are acute reconstruction and meniscal repair or isolated meniscal repair protecting the knee with a brace, mobilization and then performing a delayed ACL reconstruction when the knee had settled. Many surgeons would now perform a single procedure having warned the patient of the risk of stiffness and possible need for subsequent MUA/arthrolysis.

PCL injury

Memorandum

'On examination there is a young athletic gentleman of average height and build. What we can see from the front is fairly marked quadriceps muscle wasting of his right knee. There appear to be well-healed arthroscopic portal scars over the medial and lateral joint lines.

On inspection of his gait he demonstrates a varus thrust of the right knee during walking.

Examination supine reveals a small effusion present in this knee. There is no fixed flexion deformity. There is a range of movement of 0–110° flexion compared to 0–130° flexion on the opposite, normal side. Palpation reveals no specific areas of tenderness. On flexing his knees to 90° there appears to be a posterior sag sign. The tibial tubercle appears less prominent than usual whilst the patella appears more prominent than normal. Placing a flat card over the front of the right knee reveals a subtle concavity present with a gap between the card and the front of the knee. Also, the step-off sign is negative (the tibial plateau is flush with the medial femoral condyle), suggesting PCL disruption.

The quadriceps active drawer sign was positive for PCL disruption. The tibia moved anteriorly when the quadriceps contracted, actively extending the knee from a flexed position.'

Discussion

- Acute PCL injury – Non-operative management with bracing and quadriceps rehabilitation is standard practise
- Surgical reconstruction for symptomatic chronic PCL injuries – Patients who suffer recurrent instability may benefit from PCL reconstruction. A systematic review reported reconstruction improved stability by 1 grade with 75% returning to normal or near normal activities but reconstruction does not prevent development of OA[13]
- Acute bony avulsions – Should always be repaired when possible (via direct posterior approach or athroscopic with 70° scope and posteromedial and lateral portals)
- Acute combined injuries – Usually in combination with posterolateral corner injury. Usually require early repair and reconstruction in active patients
- Debate about the long-term history of the PCL-deficient knee: Studies suggest that there can be significant activity-related pain and possibly degenerative changes, especially in the anterior (due to lack of restraint of posterior translation) and medial compartments[14,15]

Examination corner

Intermediate case 1: Gentleman sitting on a chair in hospital gown

EXAMINER: Mr Jones is 63 years old and has a knee problem. You have 5 minutes to obtain a history.

CANDIDATE: Introduced himself and started asking about the knee pain, its site, nature, duration, severity, radiation, aggravating factors and its effect on the patient's sleep and walking distance.

EXAMINER: What else would you like to do?

CANDIDATE: I would like to examine Mr Jones' knee.

EXAMINER: Please do.

CANDIDATE: Started by walking the patient, commenting on his gait, diffuse swelling, previous scars and deformity. Then examined the knee, pointing out that there are signs of arthritis.

EXAMINER: What else would you like to examine?

CANDIDATE: Hip and spine.

EXAMINER: Anything else?

CANDIDATE: Had to be prompted to say peripheral pulses. (Losing points.)

EXAMINER: This gentleman is 63 years old and has 15° varus deformity in the knee and restricted range of movement. What is your management plan?

CANDIDATE: I would offer him total knee replacement.

EXAMINER: Even if his pain is mild!

CANDIDATE: Tries to change the plan, which the examiners did not like.

OUTCOME: Failed.

Intermediate case 2: 55-year-old man lying on a couch

EXAMINER: Examine this gentleman's knee.

CANDIDATE: Started by commenting on large anterior scar and wanting to test effusion.

EXAMINER: How do you do a patellar tap?

CANDIDATE: While trying to demonstrate it he realized that the patient has had patellaectomy.

EXAMINER: Examine the main ligaments.

CANDIDATE: Demonstrated posterior sag and loss of step-off.

EXAMINER: When PCL is injured what else do you need to examine?

CANDIDATE: Dial test at 30° and 90°.

EXAMINER: What is your management plan?

CANDIDATE: This gentleman has post-traumatic arthritis. He had patellaectomy for comminuted patellar fracture and also has a PCL-deficient knee. His pain is mild to moderate at this stage. I would, therefore, treat him conservatively as long as possible before offering him TKR.

EXAMINER: What type of knee replacement would you use?

CANDIDATE: I would use PS type TKR in patients with patellaectomy and/or PCL deficiency.

Intermediate case 3: 81-year-old man with bilateral varus osteoarthritis of the knees

Gross deformity present with associated fixed flexion deformity of approximately 20° in each knee. Thorough history, including recreation, social, stairs in house, etc.

Demonstration of:

- Varus/valgus instability
- Anterior drawer test
- Lachman's test

The usual questions were asked on possible spinal/vascular/hip aetiology for knee pain.

Asked to take informed consent for TKA in front of the examiners.

Discussion

- Shown radiographs of both knees
- What type of TKA would you do?
- What problems do you anticipate (patellar eversion, rectus snip, etc)
- How do you sequentially release the medial structures?
- How do you correct for a fixed flexion deformity intraoperatively?
- How do you recreate the joint line in TKA?

Intermediate case 4: 67-year-old female with severe varus osteoarthritis of knees with fixed flexion deformity

Typical history of painful arthritis limiting activities and quality of life. No major medical problems. Tried physio, steroid injections. Now greatly affecting life with severe pain and progressive deformity.

Examination: ROM, deformity (was a valgus knee), previous TKR other side, hip above arthritic knee also slightly painful (remember to examine joint above), no evidence of RA.

Discussion

EXAMINER: So here are some x-rays. Talk me through your thoughts.

CANDIDATE: AP radiograph of knee showing complete loss of lateral joint space and valgus angulation of >20°.

EXAMINER: What do you mean valgus angle?

CANDIDATE: The femoro-tibial anatomical angle is typically around 6° of valgus. It is greatly increased in this patient due to the arthritis and there appears to be some lateral bone loss.

EXAMINER: Yes. So what are you thinking about for treatment?

CANDIDATE: Well, options for treatment include non-surgical modalities and . . .

EXAMINER: I think its pretty obvious she's exhausted non-surgical modalities. (Obviously keen to move on to surgical discussion.)

CANDIDATE: Yes. The only real surgical option would be total knee replacement.

EXAMINER: So tell me more . . . (getting a bit pushy) What difficulties do you anticipate?

CANDIDATE: For exposure I would use a medial parapatellar approach. I would perform my distal femoral cut using a 5° block. The lateral femoral condyle may be deficient and so I would concentrate on getting my femoral rotation correct.

EXAMINER: How would you measure this?

CANDIDATE: I would estimate the correct axis using Whiteside's line and the intercondylar axis.

EXAMINER: Yes, typically the correct rotation can be achieved using 3° of external rotation on the cutting block. What else concerns you?

CANDIDATE: Ligament balancing can be problematic.

EXAMINER: So tell me about the releases . . .

CANDIDATE: Lateral osteophytes, capsule and PCL are released first. For tightness in extension, I would perform release of ITB and in flexion, I'd release popliteus.

EXAMINER: You go to the ward postoperatively and they patient has a foot drop. Have you cut the nerve?

CANDIDATE: This is often due to a large correction and the majority will resolve with release of bandages and elevation.

Bell rings

(Also consider patella maltracking due to contracted lateral retinaculum – Possible need to release.)

(Examiner could have gone into discussion of anatomical vs mechanical axis.)

Intermediate case 5: 53-year-old male with medial osteoarthritis and previous HTO

History: 53-year-old male, previous open meniscectomy (around 30 years ago) then HTO (>10 years ago). Now severe pain. Works as a plumber.

Past medical history: gout, hypertension

Examination: Obese, mature open medial meniscectomy scar, lateral scar (from closing wedge osteotomy), osteophytes, 15° FFD, fixed varus 5°. Hip OK

Discussion

EXAMINER: What is your next step?

CANDIDATE: I would like to see weight-bearing AP, lateral and skyline x-rays (shown a poor quality AP radiograph). This is an AP radiograph showing medial osteoarthritis with lateral osteophytes and joint space narrowing. There are staples in situ from the previous closing wedge osteotomy.

EXAMINER: Yes, treatment options?

CANDIDATE: The patient should exhaust non-operative methods due to his age. If the pain is too severe that it is greatly affecting everything he does then he may need to proceed to total knee replacement although I would prefer to hold off as long as possible.

EXAMINER: Sadly he cannot carry on like this. Tell me how you would plan his knee replacement.

CANDIDATE: My concerns are the previous scars and leaving a skin bridge. I would curve my incision into the medial scar distally rather than go midline. The staples may be an issue for the tibial cut.

EXAMINER: When would you take them out?

CANDIDATE: At the time of surgery? (more of a question than an answer)

EXAMINER: And leave yourself with a big hole in the tibia? I would normally take them out 6–8 weeks prior to the surgery, but I'm sure they could come out on the day too. Do you expect trouble with the approach?

CANDIDATE: Yes, the patient may have patella baja leading to difficulty with eversion and the approach may be challenging.

EXAMINER: How do you measure patella height?

CANDIDATE: Measurements are taken from lateral radiographs. Insall–Sulvati compares the length of the patella to the length of the tendon from distal pole of patella to tibial tubercle. Erm . . .

EXAMINER: (losing interest and aware of time) Yes, there is also the Blackburne–Peel ratio. Tell me about your tibial cut.

CANDIDATE: This can be difficult as both joint heights have been altered. I believe you would typically reference off the medial side.

EXAMINER: Are you aware of any differences in knee replacements after osteotomy?

CANDIDATE: I believe that, although the operation is more challenging, the results are similar to primary knee replacements.

Bell rings

Intermediate case 6: **69-year-old woman with painful total knee replacement**

History: Total knee replacement 2 years ago. Prolonged wound ooze (warfarin for AF), 1× wound washout but no prolonged antibiotics, not sure of poly-exchange (vague historian). Knee 'never felt right'. Now always swollen and painful. Smoker, diabetic.

Examination: large effusion, limited ROM, mildly warm, no erythema, no sinuses (reminded to look all around knee by examiner), painful knee to move. Hip OK. Evidence varicose veins (eczema, discoloration), poorly palpable pulses and diminished sensation feet.

Discussion: high suspicion of infection. Investigate with CRP, ESR, WCC. X-rays – No obvious loosening/lysis. Implant slight varus on tibial side. Discussed use of bone scan at 2 years postop. Usefulness of aspiration/synovial biopsy. Probably two-stage revision (discussed single-stage). Risks of amputation discussed with the patient.

Short case 1: A 41-year-old woman with post-traumatic osteoarthritic left knee

- Varus mal-alignment
- Antalgic gait
- No fixed flexion deformity, effusion or patellofemoral (PF) joint tenderness
- Normal range of movement
- Tenderness limited to medial joint line
- Varus was correctable
- Cruciates and collaterals were stable

Management

'I suggested conservative management and then, if there was significant severe symptomatic deterioration, either osteotomy or unicompartmental arthroplasty (UKA).'

Intermediate case 7

'This was a gift. The patient was an otherwise fit and healthy 79-year-old woman with predominantly right-sided osteoarthritis of the knee. I took a full history. I examined her back and hips thoroughly and noted that she had bilateral varus knees with a mild fixed flexion deformity on the right. I was asked to demonstrate the Thomas' test and correctable varus. The second examiner discussed investigation, long leg films, non-operative options, evidence for the benefit of arthroscopic washout and UKA. I was asked what type of knee prosthesis I would offer her, which led to a discussion of PCL retaining vs posterior stabilised total knee replacement.'

Intermediate case 8: 37-year-old male with chronic knee pain[10]

History

Patient is 37 years old. Knee pain limiting walking, stairs, crouching. Taking multiple painkillers every day. Done 'some physio' few years ago. Played a lot of sport when young – Multiple minor 'sprains' of the knee. No major injury. 2 previous arthroscopies – Told ACL was torn but continued sport. Works as a manual labourer – Keen to continue. No past medical history.

Examination

Effusion (ensure thorough examination for small and moderate effusion), stands with varus alignment around 5°, medial tenderness and crepitus, patella–femoral crepitus, palpable medial osteophytes, MCL stable, varus only partially correctible, ACL laxity.

Discussion

'What is your management of a young, active person with knee osteoarthritis who is not yet ready for arthroplasty?'

Discussed exhausting non-surgical methods first with physiotherapy, off-loading brace, activity modification (reducing impact activities, more muscle strength, control and endurance work such as static bike) and weight-loss. Role of injections, especially to provide a short-term 'buffer' for a special event or holiday.

Surgical options: Arthroscopy only if mechanical symptoms, and patient aware that it will not necessarily improve arthritic pain or prevent progression of symptoms. Will also allow assessment of rest of joint if considering further surgery. If pain severe enough, HTO (medial opening – May require bone-graft but can be used to tension MCL if necessary; lateral closing – More stable initially), provided the lateral compartment is intact – Determine this with MRI and/or arthroscopy. Significant lateral articular damage will lead to poor outcome from osteotomy. There is better long-term data for lateral closing osteotomy but numerous studies report improvement in pain and function with good mid- to long-term survival for both types of osteotomy[16].

There is no current evidence for stem-cell or platelet-rich plasma (PRP) injections.

If all options have been exhausted, the patient must decide whether his symptoms are affecting his quality of life enough to warrant the risks of arthroplasty. He should consider changing to a less labour-intensive job if possible due to the higher risk of failure of a knee replacement placed under high loads.

CANDIDATE DEBRIEF: I jumped in with initially using NSAIDs for pain control. The examiners weren't happy at all with this. They then grilled me about the WHO pain ladder. A silly simple mistake but a BIG issue was made of it.

Osteoarthritis (OA) of the knee

This can be a short or intermediate case. There is little room for any error. The candidate would be expected to be very familiar with this type of case from clinic.

Memorandum

'On examination from the front, with the patient adopting a weight-bearing stance, I see that this is an elderly gentleman of average height and build. There is a bilateral varus deformity of both knees. There are no scars, no skin discoloration or varicose veins. He walks in a slow moving manner suggestive of loading his knees on the medial compartment.'

'There is no effusion present in either knee but generalized synovial thickening. He has a fixed flexion deformity in both knees of 10° and demonstrates a range of movement from 10° to 70° flexion.'

'The knees are tender globally, with osteophytes over the joint lines. There is a grating sound with crepitus when the knees are moved. Both knees are stable when the anterior drawer, posterior drawer and Lachman's test are performed, with a firm endpoint noted. The varus deformity is not fully correctable.'

'Hips have full, pain-free ranges of motion at 90° and in extension. The distal circulation is good, with dorsalis pedis and tibialis posterior pulses strongly palpable, good capillary refill, no dystrophic changes in the nails and no distal hair loss. Likewise, sensation to fine touch is normal.'

History

Important details to elicit from the patient:

- Age and activities
- Nature of pain and disability caused by it (mobility, ability to self-care, get around house/shops, night pain)
- Walking distance, functional difficulties (ability on stairs, crouching, uneven ground)
- Mechanical symptoms (catching, locking, swelling, instability)
- Treatment so far (analgesia, physiotherapy, joint injections, arthroscopy/other surgery)
- Medical history (cardiac, pulmonary, diabetes, previous DVT/pulmonary embolism (PE))
- Social history (housing status, partner, stairs, support)

Examination

Clinical examination may reveal:

- Varus/valgus deformity on standing

- Scars of previous surgery
- Antalgic gait
- Quadraceps wasting
- Effusion
- Joint line tenderness and osteophytes
- Limited range of movement (ROM), possible fixed flexion deformity, limited flexion (assess hip ROM)
- Crepitus (particularly patella–femoral)
- Correctability of varus/valgus deformity (fully, partially correctible or not)
- Examination of hip
- Examination of spine (SLR)
- Examination of distal pulses, neurological examination

Discussion

Treatment options

- Analgesia, weight loss, modify life-style, physiotherapy
- Intra-articular injections (a Cochrane review in 2006 concludes good evidence of short-term benefit from steroid injection and response to hyaluronan/hylan may be more durable, with few side-effects[6]
- Arthroscopy – Increasing evidence this will not be beneficial to patients (Kirkley et al. randomised 90 patients to physiotherapy or arthroscopy in moderate/severe OA, showing no difference in outcome[7], Herrlin et al. randomised 96 patients with degenerate medial meniscal tears to physiotherapy or arthroscopic debridement with no difference in outcomes, although one-third of the exercise group patients eventually required arthroscopy for continued pain[8])
- Osteotomy – High tibial for varus, distal femoral for valgus knees. Typically reserved for younger, higher demand patients. Finnish Registry data of 3190 knees suggests high tibial osteotomy (HTO) survival at 5 years 89%, and 73% at 10 years using arthroplasty as the endpoint and, similarly, a systematic review of femoral osteotomy suggests a 10-year survival of between 64% and 82%[9]

Knee arthroplasty

This may be offered to a patient whose quality of life is significantly affected by pain despite other efforts to manage symptoms. The patient must be fully informed of risks and complications of surgery.

Total knee arthroplasty (TKA)

- Reliably relieves pain and improves function in the majority of patients. The National Joint Registry (NJR) in 2014 reported that cemented TKA have a 10-year cumulative risk of revision of 3.3% and uncemented implants 4.5%
- Goals of surgery – Improve joint pain and function whilst restoring mechanical axis, achieving balanced ligaments, preserving/restoring the joint line and a normal Q angle
- Technical considerations from preoperative x-rays – Alignment (varus/valgus), extra-articular deformity, bone loss, patella baja (making exposure difficult)
- Joint line restoration – Lies approximately 15 mm above fibular head radiographically and can be approximated from the residual meniscal roots intraoperatively (roughly two finger-breadths above tibial tubercle). Elevation of the joint line results in patella baja, lowering the joint line often occurs with over-resection of the tibia and may lead to instability in flexion. Preoperative planning is important to avoid changing the joint line, along with controlled resection of bone and cartilage equivalent only to the thickness of the implants
- Fixed flexion contracture: It is vital that the knee can achieve stable full extension by the end of the procedure. Walking with a flexion contracture increases the work of the quads and is a cause of pain and poor function in TKA. A knee with significant deformity may require an extra 1–2 mm bone resection from the distal femur. Excision of posterior osteophytes after the chamfer cuts and careful release of the capsule around the posterior edge of the femoral notch and posterior condyles will also improve extension
- Limited flexion – Flexion may be improved by down-sizing femoral implant size (to increase flexion gap) and increasing tibial slope cut
- Varus knee – ACL ± PCL excision, sequential subperiosteal medial release depending on degree of deformity (with removal of osteophytes), continue posteromedially as necessary, including semi-membranosus. Most of the time the deformity is well corrected by just pie-crusting the superfacial MCL
- Valgus knee – Results in possible contracture of ITB, LCL, popliteus, posterolateral capsule, lateral head gastrocnemius, lateral patellar retinaculum with stretching of medial structures. Be aware of hypoplastic lateral femoral condyle (BEWARE – May lead to internal rotation of femoral component if posterior referencing – Check with Whiteside's line and intercondylar axis). To balance tissues, resect PCL, remove osteophytes
  - Tight in flexion – Release popliteus from femur, posterolateral capsule from tibia
  - Tight in extension – 'pie-crust' or release ITB from tibial insertion ± lateral gastrocnemius from femur ± Z-lengthening biceps tendon
  - Patellar tracking – May require lateral release. If resurfacing, place button medially
- Implant choice
  - PCL retaining (cruciate retaining (CR)). Advantages: Retains PCL proprioception, no post-wear/jump, less femoral bone resection, slightly increased congruence

(theoretically less wear). Disadvantages: More difficult balancing, deficient PCL

- PCL substituting (posterior-stabilised (PS)). Advantages: Easier to balance, more reproducible results, slightly increased constraint. Disadvantages: Post-wear/jump, elevate joint line, possible patella clunk

- NJR reports 10-year risk of revision for all causes for CR implants as 3.1% and PS as 3.6%. Choose an implant that you are familiar with as this may be asked in the discussion
- Patients with previous PCL injury or patellectomy should receive a PS implant. After patellectomy, the PCL is the only structure resisting posterior tibial translation and tends to stretch, leading to AP instability. A number of studies have reported better pain and functional outcomes in those patients receiving PS in comparison to CR implants following patellectomy
- Surgical complications: nerve or vessel injury; stiffness/arthrofibrosis (up to 10%), infection (1–2%, commonly *Staphlyococcus epidermidis* or *S. aureus*); DVT (no treatment DVT ~70%, symptomatic PE 0.5–3.0%, death ~0.2%[10]), numbness (skin lateral to the wound), poly wear and aseptic loosening

Unicompartmental knee arthroplasty (UKA)

- Reported to combine quicker rehabilitation with better ROM and function in comparison to TKA. Higher revision rate (NJR 2014 reports 12.7% probability of revision by 10 years) in comparison to TKA although lower rates observed in high-volume practices
- Must have intact ACL, fixed flexion deformity <5°, flexion >90° (probably more 110–130°), maximum varus 15° which is correctable to neutral, older age group (>60 years), lower weight <82 kg. Contraindicated in unstable knees, evidence of OA in other compartments (with risk of progression and subsequent failure) or inflammatory arthritis
- Complications (other than those seen in TKA): Bearing dislocation, other compartment OA progression, tibial component subsidence (possibly due to over-aggressive tibial resection or tibial stress fracture), patellar impingement
- Surgical complications: nerve or vessel injury, stiffness/arthrofibrosis, infection, DVT, numbness (skin lateral to the wound), poly wear, aseptic loosening

Painful knee arthroplasty[b]

It is important to detect infection. History may reveal pain-free intervals and rest or night pain. Start-up and mechanical pain may be suggestive of implant loosening. Determine the exact location of the pain and whether it is well localized or radiating. When the pain most commonly occurs, how long it lasts and any precipitating or relieving factors.

Ask for problems with wound healing, prolonged discharge, superficial wound infections, etc. If the patient was free of pain initially following TKR and developed pain months to years later think component loosening, late ligamentous instability or haematogenous based infection. If the patient was never pain free following TKA think deep infection, instability, prosthetic mal-alignment or non-articular causes.

During examination look for hallmarks of infection, including sinuses, effusion, warmth and generalized tenderness. Inspect the skin for erythema or warmth. The knee should be examined for alignment, stability, range of motion and the presence of an effusion, synovitis or crepitus. The stability of the knee to varus-valgus and anterior-posterior stress should be tested in flexion and extension. Posterior cruciate ligament (PCL) dysfunction can be assessed with a positive posterior sag sign or quadriceps active test. The knee should be palpated for areas or points of tenderness which may represent tendonitis, bursitis or cutaneous neuroma. Assess patella tracking as this may suggest an issue with component mal-alignment. Examine gait for limp or varus thrust suggestive of mal-alignment or ligamentous instability.

Examine the spine and hip to exclude potential sources of referred pain. Check neurovascular status.

Surgical diagnosis (intrinsic):

- Prosthetic loosening and failure
- Infection
- Patellofemoral tracking problems
- Instability
- Recurrent intra-articular soft-tissue impingement/component overhang

Non-surgical diagnoses (extrinsic):

- Referred pain – Hip or back
- Reflex sympathetic dystrophy
- Bursitis or tendonitis – Pes anserine/patella/popliteal bursitis
- Persistent gout or pseudogout
- Neurovascular problems
- Expectation/Result mismatch – Multiply operated knee or unrealistic expectations
- Psychiatric disorders and depression

X-rays may be normal. Serial comparison is important for long-term monitoring for loosening.

A bone scan is very sensitive but not specific to one pathology. There may also be increased flow for upto 2 years after joint replacement surgery.

Other tests: Blood tests – ESR >30 is 80% sensitivity and specific for infection, but may be raised due to other causes. CRP >10° is 90% sensitivity and specific and has negative predictive value of 99%. Aspiration – synovial fluid white blood

[b] This is also a viva question and requires a worked-out answer beforehand as the topic is complicated.

cell counts >1700 cells/μl and a differential >69% polymorphonuclear cells should raise suspicion of infection[11]. Intraoperative gram stain has sensitivity <20% but is very specific.

Previous HTO (Figure 12.2)

Memorandum

'A lateral scar or an L-shaped lateral or a medial vertical or oblique scar suggests that the patient may have had an HTO.

Controversy remains as to whether the outcome after TKA following HTO is less successful than conventional TKA. Conversion to TKA may be technically demanding owing to difficulty with patella eversion, soft-tissue balancing and infection.'

Discussion

The goals of an HTO are to relieve pain and improve function with minimal restriction of activity, allowing heavy functional demands, often in a patient under 60 years of age.

Indications for HTO

- Medial compartment osteoarthritis with varus mal-alignment
- Typically male, <60 years old, flexion >90°, FFD <15°, vaurs <15° with no lateral subluxation and intact ACL. HTO may also be performed to protect meniscal allograft or chondral graft implantation

Long-term results of HTO tend to deteriorate with time. The Finnish Registry data of 3190 knees suggests high tibial osteotomy survival at 5 years 89%, and 73% at 10 years[17] using arthroplasty as the endpoint.

HTO medial opening wedge vs lateral closing wedge – A recent meta-analysis reported similar functional outcomes and complication rates, but opening wedge osteotomies had statistically greater posterior tibial slope, mean angle of correction and incidence of patella baja[18].

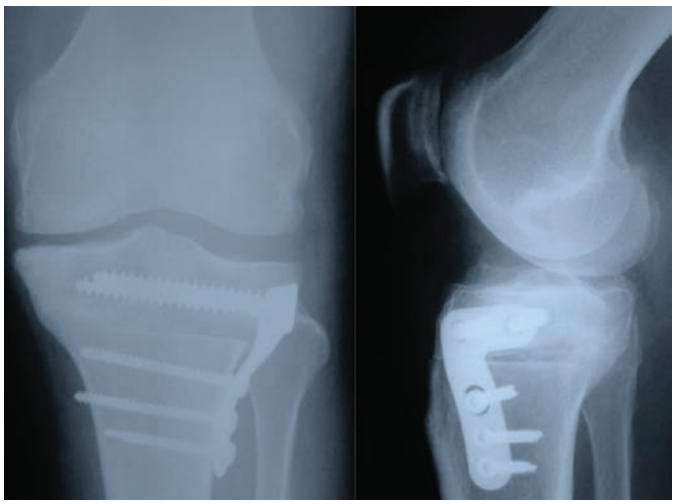

Figure 12.2 AP and lateral radiographs of a closing wedge HTO

Technical issues during arthroplasty

- Patella baja
- The need to respect a longitudinal scar: At least 3 cm should be allowed between a new midline scar and a lateral scar
- Ideally use a PCL-sacrificing implant
- Abnormal tibial slope that may require adjustment of tibial cut

Outcome of TKR following previous HTO – Recent literature reports that, although technically more demanding, TKA following HTO has similar complication rate and mid-term outcomes to primary TKA[19,20].

Pigmented villonodular synovitis

Memorandum

'The knee is the most commonly involved large joint. Two types of pigmented villonodular synovitis exist: A localized form characterized by a solitary lesion and a diffuse form aggressive in nature usually involving the entire synovial membrane. Local disease presents with mechanical symptoms such as locking and catching, whilst the diffuse type is characterized by pain, swelling, stiffness and deformity.'

Intermediate case 1: 39-year-old male with chronic knee injury

History: 39 years old. Playing social hockey – Blow from front of knee 2 years ago. Pain and swelling, settled. Knee doesn't feel right, medial pain now.

Examination: Posterior sag, no effusion, medial tenderness, posterior drawer +ve, dial –ve at 30°.

Discussion: PCL injury – Acute treatment with brace if isolated. PCL deficiency progression to medial wear. Discussion of PLC and PCL injuries (and dial test results with each) – Dial tests for increased ER of tibia (abnormal is >10° compared to the other side). If increased at 30° knee flexion – Likely isolated PLC injury. If increased at 30° and 90° knee flexion – Likely both PCL and PLC injured.

Brief discussion on multiligament injuries – Evidence supports early operative treatment over late surgery, revealing better functional scores and earlier return to work and sport[21]. This systematic review also reports that reconstruction of the posterolateral structures yields a lower failure rate than repair (9% and 37% respectively).

Valgus knee

Look for walking aids. Typically this would be a rheumatoid patient or a young patient post trauma.

Rheumatoid arthritis

The patient presents with polyarthropathy. Look for hand and wrist signs. Weight-bearing leads to marked valgus deformity

of the knee. Fullness of the knee suggests soft-tissue swelling due to a synovitis.

Discussion

- Radiographs of the knee
- Cervical spine evaluation
- Medical evaluation of the rheumatoid patient and management in a multidisciplinary team is vital. The combination of polypharmacy and the systemic effects of RA, including anaemia, pulmonary and cardiac problems, require careful management in the perioperative period
- Methotrexate (continued perioperatively), biological agents should be stopped
- Sequential release of the valgus knee as necessary
- Use a cemented PCL sacrificing (no clear evidence but reduces risk of early instability) with patella replacement (which is associated with improved outcomes in RA)[22]
- A stemmed implant may be necessary in severe valgus deformity – Bone is often softer and more constraint may be required, leading to greater forces through the implant bone interface. A stem will share these forces and protect from periprosthetic fracture

Examination corner

Short case 1: Pigmented villonodular synovitis (PVNS) of the knee

- Examine the knee for effusion and history of recurrent bleed
- Clinical differentiation of effusion from synovial thickening
- Differential diagnosis of PVNS
- PVNS: Clinical presentation, joints affected (80% knee) and management. Localized – Arthroscopic debridement, diffuse – Arthroscopic ± open synovectomy. Diffuse – Risk of recurrence. Possibly treat with concomitant radiotherapy

Examination corner

Short case 2

- Examine this man's knee
- Look at the patient (middle-aged, body-builder)
- Ask the usual questions about knee pain

Assess

- Gait: Look for varus thrust
- The degree of varus deformity. Is it correctable?
- The range of movement in the knee
- Ligament laxity
- Leg length discrepancy
- Examine the hip and foot for fixed deformity

Radiographs

- Medial compartment OA
- What are you going to do?
- Why not carry out a unicompartmental knee replacement? (Age and ACL are possible main factors to consider here.)

If the patient is <50 years old and has some articular surface preserved, in addition to the previous prerequisites, one can offer HTO. However, if the patient is older and the degenerative arthritis is severe (bone on bone), unicompartmental knee replacement would be a better choice.

Hereditary multiple exostoses

Palpable exostosis around knee. Risk of malignant transformation. Indications for investigation (increasing size or worsening pain – organize MRI) and excision (pain from impingement or compression of local structures). Inheritance pattern (autosomal dominant with almost 100% penetrance).

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Chapter 13

Foot and ankle clinical cases

Rajesh Kakwani

A significant number of the short cases in the lower limb section are generally from the foot and ankle subspecialty. These conditions are generally painless and easy to find. Although the FRCS Orth exam short cases are generally spot diagnosis, they can be sometimes tricky due to lack of opportunity to take a detailed history prior to clinical examination. The time constraint of 5 minutes for the short cases makes it essential for the candidate to practice a much focused examination technique and be slick at it. An attempt has been made to provide the reader with a general guidance for foot and ankle clinical case examination, with special tests for specific cases. The basic habits of a humble introduction to the patient, development of a rapport and hand-washing/gel application between cases would go miles towards a successful outcome to the exams. We thank and acknowledge Mr Chris Blundell, Sheffield, for allowing us to share the examination template used through the chapter.

Foot and ankle examination template summary

Have a system

Look

- Stand – Including tiptoe, range of movement (ROM), walking aids
- Walk–don't get bogged down
- Sit – Including shoes, insoles, etc

Feel

- Bony landmarks
- Tendons
- Sensation
- Pulses

Move

- Passive ROM
- Proximal to distal/distal to proximal

Foot and ankle clinical examination system

Look

Attitude of the forefoot and hindfoot – Sitting as well as standing position: cavus, planovalgus, hallux valgus, etc

Looking from the front, side and behind (If the examination room is small, it is sometimes prudent for a candidate to ask the patient to turn around rather than they look clumsy and awkward trying to jump around in a small space)

Standing tiptoe, one leg at a time, observe the heels and comment on the movement of the heel, e.g. Valgus heel going into varus on tiptoeing (Figure 13.1 a and b)

Gait: Antalgic, high stepping, externally rotated, foot progression angle, patient walking on the outer border of the foot

Deformities: Rheumatoid, curly toe, hammer toes, etc

Callosities: Locations

Scars: Location, primary/secondary healing

Footwear: Including the location of the wear of the shoe, any insoles

Walking aids: Stick, crutches, calipers, etc

Feel

Temperature: Especially in diabetic foot

Tenderness: Exact location, this needs a lot of practice to be able to accurately position the finger at the important landmarks whilst maintaining eye contact with the patient to be able to remark on any wincing. At the same time, look slick and professional. Depending on the location of the obvious pathology, you may wish to start the palpation from the forefoot working back wards or the vise-versa

Pulses

Sensations

Move

Range of movements: Ankle, subtalar, midtarsal, metatarsophalangeal

Special tests

Hallux valgus

Look

- Stand (hallux valgus/ medial bunion/pronation, hammer toes, heel valgus, pes planus)

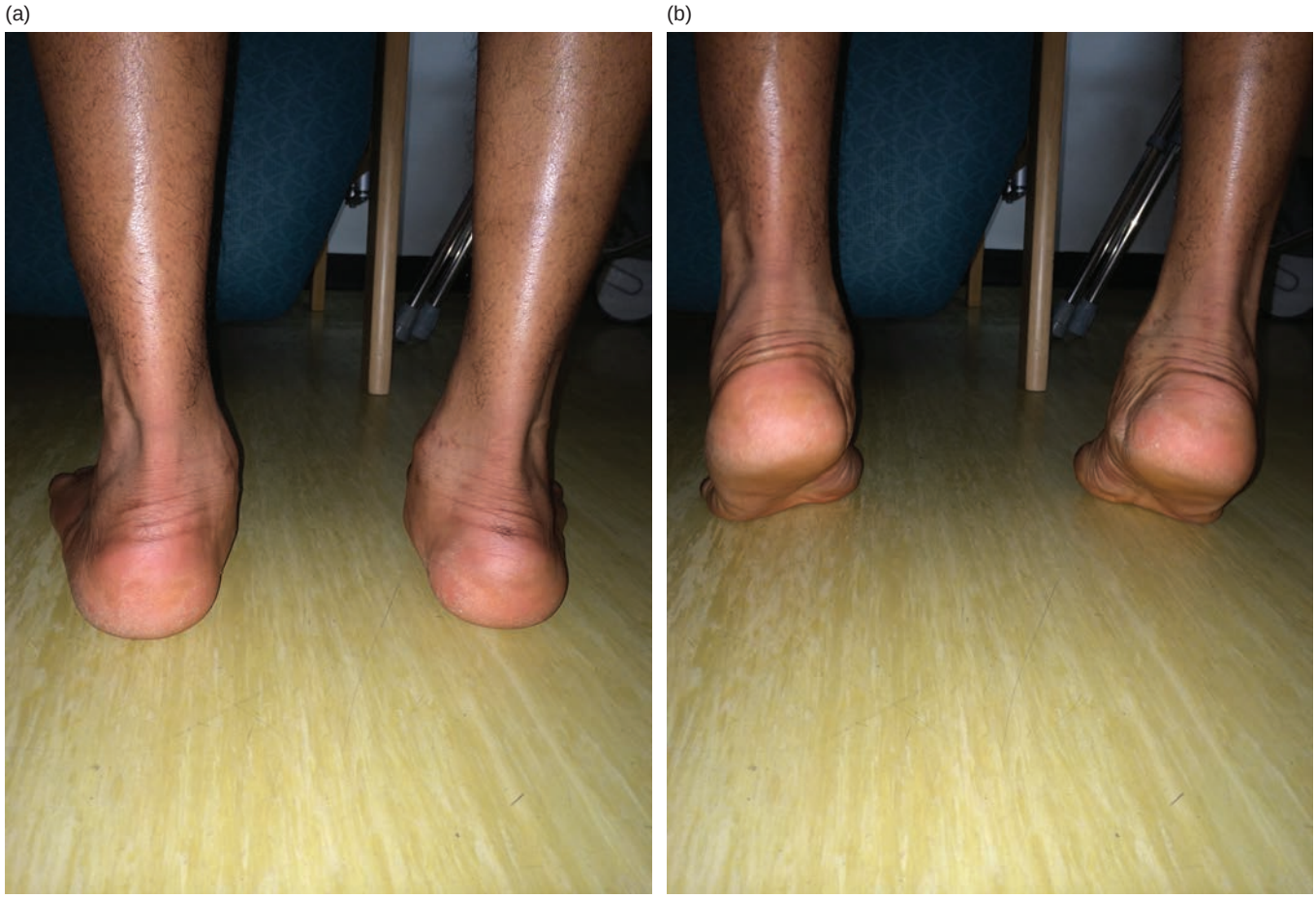

Figure 13.1 (a, b) Foot and ankle examination

- Gait (walks on outer border of the foot)
- Sit: incl shoes (wear over the bunion)

Feel

- Bony landmarks (bunion/tender first MTPJ)
- Tendons
- Sensation
- Pulses

Move

- Passive ROM (first TMT stability, pain/crepitus on MTP joint movements, mobile IP, tendo-Achilles tightness)
- Distal to proximal

Hallux valgus (Figure 13.2)

Hallux valgus is a frequently included short case in the exams, a spot diagnosis. It is a very common condition, often not painful. It is important to identify deformities that commonly coexist with hallux valgus, i.e. Hammer toes, pes planus, and gastrocnemius tightness. Essentially be comfortable describing what you see and also what you are doing when examining the big toe. Be confident describing your management and reasons behind it. Be able to roll off a list of possible surgical complications. Do not forget to mention conservative management if appropriate.

Short case 1

CANDIDATE: May I examine your feet please sir? Is there any area of soreness? If it is sore at any stage through the examination, please stop me![a]

Could I ask you to please **stand up** with feet close to each other.

On inspection of the foot and ankle from the front is a moderate degree of hallux valgus deformity of the right big toe. The first metatarsal head is prominent medially with thickening of the overlying skin, slight erythema but no ulcerations. No previous surgical scars seen (*If there is a dorso-medial scar over the MTP joint, it is important to check for sensations in the dorso-medial cutaneous nerve distribution to the big toe, which is a common iatrogenic injury, especially if the scar is a curved one*). The big toe is pronated. There is a hammer toe deformity of the second toe with a

[a] These are general courtesies that you must extend to the patient. There are no excuses for rudeness or just forgetting manners with a patient as this will score you a 4.

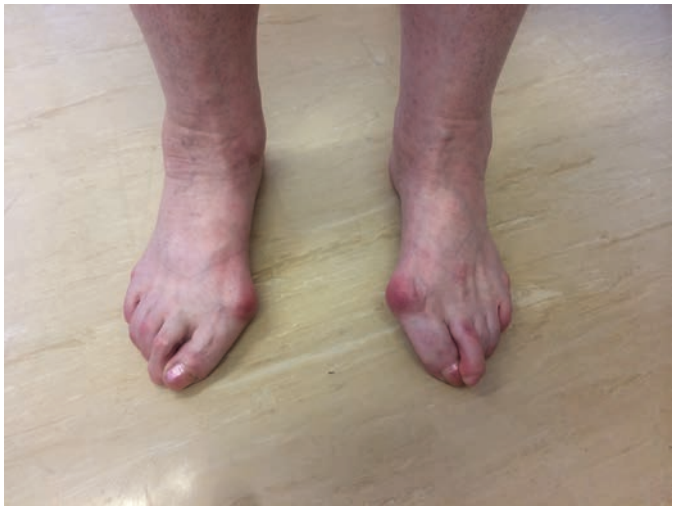

Figure 13.2 Hallux valgus

callosity over its proximal interphalangeal joint. The arches of the foot are well maintained and the heel is in slight valgus.

Could you please **stand tiptoe** for me: It is important to ascertain that there is support available for the patient if she/he loses balance. It may be a good idea to make them stand facing a wall, with fingertips touching the wall, whilst requesting them to tiptoe. The two important findings to note are:

1. The heel moves from slight valgus into varus (hindfoot is mobile)
2. Whether the big toe hyperextends at the MTP or IP joint during this manoevre

Can you please **walk** for me?

The gait may be normal (not antalgic) or have a slightly reduced big toe push-off

Can you please **sit down**/lie down on the couch?

Sole: There are callosities under the first metatarsal head as well as the medial border of the base of the big toe distal phalanx (due to the abnormal pressure caused by the pronation deformity of the toe) There are no callosities under the lesser toe metatarsal heads.

There is no tenderness over the medial prominence or the sesamoids, and the deformity is partly correctible. The range of movement of the MTP joint is well-preserved with no pain or crepitus. (*If present, suggestive of arthrosis.*)

Special test:

1. There is no tarso-metatarsal instability of the first ray (check by attempting to ballot the joint – If positive, remember to also assess for generalized laxity by Beighton's score. This has important implications on your management options – You may prefer doing a lapidus fusion in such case to correct the hallux valgus deformity)
2. Silverskiold's test: To rule out gastrocnemius tightness

EXAMINER: How are you going to manage this patient?

CANDIDATE: I would take a full detailed history to find out the patient complaints and functional limitations

EXAMINER: Oh! She is painful in the big toe!

CANDIDATE: I would like to have weight-bearing radiographs of the foot.

The radiographs show a moderate hallux valgus with:

1. Intermetatarsal angle of ___ (Normal value: <9°)
2. Hallux valgus angle of ___ (Normal value: <15°)
3. Distal metatarsal articular angle of ___ (Normal value: <15°)

Management options:

Conservative: Wide-toe box shoes, silicon spacer between the first and second toe, etc

Operative management: I would not operate for cosmetic reason

The further discussion could go onto distal/shaft/proximal metatarsal osteotomies, lateral soft-tissue release and medial capsular plication, tarso-metatarsal fusion, metatarso-phalangeal fusion. As first-ray surgery is an essential component of the Joint Committee on Surgical Training recommendations for CCT, a fair degree of knowledge would be expected from a candidate in a first-ray clinical case. It is advisable to be able to draw out the metatarsal osteotomy on a bone model or piece of paper– Especially if you intend to perform a Scarf osteotomy of the metatarsal.

Example answer: 'I would perform a Scarf osteotomy of the first ray and an akin osteotomy of the proximal phalanx with lateral soft-tissue release and medial capsular plication'.

Hallux rigidus

Look

- Stand – Inc. tiptoe, reduced First MTP extension
- Gait (walks on outer border of foot)
- Sit – Inc. shoes (wear of the outer border, insoles, etc.)

Feel

- Bony landmarks (osteophytes/tender first MTP joint)
- Tendons
- Sensation
- Pulses

Move

- Passive ROM (first TMT stability, stiff MTP, mobile IP)
- Proximal to distal

Hallux rigidus

Examination corner

Short case 1

EXAMINER: Please examine this patient's left foot.

A slim middle-aged lady standing with overall normal-looking feet

CANDIDATE: The attitude of the forefoot and hindfoot appears normal, with the arches of the foot well maintained. There appears a swelling on the dorsum of the great toe metatarsophalangeal joint.

Could you please **stand tiptoe** for me? Look for whether the big toe dorsiflexes at the MTP or IP joint during this manoevre. (In case of concomitant big toe MTP arthritis, the MTP joint may remain straight.)

Would you mind **walking** across the room, please?

The patient tends to walk along the outer border of the left foot and avoids touching the big toe to the ground

EXAMINER: What is your differential at this stage?

CANDIDATE: The patient shows a hallux limitus, with restricted MTP joint movement. I don't see any scars to suggest that this lady has had any surgery.

EXAMINER: Go on.

Patient sits and positions foot on conveniently situated footstool. The candidate kneels before patient but positions himself to look directly at the patient's face at the same time as examining the foot (anticipating a painful response to examination)

CANDIDATE: On palpation there are bony dorsal and medial osteophytes at the level of the great toe metatarsophalangeal joint. The movements of the great toe MTP joint are grossly restricted, especially dorsiflexion and are associated with pain and crepitus throughout the range.

EXAMINER: What would you like to do now?

CANDIDATE: I would request a weight-bearing foot x-ray series.

EXAMINER: Come and have a look at this x-ray.

X-ray reveals end-stage osteoarthritis (OA) with near-complete obliteration of the joint space and large dorsal osteophytes. The intermetatarsal angle is normal and there is a slight hallux valgus

EXAMINER: What are you going to offer this patient?

CANDIDATE: I would engage with the patient and find out what her problems and expectations are. In the first instance management may be conservative, offering analgesia and modified footwear, with a large toe box to accommodate the swelling, a sole stiffener to offload the hallux and a forefoot rocker to facilitate function.

EXAMINER: This lady has tried all that – She wants an operation. What operations do you have to help her?

CANDIDATE: Metatarsophalangeal fusion. (Gold standard)

Dorsal approach, protect the EHL, excision of the cartilage upto subchondral bone using nibblers and osteotomes, temporary stabilisation with K-wires and the compression using two cross 3.5 mm cortical screws in interfragmentary compression mode (or compression plate). The position of the arthrodesis would be 5–10° of valgus (no impingement of the second toe) and 25° dorsiflexion compared to the metatarsal shaft (or 10° dorsiflexion to the floor). Intraoperatively, the best way to assess this is to press the foot against a flat surface, such as the undersurface of a kidney dish. There should be roughly 5 mm clear space between the plantar surface of the pulp and the ground to prevent the toe jamming when the patient walks and to facilitate toe-off.

EXAMINER: Are you aware of any toe joint arthroplasties?

CANDIDATE: I have to say that an arthroplasty would not be my operation of choice, but I am aware of the Silastic arthroplasty and of Moje toe joint replacements. There are others on the market but I'm not familiar with them.

EXAMINER: What do you know about the Moje?

CANDIDATE: Intermediate-term survivorship data was poor, with significant proportions being revised to arthrodesis.

EXAMINER: Why not a cheilectomy?

CANDIDATE: Because the x-ray shows complete joint degeneration throughout and I think that it is likely that she would have poor pain relief from the operation.

Notes

- Remember that first MTP joint arthrodesis is a relative contraindication in the presence of DIP joint OA
- **Silastic® implant**: Not recommended because of the possibilities of transfer metatarsalgia, implant breakage, silicon synovitis, cock-up toe and stress fracture
- **Keller's excision arthroplasty**: Generally reserved for the very elderly low demand patient

Mild hallux rigidus: Surgical options

- **Cheilectomy**: Especially for dorsal osteophytes and dorsal impingement. Excision of the proliferative bone about the metatarsal head, removing approximately 30% of the metatarsal head and lateral osteophytes flush with the metatarsal shaft. The bone is resected to obtain 70–80° of dorsiflexion and to eliminate dorsal impingement. If severe arthrosis is present, a cheilectomy may lead to unsatisfactory results
- **Dorsal Closing wedge osteotomy of the proximal phalanx Moberg)**: Carried out if there is loss of dorsiflexion but no dorsal impingement
- **Manipulation under anaesthetic (MUA) and steroid injection**: Only for mild disease

Short case 2: Arthrodesed left hallux in a middle-aged woman with continuing difficulties

Short history: Instructed to ask three or four questions

- What was the original problem with the big toe that required you to have surgery?
- What is wrong with your big toe now?
- Is it painful?
- Do you have trouble walking?

Examination

- What has gone wrong?
- What is the optimum position for arthrodesis of the great toe MTP joint?
- What are the complications from surgery?

Hammer toe

A short case spot diagnosis. Generally accompanied by first-ray deformities

Memorandum

'A hammer toe refers to a toe with flexion at the PIP joint and extension at the MTP joint. The DIP joint is usually flexed although occasionally it is held in extension. The deformity can be flexible or fixed. Usually there are painful corns over the dorsum of the flexed PIP joint and callosities under the plantarly prominent metatarsal head.' (Table 13.1)

Table 13.1 Lesser toe deformities

	MTP joint	PIP joint	DIP joint
Hammer toe	Hyperextended	Flexed	Extended
Mallet toe	Extended	Extended	Flexed
Claw toe	Hyperextended	Flexed	Flexed
Curly toe	Extended	Flexed	Flexed

Management

Flexible

- Girdlestone procedure (fallen out of favour)

Fixed

- PIP joint arthrodesis
- Partial proximal phalangectomy. It relieves symptoms but has poor cosmesis
- Excision arthroplasty of the PIP joint ± extensor tenotomy plus MTP joint capsular release
- Du Vries metatarsal head arthroplasty

Complications of lesser toe surgery include swelling, reoccurrence of deformity, infection and neurovascular compromise.

Clawing of the lesser toes (Figure 13.3)

A claw toe refers to a toe with flexion at the DIP and PIP joints and extension at the MTP joint. Rarely occurs in isolation. Generally the condition is neurological in origin.

A flexible deformity indicates an imbalance between extrinsic and intrinsic muscle forces, whilst a fixed deformity may result from joint damage, capsular and/or tendon /ligamentous shortening.

Causes include neuromuscular disorders such as Charcot–Marie–Tooth disease, cerebral palsy and diabetic neuropathy. Other causes include compartment syndrome, poliomyelitis, cerebrovascular accidents and multiple sclerosis.

Memorandum

> 'On examination there is hyperextension at the MTP joints, plantar displacement of the metatarsal heads and distal migration of the fat pad. There are also plantar keratotic lesions under the metatarsal heads and callosities present over the dorsal surface of the PIP joints of the second, third and fourth toes of the right/left foot. There is plantarflexion of the PIP joints and the DIP joints. The deformity is fixed/flexible.'

Management

Try to identify an underlying cause if possible.

Conservative

Padding and protection of specific callosities, metatarsal pads, etc.

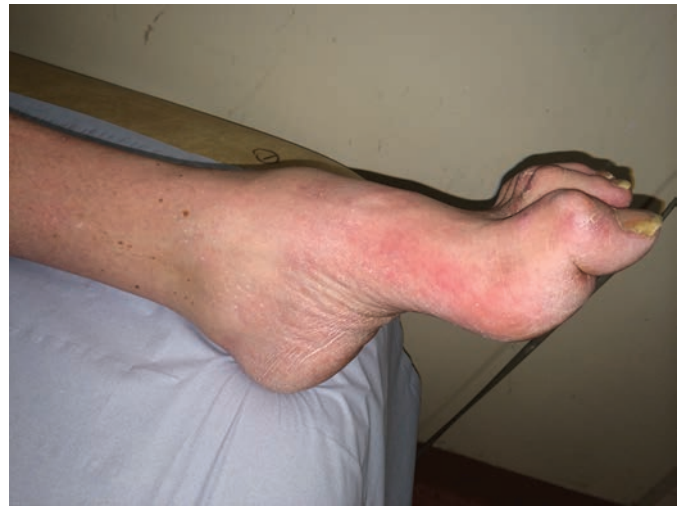

Figure 13.3 Claw toes

Surgery

The goal is to bring the MTP joints and PIP joints into a neutral position.

If there is a flexible deformity, consider a Weil's osteotomy or Girdlestone flexor–extensor transfer. With Weil's there is a functional shortening of the skeletal tissue relative to the shortened soft-tissue envelope. Problems include stiffness of the MTP joints and plantar displacement of the metatarsal heads, which can be corrected by an additional BRT osteotomy. The principle is to preserve the anatomical parabola of the relative lengths of the metatarsals. Thus, several Weil's osteotomies may be required to preserve this relationship across the forefoot. To correct the MTP joint deformity, extensor tendon Z-lengthening and dorsal capsulotomy may be required.

If there is a fixed deformity of the PIP joint this will not be corrected with a soft-tissue procedure. Again, the principle is to shorten the skeleton relative to the soft tissues, whilst correcting the fixed deformity by excision and then arthrodesis of the PIP joint. Thus, one solution is to undertake EDL Z-lengthening with EDB tenotomy, dorsal MTP joint capsulotomy with collateral release and relocation of the plantar plate, in combination with shortening excision of the PIP joint and K-wire arthrodesis.

Short case 1

- Claw toes and mild claw foot
- Mild pes cavus deformity described to examiners
- Big toe MTP hyperextension and PIP joint flexion
- Examined for metatarsalgia (tenderness when metatarsal heads palpated) with associated skin changes of keratosis
- I confirmed that the claw toe deformities of the lesser toes were fixed. I assessed flexibility of the toes in dorsiflexion and plantarflexion. The deformity remained static with

plantarflexion – If it had disappeared then the deformity is considered flexible
- Discuss the differential diagnosis of this condition. (Rheumatoid arthritis, diabetes, compartment syndrome, polio, cerebrovascular accident, Charcot–Marie–Tooth)

Curly toe

Background

This is a common condition affecting one or more toes. It is often bilateral, symmetrical and familial. It involves mal-rotation of the toe with a digit flexion deformity at the PIP and DIP joints. Deformity may resolve as the child grows. It is usually asymptomatic but may occasionally cause discomfort if rotation of the toe is such that the nail becomes weight-bearing. It can cause difficulty with shoe wear or may catch when putting socks on.

Memorandum

'On examination the third to fifth toes are curled medially and plantarflexed with lateral rotation at the DIP joint. The third toe is flexed and deviated medially to under ride the second toe, pushing it dorsally.'

Management

Manage conservatively if possible. Surgery is indicated if there are significant symptoms and deformity such as toes underriding adjacent medial toes. Delay surgery until after 4 years of age as at that stage the toes are bigger and the condition may have improved in a number of patients.

- Open flexor tenotomy of both FDL and FDB via incision beneath the proximal phalanx
- Additional flexor skin Z-plasty may be required
- Transfer of flexors to extensors (Girdlestone transfer)

No longer recommended; this has fallen out of favour as it is technically difficult and produces a toe stiff in extension

Ankle OA

Look
- Deformity/swelling/scars/erythema
- Stand – Inc. tiptoe, ROM
- Walk – Externally rotated (foot progression angle)
- Sit – Inc shoes, walking stick, etc

Feel
- Bony landmarks – tenderness
- Tendons
- Sensation
- Pulses

Move
- Passive ROM – Stiffness: Ankle/subtalar
- Proximal to distal

Ankle OA

History

- Pain is usually felt anteriorly and is mechanical: Made worse with activity and long periods of standing. Resting generally eases the pain. In severe arthritis, one can have night pain. Difficulty in walking on uneven terrain
- There is impingement due to osteophytes developing anteriorly on the joint line, first on the tibia, and then later kissing lesions develop on the talus, with impingement occurring in full dorsiflexion. It is worse in situations of increased dorsiflexion such as ascending slopes and stairs
- Deformity
- Restriction of movement (stiffness)
- Occasionally there is instability or weakness of the ankle

Examination

Both feet should be examined and range of movement compared. Any restriction of dorsiflexion is significant. Passive movement will often reveal crepitus. Active and passive movements are usually both restricted in ankle arthritis, and dorsiflexion is typically more affected than plantarflexion. An effusion may be present, which can be felt either anteromedially in the notch of Harty or anterolaterally. Osteophytes along with synovial thickening and swelling may be palpated. Pain is usually present on palpation of the anterior joint line. There is no significant valgus or varus deformity of the ankle. Test for ankle stability using the anterior drawer and tilt tests[b].

Having assessed the ankle joint, assess both the subtalar and midtarsal joints for signs of degenerative changes as this will influence management options. Finally, assess the neurovascular status of the foot.

Memorandum

A typical case would possibly be a middle-aged or elderly patient or a young patient post trauma.

The deformity would be either a valgus or varus deformity of the ankle (best seen from behind). There may be scars from previous surgery in a trauma case. There may be swelling of the medial or lateral malleoli, or of both. Assess the patient with them standing on tiptoe, and then ask them whether they can roll back onto their heels.

'On inspecting this patient's gait, he does not demonstrate any asymmetry or abnormal contact with the ground. The patient walks with an externally rotated gait to avoid tibiotalar movements. The patient demonstrates a restricted painful range of dorsiflexion/plantarflexion of the ankle. The subtalar movements are well preserved. Tibialis posterior function is normal.'

[b] Go to www.youtube.com (You Tube Broadcast Yourself™) and search for anterior drawer test to find some useful videos of this.

Management

Conservative

Conservative management consists initially of rest, physiotherapy, shoe modification (stiff sole and rocker bottom shoes), steroid injections, splints and orthosis, etc.

Surgery

- Open or arthroscopic ankle debridement with cheilectomy
- Joint distraction using Ilizarov fixator
- Ankle fusion
- Ankle arthroplasty

Position of ankle fusion

- Neutral dorsiflexion (10° plantar flexion in cerebral palsy)
- 5° valgus – Any more results in a stiff gait owing to poor push-off
- 5–7° external rotation

There are many methods of ankle fusion but the key point is that, if you are considering ankle fusion, ask the patient to wear an ankle–foot orthosis (AFO) for a few weeks. If it relieves pain then one is much happier to go ahead and perform an arthrodesis. Postoperatively, keep the patient in a plaster, non-weight-bearing for 6–8 weeks and then allow partial weight-bearing for a further 6–8 weeks in plaster until there are radiographic signs of bony union. Try to delay arthrodesis in post-traumatic osteoarthritis for at least 2 years.

Advantages of ankle fusion: Stable joint, proven long-term pain relief, tolerates heavy activity

Disadvantages of ankle fusion: Loss of motion and late degenerative effects on joints adjacent to and distal to the ankle

Approaches for ankle fusion:

1. Arthroscopic using the standard anterolateral and anteromedial portals with separate stab incisions for the compression screws
2. Anterior approach between the tibialis anterior and EHL – Protect the neurovascular bundle – Generally found deep to or just lateral to the EHL
3. Lateral approach with excision of the distal 8–10 cm of the fibula
4. Posterior approach – Used rarely. Reflecting the tendoachilles and using the interval between the FHL and peroneal tendons

Fixation methods used can be either two cannulated compression screws from the medial distal tibia into the talus, cross screws from distal tibia into the talus, with or without a neutralisation plate. Overall successful fusion rates published are around 90%.

Complications

- Wound breakdown
- Non-union
- Delayed union
- Infection
- Peripheral neurovascular complications

Short case 1: Post-traumatic OA of the ankle

- Examine this gentleman's feet

Short case 2: OA of the ankle

- Diagnosis
- Management

Short case 3: Ankle pain in an elderly woman

- Why are you making her stand on her toes?
- Assessment of tibialis posterior function
- Demonstration of active and passive ROM of the ankle
- Demonstration of subtalar range of movement

Arthrodesed ankle

This one can be a difficult one sometimes as the patient may have a near normal gait. As in a short case, with the time constraint and no opportunity to take a history, awareness of the possibility of this diagnosis is necessary to score in this relatively simple case

Short case 1

- Examination of ankle and subtalar movements
- Complications of arthrodesis
- Position of fusion
- Describe the rockers of the foot
- What approach would you use to arthrodese an ankle, how will you stabilise it?

Ankle arthroplasty

Various third-generation ankle arthroplasty implants are available in the UK market like the STAR®, Salto®, Zenith®, Hintegra® and Inbone®.

The implants can be either two components with the plastic insert bonded onto the tibial base plate, or else three components with a freely mobile plastic insert.

Extramedullary jigs are used to make a flat tibial cut first – Perpendicular to the mechanical axis of the tibia, and then the talus jigs are used. Both the tibial and talar components are uncemented.

Postoperatively, most surgeons prefer to keep the patient in a plaster, non-weight-bearing for 2 weeks and then allow partial weight-bearing in a boot for a further 4 weeks.

Contraindications:

1. Infection
2. Avascular necrosis of the talus
3. Younger patient (relative contraindication)
4. Severe mal-alignment of the tibio-talar joint (>20%)

Complications:

- Wound breakdown
- Residual pain
- Malleolar fracture
- Loosening of implants
- Infection
- Peripheral neurovascular complications

Evidence corner: Management options for end-stage ankle arthritis

1. SooHoo NF, Zingmond DS, Ko CY. Comparison of re-operation rates following ankle arthrodesis and total ankle arthroplasty. *J Bone Joint Surg Am*. 2007;**89**:2143–9.
   a. The rates of major revision surgery after ankle replacement were 23% at 5 years compared with 11% following ankle arthrodesis
   b. Patients treated with ankle arthrodesis had a higher rate of subtalar fusion at 5-years postoperatively (2.8%) than did those treated with ankle replacement (0.7%)
2. Gougoulias N, Khanna A, Maffulli N. How successful are current ankle replacements? A systematic review of the literature. *Clin Orthop Relat Res*. 2010;**468**:199–208.
   a. Residual pain was common (range: 27–60%)
   b. Ankle function improved after total ankle arthroplasty
   c. The overall failure rate was approximately 10% at 5 years

Charcot foot

Memorandum

Make a quick scan of the surroundings for possible clues to the diagnosis. There may be a foot orthosis present.

> 'The ankle joint is grossly deformed and swollen. There is loss of the normal medial longitudinal arch of the foot and a rocker bottom deformity of the foot. There is a chronic painless ulcer present on the plantar surface of the collapsed midfoot caused by excessive pressure in this area (mal-perforans). It does not appear infected. No ulcers or blisters/skin breakdown are seen over the first, third or fifth metatarsal head. Movement is abnormally increased and associated with loud audible crepitus, but it is painless. There is loss of light touch and vibration sense in the foot. There is wasting of the intrinsic muscles of the foot and clawing of the lesser toes.
>
> This is a Charcot joint.'

A Charcot joint (neuropathic arthropathy) is gross arthroses with new bone formation. It is caused by repeated minor trauma without the normal protective responses that accompany pain sensation. The joint is painlessly destroyed.

Charcot arthropathy

Charcot arthropathy is defined as a non-infectious, destructive process culminating in eventual dislocation and periarticular fracture in patients with peripheral neuropathy and the loss of protective sensation.

Clinical presentation

Presents with acute or subacute inflammation with the cardinal signs of inflammation.

Pain can be a feature despite the presence of neuropathy. Differential diagnosis includes sepsis, gout, chondrocalcinosis and cellulitis.

Although it is unlikely to get a case of acute Charcot arthropathy in the clinical exam, the discussion can always happen either during the clinical case or in the viva stations.

Examination corner

EXAMINER: How will you differentiate between an acute Charcot arthropathy and septic arthritis of the foot?

CANDIDATE:

1. Clinically: Both the conditions are likely to have local warmth, erythema and swelling. Charcot arthropathy is likely to be have minimal or no pain. Elevation of the leg is likely to slightly reduce the erythema in case of infection
2. Haematalogically: the inflammatory markers (WCC, ESR and CRP) are likely to be normal or slightly raised in Charcot compared to being sky high in sepsis
3. Radiologically
   a. MRI shall show edema in soft tissues as well as in the subchondral region in both cases. Collections of pus are likely to point towards infection
   b. Isotope bone scan shall show a hot spot in both these conditions. An addition of a hot WBC-labelled scan may be point towards sepsis
4. Bone biopsy: May be needed for a definitive diagnosis in cases of doubt, with the obtained tissue being subjected to both culture as well as histology

Three stages of Charcot arthropathy

- **Fragmentation stage**

  Plain radiographs demonstrate osteopenia, periarticular fragmentation and subluxation or frank joint dislocation. The foot is warm and oedematous and may demonstrate increased laxity
- **Hypertrophic or reparative stage**

  The oedema and warmth decrease. Radiographs show absorption of debris, fusion of bony fragments and early bony sclerosis
- **Consolidation or residual stage**

  Absence of inflammation and progression to a more stable, deformed foot or ankle. Radiographs show osteophytes, subchondral sclerosis and narrowing joint spaces

Causes

Any loss of sensation in a joint may render it susceptible to the development of a neuropathic arthropathy. It is seen most commonly in diabetic neuropathy, but is also associated with spinal cord injury, cerebral palsy, meningomyelocele,

syringomyelia, leprosy, syphilis, renal, failure congenital insensitivity to pain and chronic alcohol abuse.

Radiographs

Radiographs reveal bone fragmentation, joint subluxation, resorption and coalescence of the bony fragments. The bone surrounding the joint becomes sclerotic[1].

Management

- Bisphosphonates
- Immobilization in total contact cast
- Midfoot or ankle arthrodesis

Pathology

The natural history of Charcot neuroarthropathy is of bone demineralisation with loss of the bony architecture, fracture, fracture dislocation, progressive deformity, foot ulceration and infection.

Although the exact nature of Charcot arthropathy remains unknown, two main theories exist regarding the pathophysiology of the condition.

Neurotraumatic theory: Sensory–motor neuropathy results in abnormally high plantar foot pressures, atrophy of the intrinsic muscles, and both intrinsic/extrinsic imbalance and flexor/extensor imbalance.

Proprioceptive dysfunction combined with sensory–motor dysfunction leads to loss of protective function and repetitive microtrauma, resulting in ligament dysfunction and joint fracture dislocation.

Neurovascular theory suggests that autonomic neuropathy leads to a hyperaemic state, with vasodilatation and arteriovenous shunting creating a hyperdynamic circulation, which results in a mismatch in bone destruction and synthesis, leading to osteopenia and subsequent fractures.

It is the combination of these mechanisms, and the ability of the patient to walk with indifference upon a failing foot, which leads to the destruction of the foot architecture.

More recently, an **inflammatory theory** has also been proposed. A triggering factor, such as an injury, which often times goes unnoticed, sets up an inflammatory process with the elaboration of cytokines (such as interleukin-1 and tumour necrosis factor-alpha), which, in addition to causing inflammation, increases RANK-L expression, leading to osteoclast differentiation and bone resorption.

RANK-L overexpression has been noted in Charcot sufferers. RANK-L stimulates the expression of nuclear factor kappa B (NF κB), which causes pro-osteoclasts to differentiate.

However, simultaneously, NF κB increases expression of osteoprotegerin, which neutralizes the effect of RANK-L and avoids excessive osteolysis.

It is thought that genetic factors and osteoprotegerin polymorphism increases the risk of developing Charcot. In normal individuals immobilization of the limb in response to painful injury limits inflammation. The lack of pain in neuropathic patients drives the inflammation as the patient continues to use the limb[2].

Brodsky and Rouse classification

This system describes three anatomical Charcot joints:

Type 1: Charcot joints affect the midfoot area, including the metatarsocuneiform and naviculocuneiform joints

Type 2: Charcot joints affect the triple joint complex

Type 3A: Ankle joint

Type 3B: Fractures of the tubercle of the calcaneus

Aims of management in Charcot neuroarthropathy

- Stop the inflammation
- Protect and maintain the architecture of the foot (prevent deformity formation)
- Relieve pain
- Arrest and reverse bone demineralisation
- Judicious use of fracture fixation, osteotomy and arthrodesis to prevent and limit deformity

The cornerstone of management is protective supportive immobilization. This can be achieved either with total contact casting or with one of a number of proprietary total contact boots available on the market. However, the mean time of immobilization during the acute inflammatory phase is 18 months.

EXAMINER: How long will you continue the total contact cast for?

CANDIDATE: Till the skin temperature in the affected region reaches normal level.

Medical management

New understanding of the biological pathways involved has provided some medical tools to reduce the cytokine-mediated demineralisation.

Randomised controlled trials exist that have demonstrated measurable improvement in markers of bone demineralisation, e.g. the bisphosphonate pamidronate given over a 12-month period as a 90 mg infusion to 39 diabetic patients with evidence of acute-onset Charcot neuroarthropathy[3]. In addition to standard limb immobilization techniques, this led to an improvement in pain, to a significant reduction in limb temperature and to a measurable reduction in the patients' markers of bone turnover, urinary deoxypyridinoline and serum bone-specific alkaline phosphatase.

Similarly, the bisphosphonate alendronate given as a weekly 70-mg dose over 6 months led to significant reduction in pain scores and limb temperature, as well as a reduction in markers of bone resorption, serum collagen COOH-telopeptide of type 1 collagen and hydoxyproline. DEXA scanning showed a significant increase in bone mineral density.

Surgery for Charcot foot deformity

The surgical aims of treatment are to maintain and support bone and joint alignment, stabilise fractures, to prevent deformity and prevent soft-tissue breakdown and secondary ulcer infection.

Principles of surgery

- Fracture and fracture dislocation stabilisation
- Excision of bony prominences which threaten the soft-tissue envelope
- Arthrodesis and osteotomy to realign deformity and maintain foot architecture

Diabetec ulcer (Figure 13.4)

Despite being a common condition, this does not appear to be a particularly common short or long case.

Memorandum

'There is an ulcer on the sole of the right foot (most commonly at the site of maximal pressure under the head of the first metatarsal). The ulcer does not appear to be infected; there is no surrounding cellulitis or discharge from the ulcer base. There is thick callus formation over the pressure points of the feet. Two toes have previously been amputated and the remaining toes are clawed. There is loss of the normal medial longitudinal arch of the foot. Both the metatarsal and heel pads are atrophied. There appears to be reduced sweating of the foot. There is loss of sensation to light touch, vibration and pinprick in a stocking distribution. The feet are cold, the pulses are not palpable and there is loss of hair on the lower legs, which are shiny.'

'The toenails have no chronic changes present such as onychomycosis, ingrowing or incurvated changes onchymycosis, ingrowing or incurvated changes.

The patient has a peripheral neuropathy, a neuropathic ulcer on the sole of his foot and evidence of peripheral vascular disease. It is likely he has diabetes.'

The neuropathic ulcer

- Thick hyperkeratosis
- Pink punched-out base, which readily bleeds
- Painless

The ischaemic ulcer

- Not surrounded by hyperkeratosis
- Dull fibrotic base, does not bleed easily
- Painful to touch
- Ulcers present over the curve on the first and fifth metatarsal heads

Factors that may contribute to the development of the diabetic foot lesion include:

- Injury
- Neuropathy

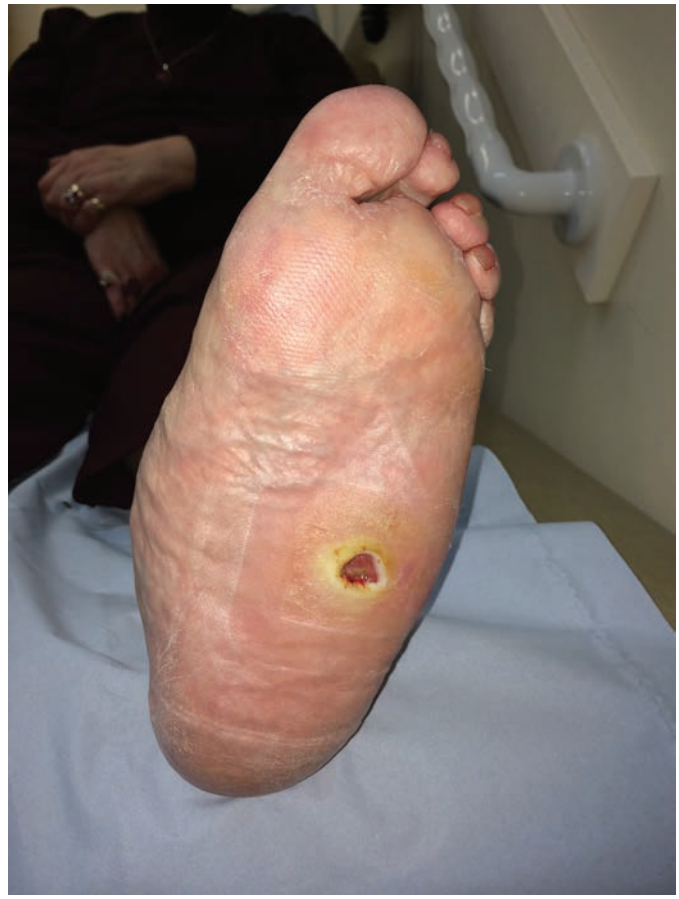

Figure 13.4 Neuropathic ulcer

- Small vessel disease
- Large vessel disease
- Increased susceptibility to infection
- Foot deformity leading to increased possibility of mechanical stress and trauma

Causes of a peripheral neuropathy[c]

- Diabetic foot
- Tabes dorsalis (lower extremity)
- Syringomyelia (upper extremity)
- Hansen's disease (leprosy)
- Myelomeningocele (ankle and foot)
- Congenital insensitivity to pain
- Other neurological problems
- Peripheral neuropathies (alcohol, amyloidosis, pernicious anaemia)
- Infection (yaws, tuberculosis)

[c] In the neuropathic foot the pulses may be palpable or even bounding. The foot is warm, dry and insensate with pulses and distended veins.

Pes cavus

Look

- Stand – Heel varus, cavus
- Walk – Recruits toe extensors
- Sit – Inc. shoes, etc

Feel

- Bony landmarks
- Tendons
- Sensation
- Pulses

Move

- Special –
  Coleman's block
  Hands
  Spine

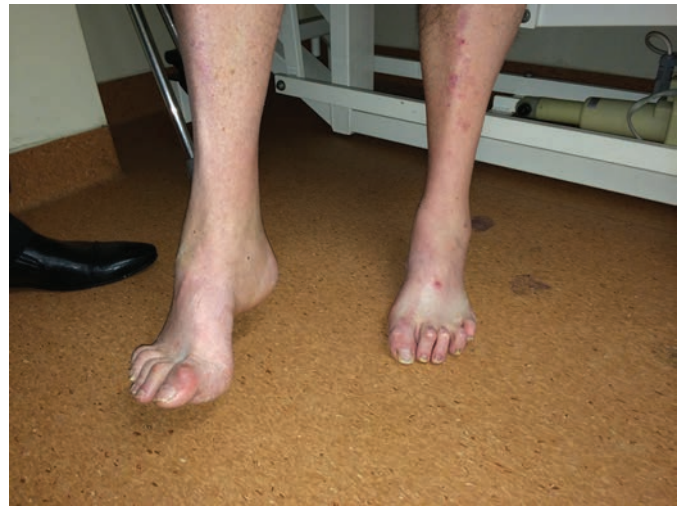

Figure 13.5 Pes cavus

Charcot–Marie–Tooth disease (Figure 13.5)

- Commonest cause of bilateral cavus foot deformity
- Exclude spinal cord pathology with MRI

Differential diagnosis

Differential diagnosis includes poliomyelitis, cerebral palsy, Friedreich's ataxia, spinal muscular atrophy, spinal cord tumour, syringomyelia, spinal dysraphism and diastematomyelia.

Heritance/aetiology

- Charcot–Marie–Tooth (CMT) disease comprises a family of heritable neurological diseases, owing their pathology to defective peripheral nerve myelin sheath proteins
- It is variously named CMT, peroneal muscular atrophy and hereditary motor sensory neuropathy types 1–7
- The genetic nomenclature describes four major subgroups: CMT 1(a, b, c) (50%); CMT 2 (20%); CMTX (20%); and CMT4 (10%), with CMT1a being the commonest (40%)
  - CMT1a (AD inheritance) is associated with defective 'peripheral myelin protein 22' (PMP-22), which is associated with a recombination error, causing a segmental trisomy on chromosome 17. The defect produces a shortage of normal protein in the myelin sheath, rather than a completely defective protein

Clinical features

- CMT1 is associated with a progressive deterioration in peripheral neurological function. Patients are apparently normal in infancy but develop weakness, ankle instability and cavovarus deformity in their teens or early adulthood
- An early sign, symmetrical reduction in lower limb reflexes precedes symptoms
- There is classically selective anterior compartment weakness, which is associated with peroneal compartment weakness (peroneus brevis more than peroneus longus). EHL may be spared
- The posterior compartment is spared until late in the disease

Foot deformity

- Foot intrinsic wasting/extrinsic sparing produces a clawed toe posture. The intrinsics normally flex (plantarflex) the MTP joints and extend the IP joints. Unopposed extrinsics extend (dorsiflex) the MTP joints and flex the IP joints
- Cavus foot deformity is attributed to a continued imbalanced pull of extrinsics, in particular tibialis posterior and peroneus longus, in combination with deficient intrinsics
- Peroneus longus sparing causes plantarflexion of the first ray, effectively pronating the forefoot in the absence of the supinating effect of the tibialis anterior, producing an increase in arch height. The forefoot deformity drives the hindfoot varus
- If the first ray is plantarflexed relative to the adjacent rays, when it strikes the ground first this will cause the foot to roll from a pronated position to a supinated position. This rotation will be transmitted along the foot and will twist the hindfoot from neutral to a varus position. This mechanism is the basis for the Coleman block test

Coleman block test

This test is applied to a patient with a cavovarus foot deformity. A block is applied to the lateral border of the forefoot and the hindfoot, such that the first ray (which is pronated) is off the block and can plantarflex fully. This eliminates the twisting moment through the foot and allows the hindfoot to assume a normal position

- If, with the block in place, the hindfoot corrects to neutral, this shows that the subtalar joint is mobile and the forefoot deformity is driving the hindfoot deformity. The hindfoot varus is potentially correctable with a dorsiflexion osteotomy of the first metatarsal. If, with the block in place, the hindfoot remains in varus, this shows that the subtalar joint is fixed and not correctable with a dorsiflexion osteotomy of the first metatarsal
- In addition, the hindfoot is inverted by the tibialis posterior, the action of which is unopposed by its normal antagonists, wasted by disease, namely tibialis anterior and peroneus brevis
- Foot drop occurs as a consequence of loss of tibialis anterior
- Clawed great toe: In the absence of tibialis anterior, EHL functions as an accessory dorsiflexor of the foot and, as such, its overaction winds the MTP joint up into an extended posture

Foot deformity summary

- Clawed lesser toes (intrinsic/extrinsic imbalance)
- Clawed great toe (EHL acts as an accessory dorsiflexor in absence of tibialis anterior)
- Forefoot cavus (unopposed peroneus longus)
- Hindfoot cavus (unopposed tibialis posterior)
- Foot drop (loss of tibialis anterior)
- Ankle instability or a high stepping case; new-onset cases may present with a history of ankle instability or foot drop gait

Upper limb involvement (Figure 13.6)

Upper limb involvement can lead to intrinsic minus deformity with small muscle atrophy, thenar and hypothenar muscle atrophy, interossei wasting.

Operative planning

Lower limb MRI can be used to assess muscle group atrophy in preparation for tendon transfer planning.

Conservative management

Bracing in combination with physiotherapy can be used to treat symptomatic foot drop and ankle instability, treating symptoms and maintaining range of motion.

Surgery[4,5]

Soft-tissue reconstruction with tendon transfer

- Peroneus longus to peroneus brevis transfer
- Gastrosoleus lengthening
- Jones transfer (transfer of EHL from the great toe to the neck of the first metatarsal, with DIP joint arthrodesis)
- Plantar fascia release

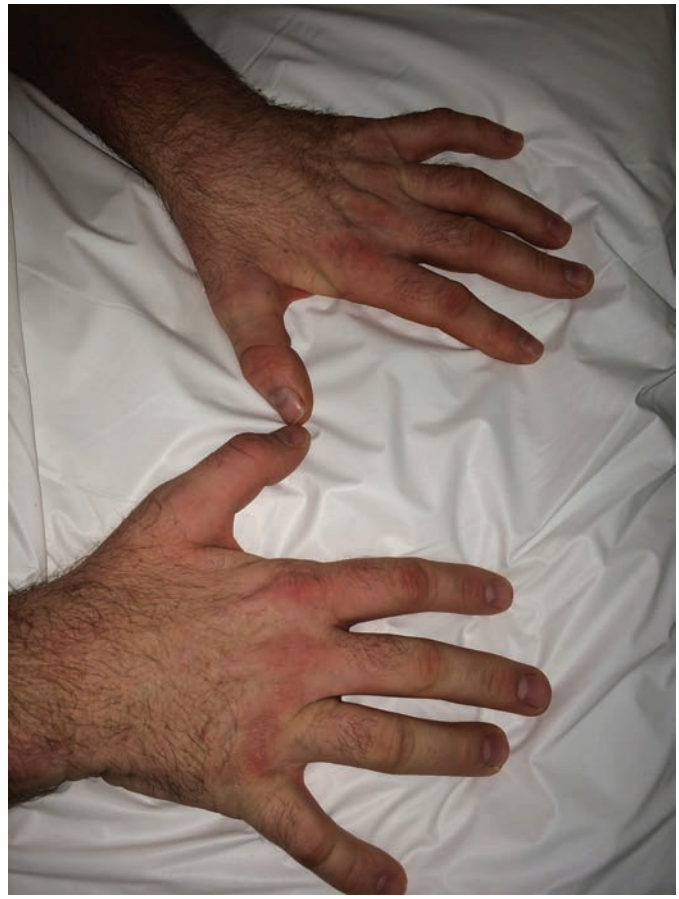

Figure 13.6 Wasting of interossei

- Claw toe correction through PIP joint shortening arthrodesis, with EDL lengthening and EDB tenotomy, with relocation of the plantar plate

Bony procedure, arthrodesis and ostetotomy

- Dorsiflexion osteotomy of the first metatarsal
- Lateral displacement calcaneal osteotomy. This corrects hindfoot varus and to some extent helps to stabilise the hindfoot (and ankle)
- Where hindfoot varus is associated with increased calcaneal pitch, a crescentic osteotomy can correct both deformities
- Triple arthrodesis can be used for fixed cavovarus deformity, treating both fixed deformity and attendant joint degeneration in a single procedure. Rebalancing of the foot with tendon transfer should be considered in combination with triple arthrodesis, to prevent late ankle instability or forefoot-driven deformity

Short case 1

History

Patients complain of having tired feet and having difficulty buying shoes that accommodate their high arch and the

clawing of their toes; they have metatarsalgia and callosities secondary to lateral weight-bearing.

Examination

'On inspection, there is distal wasting of the lower limb muscles that stop abruptly (state where). The legs are spindle-shaped, and the calves have an inverted champagne bottle appearance. The feet show a pes cavus-type of deformity with associated clawing of the lesser toes. The patient has a bilateral drop foot gait (steppage gait). There is recruitment of the extensors of the long extensors of the toes during forward propulsion. Ankle jerks are absent. The ankle is in equinus; there is midfoot and hindfoot varus and cavus. The forefoot is in valgus and there is a plantarflexed first ray.

Weakness of the foot intrinsics and contracture of the plantar fascia add to the fixation of the cavus deformity and secondary clawing of the toes. There are no neuropathic ulcers present.

The patient also demonstrates wasting of the small muscles of the hands. There is a tendency for the fingers to curl and the patient has difficulty in straightening and abducting them.

The spine does not show any cutaneous manifestation of spinal dysraphism.'

Short case 2

History

- The presenting complaint is of walking on the outer borders of the feet, difficulty in wearing shoes and painful callosities
- Walking distance is reduced to 100 yards
- Previous surgery of Jones procedures and PIP joint fusions
- No regular medication
- Otherwise fit and healthy

Examination

'On inspection, this gentleman stands with some difficulty. He has obvious wasting of both calves. On the left foot, he has a varus heel. Both feet are in equinus. Looking from behind, one can again see the obvious varus left heel.'

'*Just tell me if you have any discomfort on palpation.*'

'There are thick callosities over the lateral border of both feet. The hindfoot will not come to neutral. The subtalar joints will not correct to a neutral position; they are fixed in 10° of inversion. There is a jog of movement of the hindfoot. He has grade 4 power of tibialis posterior. There is almost no power of eversion.'

'*Push your big toe down.*'

'He does appear to have good peroneus longus power. The Coleman block test does not improve the hindfoot varus, suggesting that it is fixed.'

EXAMINER: Can you explain the principles of the Coleman block test?

CANDIDATE: Initially the hindfoot is in varus. The Coleman block test is performed by placing a block under the lateral column of the foot and allowing the first metatarsal to drop to the floor. Heel varus correction indicates that the hindfoot deformity is flexible and that the varus position is secondary to the plantarflexed first ray, or valgus position of the forefoot. A fixed hindfoot deformity will not correct.

EXAMINER: What is Charcot–Marie–Tooth disease?

CANDIDATE: This is a hereditary neurological disorder characterized by weakness and wasting of intrinsic muscles of the foot, the peroneal muscles, the dorsiflexors and plantarflexors of the foot and toes.

The end result is a progressive equinocavovarus foot with:

- Clawing toes
- Forefoot valgus
- Plantarflexed first ray
- Midfoot and hindfoot cavus
- Ankle equinus

Management

Conservative

Conservative management includes the use of insoles, orthotics, physiotherapy, etc.

Soft-tissue procedures

- Achilles tendon lengthening
- Split anterior tibial tendon transfer
- Plantar fascia release
- Claw toe procedures

Bony procedures

- Dwyer's calcaneal osteotomy
- Jones procedure: Interphalangeal arthrodesis of the hallux and transfer of the EHL tendon into the distal first metatarsal (to decrease clawing of the big toe)

Examination corner

Short case 3

A candidate was asked to examine a patient's gait and his lower legs. As soon as the patient walked, the bilateral foot drop and wasted anterior muscle compartments were obvious, but the candidate failed to pick up the sign. The examiners were critical of the candidate's neurological examination of the lower legs as the candidate insisted on performing a myotomal type of power assessment – He required prompting and appeared hesitant. The candidate failed to appreciate that the small muscles in the hand can be affected in this condition.

Short case 4: Cavus feet, young man with hereditary sensory–motor neuropathy

Differential diagnosis and management
Types of hereditary sensory–motor neuropathy
Surgical management: Jones procedure

Other causes of pes cavus

Aetiology

- Idiopathic
- Congenital: Arthrogryposis, residue congenital talipes equinovarus (CTEV)
- Traumatic: Compartment syndrome, crush, burns

- Neuromuscular: Disorder of muscles, peripheral nerves, spinal cord, central nervous system

History

- Congenital or acquired? When did you notice this deformity? Were you born with it? When did it develop?
- Progression: Is it getting worse?
- Full family history: This often runs in families, so check the family history for neurological disease/neuromuscular disease
- Any problems with your back?
- Any problems with the bladder or bowel? (Bladder or bowel dysfunction)
- Have you any pins or needles or loss of power in your legs? (Sensation and motor power)
- Any difficulty with walking, weakness or tremor?
- Patient may complain of difficulty with footwear, of tired aching feet, of metatarsalgia and lateral foot pain because of the area of contact with the floor, pressure over the dorsum of the PIP joints, or recurrent giving way of the ankle

Memorandum

Look for orthosis, splints and special shoes.

'On inspection of the feet there is a unilateral/bilateral accentuation (exaggeration) of the medial longitudinal arch of the foot. There is clawing of the lesser toes with callosities over the dorsal PIP joints and heads of the metatarsals (examine the soles of the feet). There is clawing of both hallux and prominence of the EHL, which is overactive as a dorsiflexor to compensate for a weak tibialis anterior. There is a varus of the hindfoot, as well as a high arch, clawing of the toes and callosities. There are no visible ulcers or surgical scars present. There is a generalized wasting of the calf muscles. On the double heel raise test, the hindfoot remains fixed in varus.'

'I would like to examine this gentleman's spine, please. On inspecting this man's spine, there are no obvious hairy patches, skin discoloration or swelling suggestive of either occult spina bifida or diastematomyelia.'

Carry out a full neurological examination of the lower legs, testing for sensation, muscle power and reflexes.

'On examination of gait, the patient demonstrates a drop foot gait. Examining his hands, he demonstrates intrinsic muscle wastage.'

The Coleman block test

The Coleman block test is used to check whether the subtalar joint is mobile or rigid. It is performed by placing the patient's foot on a wooden block with the heel and the lateral border of the foot on the block full weight-bearing whilst the first, second and third metatarsals are allowed to hang freely into plantar flexion and pronation. If the heel varus corrects while the patient is standing on the block, the hindfoot is considered flexible.

Two main patterns of deformity tend to occur: Calcaneocavus and cavovarus.

Calcaneocavus

This mainly involves hindfoot abnormalities. Dorsiflexion of the calcaneum occurs because of a weak Achilles tendon with normal tibialis anterior. There is no pronation of the forefoot and, therefore, no varus deformity of the hindfoot. It is more common in polio.

Cavovarus

This mainly involves forefoot abnormalities. The forefoot is pronated and the heel is in varus. This is more common in hereditary motor sensory neuropathies (HMSN).

Investigations

- Weight-bearing lateral radiograph
- X-ray spine for spina bifida
- MRI scan of the spine
- Neurological referral

Management

Conservative

- Moulded insoles, heel pads, etc

Surgery

- Jones procedure: Fusion of the IP joint and proximal transfer of EHL to the neck of the first metatarsal
- IP joint fusions as part of claw toe correction
- Calcaneum osteotomy – Closing wedge lateral osteotomy
- Plantar fascia release (Steindler release) if mobile
- Triple arthrodesis as a salvage procedure for a severe deformity

Examination corner

Short case 1: Multiply operated bilateral cavovarus feet

- Examination features
- Differential diagnosis
- Suggest further surgery options

Short case 2: Bilateral pes cavovarus due to diastematomyelia

- Candidate asked to take a short history
- Assessment of gait
- Examination of motor and sensory function of the lower legs to locate the level of the lesion
- Demonstration of knee reflexes and ankle clonus
- Surgical scar from a previous Jones procedure: 'What is this scar suggestive of, why is the operation performed?'

Short case 3: Young woman in her early 20s, sitting down on a chair; unilateral pes cavus deformity

Candidate asked to examine left foot.

CANDIDATE: On inspection of the left foot there is an obvious severe pes cavus deformity present with clawing of the lesser toes. There are callosities present over the dorsal surfaces of her PIP joints. There is a well-healed longitudinal surgical scar present over the dorsal surface of her big toe.

CANDIDATE: I would like to examine her spine now.

EXAMINER: The spine is normal – Don't bother. What operation has she had on her big toe and why?

CANDIDATE: She has had a Jones procedure; this is where the IP joint is fused and the EHL tendon is transferred to the metatarsal neck to prevent hyperextension of the hallux.

The candidate was not asked to demonstrate anything else clinically, particularly the Coleman block test, gait or neurological examination.

Short case 4

- Postoperative resection of a spinal tumour
- Bilateral wasted calves with resolving neurology
- Stiff cavovarus foot – Differential diagnosis and management
- Management

Rheumatoid foot

A classic short case. It is unlikely that you would go much further than a general inspection of the feet. You may then be asked about the management principles of the rheumatoid foot. **Remember to mention the need to assess the hip and knee first** before considering foot surgery.

Memorandum

(*Examination tip is to start from the ankle and work distally.*)

'On general inspection there is a bilateral symmetrical deforming arthropathy. There is a hindfoot valgus and localized swelling over both the medial and lateral malleoli. This could be due to tenosynovitis of the tibialis posterior and peroneal tendons. There is also collapse of the medial longitudinal arch of the foot, suggestive of possible tibialis posterior tendon rupture.'

'There is pronation of the forefoot (forefoot abduction). There are severe bilateral hallux valgus deformities and clawing of several lesser toes and a hammer toe deformity of the third left toe. There are callosities over the dorsal surfaces of the PIP joints of the lesser toes. The lesser toes are dislocated/inflamed/ulcerated. There is swelling over the MTP joints with the appearance of possible subluxation of these joints.'

'The skin appears papery, thin and fragile with a possible vasculitis. There are no obvious rheumatoid nodules or ulcers present. There is normal sensation of the foot and ankle. Similarly, the dorsalis pedis and posterior tibia pulses are present and capillary refill is <2 seconds.'

'The patient was not able to perform a double heel raise test because of pain. On double heel raise test, the heel failed to invert and the medial longitudinal arch remained collapsed. This suggests tibialis posterior insufficiency. The patient demonstrates an antalgic gait with an externally rotated attitude of both feet. A roll-over gait is present.'

'On inspection of the plantar surfaces of both feet, there are callosities present over the metatarsal heads. There are no ulcers, scars or sinuses. There is crepitus, tenderness and swelling present over both malleoli. Movement, both actively and passively, of the ankle and subtalar joint is reduced. Specifically, dorsiflexion is painful and limited both actively and passively to neutral. Similarly, plantarflexion is limited to 20° bilaterally both actively and passively.'

'The left/right subtalar joint is irritable with only a jog of movement present. There is reduced power of dorsiflexion, plantarflexion, inversion and eversion, probably MRC grade 4.'

Check the vascular status of the foot. Perform a careful neurological assessment as there may be a neuropathic component.

Management

The general surgical principles applicable to the rheumatoid foot depending upon the severity of disease include:

- Forefoot reconstruction with hallux valgus and claw toe reconstruction
- Forefoot arthroplasty, e.g. Hoffman excision arthroplasty lesser MT heads and first MTP joint fusion (A stable first ray)
- Subtalar or triple arthrodesis
- Ankle arthrodesis
- Pan talar arthrodeses
- Ankle arthroplasty

Intermediate case 1: Rheumatoid foot oral

EXAMINER: Examine this patient's foot.

The patient, an elderly lady, sat in a chair with both feet exposed below the knee. There are scars from bilateral total knee replacement. The left foot has severe clawing of all the lesser toes and severe hallux valgus. The right forefoot bears surgical scars on the dorsum of the forefoot, with neutrally aligned lesser toes which appear somewhat foreshortened and the hallux is neutrally aligned, though slightly extended, the pulp not quite touching the ground.

Her hands, resting on her lap, have typical changes of advanced rheumatoid arthritis: There are walking aids and orthotic shoes beneath her chair. The skin on her lower limbs is atrophic, her lower limbs are thin and the calf muscle mass is similarly atrophic.

CANDIDATE: On examination I note that this patient has evidence of severe arthropathy affecting her left foot, and that her right foot appears to have undergone some surgical correction.

I also notice evidence of arthropathy affecting her upper limbs and evidence of possible knee arthroplasty, suggesting a systemic arthritides such as rheumatoid arthritis.

EXAMINER: Just focus on the feet for now.

CANDIDATE: Examining the feet sequentially, the left forefoot shows severe clawing and dorsal subluxation of all the lesser toes: The toes

effectively sit upon the dorsum of the distal forefoot and are severely clawed, there is a callosity on the dorsum of all the PIP joints and there is healing ulceration on the dorsum of the first toe PIP joint. It does not clinically appear to be infected.

The great toe is severely mal-aligned, in a hallux valgus deformity, there is a large bunion and the MTP joint appears swollen and with possible prominent osteophytes distorting the skin.

The great toe is also markedly pronated and there are dystrophic changes affecting the great toe toenail.

The skin of this lady's lower leg and foot is atrophic and pigmented and I notice some swelling around her ankle and evidence of muscle atrophy in her lower legs.

'Would you mind if I look at the sole of your foot, madam?' (Extending the patient's leg very gently, being aware of the knee arthroplasty)

The fat pad has subluxed forward as the lesser toes have subluxed and the metatarsal heads of all rays, but in particular the second and third rays are very prominent. There is callus on the skin under all the lesser toe metatarsal heads, though relative sparing of the great toe.

The metatarsal heads are subcutaneous and are immediately palpable. (*Watching the patient for signs of discomfort.*)

(*Replacing the patient's foot on the ground.*) The lesser toes are all subluxed or dislocated; 2 and 3 are not passively correctable although 4 and 5 are slightly more so. I note that the IP joints of the second and third toes are stiff and possibly ankylosed, and the fourth and fifth toes are similarly stiff at the level of the IP joints.

The great toe hallux valgus deformity is not passively correctable, and I notice that movements within the great toe, and in particular extension, are markedly reduced. There is palpable osteophytosis associated with the great toe MTP joint.

'Do you feel me touch you here and here, madam?' The patient's sensation is grossly intact, and I can palpate the dorsalis pedis pulse . . . but not the posterior tibial pulse.

'I would now like to continue to examine this patient's midfoot, hindfoot and ankle' . . .

EXAMINER: Go on.

CANDIDATE: If you don't mind, madam, I would like to test the movement of your foot and ankle. I will try not to hurt you, but please let me know if I cause you any discomfort.

PATIENT: Fine.

(With due diligence in avoiding hurting the patient:)

CANDIDATE: I notice that this lady has preservation of her medial longitudinal arch, albeit at the moment without weight-bearing. I note that there appears to be neutral alignment of the hind-mid- and forefoot, though I will assess this better when I ask the patient to stand.

Gently controlling subtalar joint and hindfoot movement with one hand, passively abduct and adduct and invert and evert the mid/forefoot. The movements are generally reduced.

Gently controlling the talar neck with one hand, and grasping the heel with the other, attempt to rock the heel from side to side. The movements are markedly reduced.

Palpating the talar neck, and manipulating the ankle by holding the heel, assess ankle movement. These are also reduced, though at rest the ankle sits in neutral alignment and there is both dorsiflexion and plantarflexion through the ankle.

'I would now like to test the patient's power in the major muscle groups.'

EXAMINER: Go on.

It is important to place the patient's foot into the position in which you want to test mucle power; asking them to 'invert', 'evert' or push up or down will confuse the patient and make you look like an amateur. Rehearse the following for a generic test of foot motor function, but exercise caution in the rheumatoid foot as you may hurt the patient.

'Extensors: Tibialis anterior, extensor digitorum longus, extensor hallucis longus'

Ankle dorsiflexion – Place the ankle and foot into maximum dorsiflexion, place your hand on the dorsum of their foot and say to the patient, '*Hold your foot in this position*', '*Resist me when I push against you*'.

Notice the tibialis anterior fire up, and also notice the active contraction of the toe extensors.

Toe extension – Maintain the dorsiflexed posture, but this time place hand on dorsum of toes, and say to the patient, '*Hold your foot in this position*', '*Resist me when I push against you*'.

Plantiflexors – Gastrosoleus

Place the foot and ankle into extreme plantarflexion, place your hand under the sole of the foot and say to the patient, '*Hold your foot in this position*', '*Resist me when I push against you*'.

Toe flexors: Flexor digitorum and hallucis

Maintain the plantarflexed position, but this time place your fingers beneath the lesser toes and then the hallux, and say to the patient, '*Hold your foot in this position*', '*Resist me when I push against you*'.

Evertors: Peroneus longus and brevis

Place the foot into extreme eversion, place your hand against the lateral border of the foot, and say to the patient, '*Hold your foot in this position*', '*Resist me when I push against you*'

Inversion: Tibialis posterior

Place the foot into maximal inversion (ideally in slight plantar-flexion to neutralize tibialis anterior), and place your hand against the medial border of the foot. Say to the patient, '*Hold your foot in this position*', *'Resist me when I push against you*'.

CANDIDATE: Accepting that this patient has globally reduced movement throughout the hindfoot and midfoot, and some painful inhibition as a consequence, there does not seem to be any overt weakness of any muscle group.

EXAMINER: What would you like to do now?

CANDIDATE: I would like to examine the patient weight-bearing and then walk the patient.

On examination the patient's lesser toes maintain their subluxed position, the plantar fat pad remains subluxed forward and the lesser toes remain subluxed on the dorsum of the forefoot, not contacting the ground, and the hallux remains in valgus and pronation and does not seem to be loaded effectively.

The height of the medial longitudinal arch is preserved in the weight-bearing stance. Beware, as the rheumatoid foot may be accompanied by midfoot collapse due to arthrosis or to tibialis posterior insufficiency.

The hindfoot is normally aligned in slight valgus, relative to the forefoot.

CANDIDATE: Would you mind walking, please?

(*The patient advises that she can only take a few steps, because of pain walking barefoot.*) She walks with a normal foot progression angle and a shortened step length, there is reduced ankle movement with reduction of all three rockers, there is no heel strike as such, initial contact is flat-footed and there is no toe-off in terminal stance.

EXAMINER: OK to the patient, thank you. Now have a look at the patient's x-rays.

CANDIDATE: This is an AP and a lateral of the patient's left foot. The AP shows erosive arthropathic changes of the lesser toe metatarsal heads, with frank dorsal dislocation of the MTP joints. There is also erosive change of the articular surfaces of the proximal phalanges. The clawing is apparent on the AP.

EXAMINER: OK, we won't discuss the contralateral foot, which you have correctly observed has been operated upon, but talk me through the general principles for the management of this lady's forefoot.

CANDIDATE: The principles are to offer initially conservative and, if unsuccessful, operative management.

Conservative management is centred around providing accommodative and supportive footwear, with custom-made shoes with a wide-toe box to accommodate the toes, and insoles which are made of an appropriate material which protects the metatarsal heads. Scoops can be applied to the insole to accommodate individual prominent heads.

If the patient's symptoms are intolerable and are significantly affecting their quality of life, then the operative options include the management of rheumatoid disease in general during surgery and then specific to the foot.

The patient must be appropriately prepared for surgery, with specific attention in rheumatoid patients to the safety of intubation with respect to the stability of the C-spine and management of disease modifying anti-rheumatoid drugs (DMARDs). Steroids and non-biological agents can be continued in the perioperative period, and biological agents should be stopped one half-life prior to surgery and suspended until 2 weeks following surgery.

The principles of management for the severely arthritic rheumatoid foot are to realign, stabilise and refunction the first ray by performing either a first MTP joint arthrodesis or a Keller's arthroplasty, though the evidence in the literature suggests that first MTP joint arthrodesis provides greater stability and a more predictable outcome, accepting the risks of non-union in arthrodesis.

With respect to the lesser toes in end-stage rheumatoid disease, the options are to perform a Hoffman-type excision arthroplasty, with excision of all the metatarsal heads, through either a plantar or a dorsal approach, sectioning the metatarsal necks to create an even parabola of gradually decreasing length from the second to the fifth metatarsals. The alternative is to perform an excision arthroplasty of either a Stainsby type, removing the bases of the proximal phalanges, or of a Fowler type, excising both the metatarsal head and the base of the proximal phalanx. However, the principle in all cases is to try to relocate and refunction the plantar plate by passing an elevator under the metatarsal head to relocate and reposition the plate.

EXAMINER: Are you aware of any publications supporting the principles that you have described?

Bell. (Pass.)

Short case 1

Examine the feet.

- Hallux valgus with pronated great toe
- Clawed toes
- Callosities under metatarsal heads
- Varicose eczema

Pes planus

Look (Figure 13.7)

- Stand – Incl. single leg tiptoe, ROM (heel valgus > varus)
- Walk – Failure of restoration of arch
- Sit – Inc. shoes, etc

Feel

- Bony landmarks
- Tendons – Tibialis posterior
- Sensation
- Pulses

Move

- Passive ROM (reduced subtalar and midtarsal movements)
- Proximal to distal
- Special tests –
  Beighton's criteria (flexible flatfoot) (young/adolescent)
  Marfan's/Ehler–Danlos

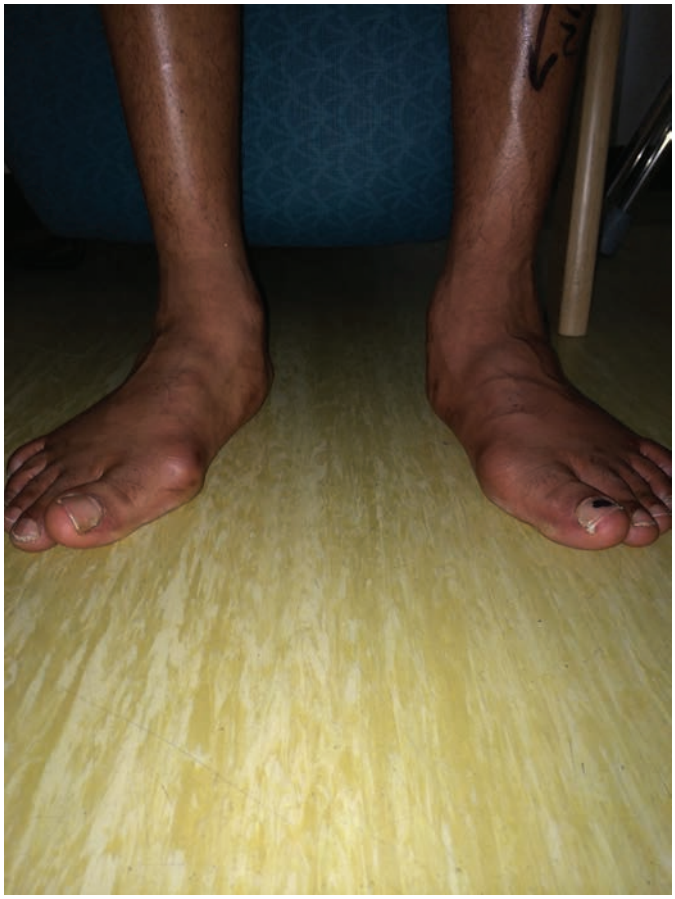

Figure 13.7 Pes planus

Tarsal coalition

This is not an uncommon lower limb short case, with usually at least one patient in the examination hall with the condition.

Short case 1: Young boy (approximately 11 years old) sitting on a chair

Examiner looks around and spots the young boy.

EXAMINER: Why don't you start by examining this young man's left foot and tell me what you find. He has been complaining of some vague pain in this foot for several months.

CANDIDATE: (*After a brief introduction and handshake with patient.*) May I examine your foot? Would you stand up for us so that we can have at look at your feet?

Examination started with inspection from behind (the rules were immediately broken!).

CANDIDATE: On inspection there is a marked pes planus deformity of the left foot with a valgus heel compared to a normal looking right foot.

(A spot clinical diagnosis – Even at this early stage I picked up that it was a probable tarsal coalition. However, one cannot assume anything and has to carry on examining, but the rest of the examination should be directed towards confirming this clinical diagnosis.)

CANDIDATE: The medial longitudinal arch of the left foot is not visible.

EXAMINER: What do you think the diagnosis is?

CANDIDATE: I think the diagnosis may be tarsal coalition.

EXAMINER: You have one test only to confirm this – Which one will you use?

CANDIDATE: Can you stand on your tiptoes for me? On double heel raise stance the left heel fails to invert and go into varus. The medial longitudinal arch of the foot remains flat. Jack's test fails to correct the pes planus deformity.

(Jack's test: The patient is asked to stand weight-bearing with the foot flat on the ground, the candidate then dorsiflexes the big toe, and watches for the re-creation of the medial arch: This is suggestive of a flexible flat foot.)

EXAMINER: Good.

I carried on examining the patient.

CANDIDATE: The patient walks with an antalgic gait. Palpation revealed tenderness at the anterolateral aspect of the foot – The sinus tarsi (location of the coalition). There is no peroneal spasm/protective spasm of the peroneal muscles. There are normal ankle movements of both feet but no passive movement present in the left subtalar joint compared to the opposite side.

EXAMINER: Why don't you go on to test motor function of the feet?

Plantarflexion, dorsiflexion, inversion and eversion were tested whilst the patient was sitting in the chair. There was grade 5 MRC power compared to the opposite side. This was done very slickly as I had practised it before with somebody sitting in a chair

EXAMINER: What are you going to do for this patient?

CANDIDATE: Resection of the coalition.

EXAMINER: What about conservative management?

CANDIDATE: The patient is markedly symptomatic and I do not think conservative management will work although it may have a role in less severe cases.

Discussion

The subtalar joint may be rigid and any attempt to bring the foot into inversion aggravates pain and causes peroneal muscles to go into spasm.

- Calcaneal navicular accounts for two-thirds of cases; pain in the anterolateral aspect of the foot in the region of the sinus tarsi
- Talocalcaneal coalition (one-third of cases) presents with pain under the medial malleolus and reduced subtalar movement
- Talonavicular – Very rare

Depending on the nature of tissue involved the coalition can be a synchondrosis (cartilage), syndesmosis (fibrous tissue) or synostosis (bone).

Fifty per cent of the cases have bilateral affection, and multiple coalitions are present in 20% of the cases.

The natural history is unclear; it is not uncommon to find relatives with the condition who are entirely symptom free. Presentation features are generally either recurrent ankle sprains or else a vague ache in the foot. Management depends on the severity of symptoms. Mild cases may initially be managed conservatively; observe with shoe modification and change in activity. Established symptomatic cases with pain would be an indication for surgery, usually excision of the bar. If symptomatic degeneration is present one could consider a triple arthrodesis; however, this should not be done until late adolescence/adulthood. A talocalcaneal coalition is more likely to require arthrodesis owing to disturbance of the weight-bearing relationship of the foot (especially so if the coalition extends over >50% of the posterior facet).

Tibialis posterior tendon dysfunction/rupture

Memorandum 1

'In the standing position from the front, we notice that the patient's feet are externally rotated, that her hindfeet are in valgus and that her forefeet are abducted. When seen from behind, these deformities are even more obvious. She demonstrates the sign of 'too many toes' and requires assistance to rise up on to tiptoes. The hindfoot remains in valgus and the medial longitudinal arch remains flattened. She has even more difficulty in the single foot stance.'

'As she walks in bare feet one notices the heel strikes briefly and collapses into valgus before the forefoot goes into marked pronation during the stance phase. Toe-off is markedly reduced and the feet are in an attitude of external rotation with the forefeet abducted, which produces a roll-over gait.'

'I would like now to examine the patient's shoes. There is increased wear on the medial aspect of the sole, with roll-over onto the medial side of the toe. The uppers are not broken but there are orthoses in the shoes, which show some evidence of wear on the medial side.'

'We are now going to examine the patient in the seated position.'

'The lesser toes are held in a somewhat flexed position and there are small pressure lesions on the dorsal surfaces of the proximal interphalangeal joints. The great toe is slightly extended at the interphalangeal joint and there is dystrophy of the toenail.'

'There is some swelling around the tarsometatarsal region of the first ray. There is also some swelling around the outside of the left ankle. On examining the plantar aspect of her feet, there is obvious swelling on the medial aspect of the midfoot with overlying callosity formation. With the thumb on the neck of the talus, the hindfoot is put into the neutral position and when it is held there it is quite obvious that the forefoot is supinated.'

'When we examine her for tenderness, we find marked localized tenderness just below the tip of the left lateral malleolus.'

'Once more palpating the neck of the talus we can assess tibiotalar dorsiflexion and plantarflexion followed by talocalcaneal inversion and eversion. My hand now slides down to stabilise the hindfoot so that we can assess midfoot inversion and eversion. I am moving my hand down to stabilise the hindfoot so that we can invert and evert the midtarsal joint. In this case, midtarsal eversion is painful. The midfoot can be stabilised and the tarsometatarsal joint moved in a combined fashion and then individually. The metatarsophalangeal joint movements and the interphalangeal joint movements are full and pain-free. We will now look at the active movements of the foot and ankle. With the hindfoot held in neutral, active dorsiflexion is limited to neutral. If the hindfoot is allowed to go into pronation, dorsiflexion is markedly increased. There is a good range of plantarflexion and this can quite clearly be seen to be a combined movement of all the joints of the foot and ankle. There is a good range of inversion and eversion, but there is weakness of resisted inversion and the activity of tibialis posterior is substantially reduced.'

Memorandum 2

'On inspection there is a planovalgus deformity of the right/left foot. There is hindfoot valgus, forefoot varus, external rotation of the foot and forefoot supination. In addition, there is splaying of the forefoot. Medially, there is a swelling over the posterior tibial tendon, posterior and distal to the medial malleolus. From behind, the patient demonstrates the sign of too many toes.'

'There is a rigid/flexible flat foot and heel inversion is not/is occurring. The patient is unable to perform a single limb heel raise test (supported on wall).'

'I am feeling for any areas of tenderness or crepitus; there is anterolateral ankle and sinus tarsus pain. There is loss of power and weakness of inversion from an everted position. Passive range of motion of the hindfoot and midfoot is reduced to about half normal.'

Classification (Johnson and Strom, 1989[6])

Johnson and Strom devised three classification stages, with Myerson[7] adding a fourth:

Stage 1 (tendinopathy)

- Tenosynovitis, mild symptoms
- Medial ankle and foot pain
- Swelling without deformity
- Still able to perform a single limb heel rise test
- Normal tendon excursion
- Radiographs normal, MRI shows oedema around the tendon ± intrasubstance degeneration of the tendon

Stage 2 (flexible deformity)

- Deformity still remains flexible
- Pain and tenderness over the tendon; palpable enlargement/defect
- Too many toes sign; single heel raise test abnormal
- Radiographs show increase in the lateral talocalcaneal angle; MRI shows tendon degeneration ± discontinuity

Stage 3 (fixed deformity)

- Rigid flat foot deformity associated with hindfoot valgus and loss of subtalar joint motion
- Forefoot varus, absent single heel rise test, secondary degeneration

- Predominantly anterolateral ankle pain secondary to impingement of the calcaneus on the fibula and not anteromedial ankle or foot pain as seen in the earlier stages of the disease

Stage 4 (fixed deformity with generalized arthritic changes of the foot)

- Significant soft-tissue attenuation and loss of the deltoid ligament
- Valgus angulation of the talus
- Subtalar joint degeneration
- Fixed forefoot supination

Management

Stage 1

- NSAIDs, physiotherapy, insoles, immobilization in a short leg walking cast, tendon decompression and debridement

Stage 2

- Non-operative, orthotic arch supports, etc
- FDL tendon transfer with medial displacement calcaneal osteotomy, which redirects the strong pull of gastrocnemius muscle; lateral column lengthening.
- Cobb repair involves a split anterior tibial tendon transfer often combined with a medial displacement calcaneal osteotomy

Stage 3

- Subtalar or triple arthrodesis depending on the degree of joint arthrosis and age of the patient

Stage 4

- Tibiotalocalcaneal arthrodesis may be required if the tibiotalocalcaneal joints are incongruent and arthritic

Rarely

- Triple fusion and ankle arthroplasty with ligament reconstruction and tendon transfer
- Pan talar fusion

Deformities of the foot

Part of the problem with examination of the foot and ankle is that various terminologies are used in a very loose fashion and this can confuse even the best prepared of candidates. Despite efforts towards standardisation of terms there can still be considerable confusion.

In addition, the foot is a complex structure which has multiple joints and is, therefore, much more difficult to examine than single joint systems. This means that various parts of the foot interact with each other and create complex deformities that require discrete and definite descriptions and makes sequential sensible examination difficult to complete.

Equinus

The majority of weight is borne by the forefoot, the hindfoot remaining off the ground. The equinus deformity may be compensatory for either quadriceps or gluteus maximus weakness, or because of shortening of the limb. The equinus deformity may also be caused by contracture of gastrocnemius, ankle contracture or post-traumatic deformity.

Calcaneus

Here the weight is borne mainly by the hindfoot. The forefoot may have varying degrees of weight-bearing, but definitely below normal.

Varus

The weight is borne mainly on the outer side of the foot. This deformity is mainly at the hindfoot with associated forefoot pronation.

Valgus

Weight-bearing is borne mainly on the inner side of the foot. This deformity is of the hindfoot or of both the forefoot and hindfoot with associated forefoot supination.

Inverted foot

When the hindfoot and forefoot are both in a varus position, the deformity is termed an inverted foot. The accentuation of this position will gradually turn the sole towards the sky – Supination of the foot. In these positions, i.e. in inverted and supinated foot, adduction of the forefoot and plantar flexion of the ankle will coexist.

Everted foot

The hindfoot and forefoot are both in a valgus position. The outer part of the sole increasingly bears less weight. In this position, abduction of the forefoot and dorsiflexion at the ankle will coexist.

Forefoot position

Pes cavus

A normal foot has a medial longitudinal arch that is higher than the lateral one. When this normal proportion is exaggerated, the medial side of the foot tends to assume the shape of a high arch. It rarely occurs as a single deformity. It is a common accompaniment of equinovarus, equinus and clawing of the toes.

A forefoot cavus describes cavus owing to excessive plantar flexion of the first metatarsal, and results from the action of peroneus longus, when its action is preserved and its normal antagonists are weak, i.e. tibialis anterior in CMT disease.

Hindfoot cavus describes cavus which is caused primarily by increased calcaneal pitch, which may be seen in idiopathic cavus, but is also seen in polio where gastrosoleus is weak.

Pes planus

Collapse of the medial longitudinal arch. The normal concavity caused by the medial longitudinal arch is absent and instead the medial side of the foot may bulge as a medial convexity, particularly on weight-bearing.

The relationship of the forefoot to the hindfoot

- Forefoot neutral: With the heel in neutral, the plane of the metatarsals and the plane of the heel are colinear
- Forefoot varus: Forefoot supination: With the heel neutral, the lateral border of the forefoot is lower than the medial border of the forefoot
- Forefoot valgus: Forefoot pronation: With the heel neutral, the medial border of the forefoot is lower than the lateral border of the foot

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Chapter 14

Paediatric clinical cases

Sattar Alshryda and Philip Henman

Introduction

Preparation for the FRCS paediatric orthopaedic section is confusing not only for the candidates but for the examiners as well. The GMC has outlined and approved the depth and breadth of knowledge required for specialty training in trauma and orthopaedic surgery including paediatric orthopaedic. The expected levels of knowledge are indicated on the following four-point scale:

1. knows of
2. knows basic concepts
3. knows generally
4. knows specifically and broadly.

Figure 14.1 is a snapshoot from the GMC documents for specialty training in trauma and orthopaedic surgery. It is evident that candidate should achieve level 4 in common topics such as developmental dysplasia of the hip (DDH), slipped upper femoral epiphysis, leg length discrepancy, clubfoot and scoliosis.

Having helped hundreds of candidates to pass their exams, we rarely come across candidates who attained the above levels; nevertheless, most succeed to pass the exam! Based on previous candidates experience collected over the years, we have rewritten the two paediatric orthopaedic chapters of this book to cover most topics that featured in these exams. Both chapters are complementary to each other. Some repetition is unavoidable and may be advantageous. We have added clinical photographs to create a mental association to these clinical scenarios.

Table 14.1 summarizes cases that have been featured in the clinical section of the exam. The wide range of these cases can be disconcerting and we will try in this chapter to guide you on how to tackle these or similar cases successfully even if you know little about the condition. Some of these topics are covered in other section of the book such as scoliosis, congenital hand deformities and trigger thumb.

Approach

Children come to clinic (and exam) because of one or more of three **complaints**:

1. Deformity
2. Altered function (commonest is gait abnormality)
3. Pain

In intermediate cases, a thorough but relevant history is essential to reach a diagnosis (and pass the exam!). In short cases the diagnosis is usually obvious (listen to the examiner carefully. The answer is in the question!). Do not let the stress of the exam prevent you interacting with the child. (A candidate reported that a child told him the diagnosis 'I have Perthes' disease'!).

The duration of symptoms, mode of onset, history of any injury, frequency and timing of symptoms, aggravating/relieving factors, any functional impairment, previous investigations or treatment received should be noted.

In children with cerebral palsy (CP), a thorough **prenatal and perinatal history** is expected. A history of bleeding during pregnancy, maternal diabetes and reduced fetal movements during late pregnancy, breech presentation, difficult labour, premature birth, and jaundice at birth are significant factors to be enquired about.

Enquire about **developmental milestones** (Table 14.2); e.g. when did the child first sit and walk?

It is useful to enquire if other members of the **family** have similar problems. A number of orthopaedic clinical conditions run in families; e.g. DDH, clubfeet, pes cavus, etc. Finally, a history of past illnesses and hospitalisations completes the history.

Examination of a child

Start examination whilst taking the history; observe the child activities, interaction with people and environment. Notice if there is any walking aids, wheelchairs, braces or SOS bracelets. School uniforms may indicate whether the child goes to mainstream schools or schools for children with special needs.

Like any other examination, it usually involves:

- General assessment
- Specific muscular-skeletal examination related to the main complaint

The examiner usually guide you on what is required to assess (so listen to the question carefully and do not hesitate to ask for clarification if the instruction was not clear).

General assessment

This serves two purposes:

- Support the primary diagnosis; for example, finding plagiocephaly and congenital torticollis will support the diagnosis of a dislocated hip

Competence Levels	
1 = Knows of	3 = Knows generally
2 = Knows basic concepts	4 = Knows specifically and broadly

Topic	CORE	ST3-ST8	SPECIALTY INTEREST
PAEDIATRIC ORTHOPAEDIC SURGERY			
Basic Science			
Anatomy			
Growth of bones, physeal anatomy and its application to fracture types and pathological processes and infection in particular	2	4	4
Anatomy of bones and joints in the growing child and its application to growth and deformity	2	4	4
Neurological processes involved in the production of deformity e.g. spina bifida, cerebral palsy and muscular dystrophy	1	3	4
Clinical Assessment			
History and examination of the child	2	4	4
Involving the parents in the assessment	2	4	4
Assessing the disabled child	1	3	4
Investigations			
Indications for plain x-ray, arthrogram, CT, MRI and the how to interpret the images	2	4	4
Indications for the use of ultrasound and nuclear imaging	2	4	4
Limitations of investigations in paediatric practice	1	3	4
Critical Conditions			
The painful hip			
Treatment			
Operative			
Fractures (including non-accidental injury) and growth plate injuries and their sequelae	2	4	4
Bone and joint infection	2	4	4
Common childhood orthopaedic conditions, e.g. irritable hip, anterior knee pain	2	4	4
Slipped epiphysis	1	4	4
Perthes' disease	1	3	4
Developmental dysplasia of the hip	1	4	4
Talipes	1	4	4
Scoliosis	1	4	4
Forefoot deformities	1	4	4
Congenital hand abnormalities	1	3	4
Osteogenesis imperfecta	2	3	4
Skeletal dysplasias	1	3	4
Tarsal coalitions	1	3	4
Torticollis	1	3	4
Leg length discrepancy	1	4	4
Non-operative			
The treatment of normal variants such knock knees, flat feet, femoral anteversion	1	3	4
Orthoses	1	3	4
Rehabilitation of the child	1	3	4
Determining physical disability	1	3	4
Screening for congenital abnormalities	1	3	4

Figure 14.1 GMC documents for specialty training in trauma and orthopaedic surgery

- Find other orthopedic problems that may or may not be related to the presenting problem but requires attention. For example, finding torticollis may require specific treatment. I am often referred patients with benign feet deformities but examination uncovers a dislocated hip. This can be simulated in the exam

General assessment usually involves three aspects:

- General physical signs (Figures 14.2–14.5)
- Posture
- Gait

With some training and experience, these three aspects can be covered simultaneously rather than sequentially.

Eliciting general physical signs and correctly relating them to an underlying condition can be very impressive in the exam. The following list suammrizes common physical signs:

- Plagiocephaly (DDH)
- Dysmorphic features (dysplasia and genetic syndromes)
- Eyes (slanted with epicanthal fold in Down's syndrome, blue sclera in osteogenesis imperfecta)

Table 14.1 Common, frequent and rare paediatric cases that featured in previous FRCS exams

Common	Frequent	Rare
Cerebral palsy	Long bone deformity (traumatic)	Leg deformity (congenital)
Knee deformity	Achondroplasia	Multiple epiphyseal dysplasia
Limb length discrepancy	Tarsal coalition	Sprengel's shoulder
Toes deformity	Radioulnar synostosis	Trigger thumb
Clubfoot	Madelungs deformity	Patellar dislocation (congenital)
Scoliosis	Patellar dislocation (traumatic)	Congenital absent limb
Bony exostosis	Proximal femoral focal deficiency	Perthes' disease
Neurofibromatosis	Arthrogryposis	popliteal cyst
	Osteogenesis imperfecta	Congenital dislocation of the radial head

Table 14.2 Normal developmental milestones

Age	Motor skills	Social skills
3 months	Lifts head up when prone	Smiles when spoken to
6 months	Sit with support, head steady when sitting	Laughs and smiles spontaneously
9 months	Sit without support Crawl	Waves 'bye-bye', vocalize 'ma-ma or 'da-da'
1 year	Walks with one hand support	Starts cooperating with dressing
2 years	Runs forward	Use 3-word sentences, match colours
3 years	Jumps in place	Dresses oneself, put shoes own
5 years	Hops	names four colours; counts 10 objects correctly
6 years	Skips	Does small buttons on shirt; ties bows on shoes

- Large (may be asymmetrical) tongue (Beckwith–Wiedermann syndrome)
- Height (short in dysplasia; tall in Marfan's syndrome)
- Weight (overweight in Prader–Willi syndrome)

Figure 14.2 General signs associated with DDH. Top left: Congenital torticollis, top right plagiocephaly, and bottom pictures calcaneo-valgus feet

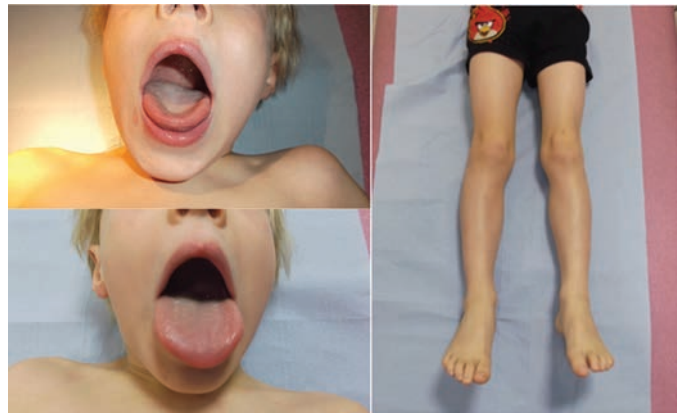

Figure 14.3 Hemi-hypertrophy. Right hemi-hypertrophy (including the tongue) in a child with Beckwith–Wiedermann syndrome. Do not forget to examine for internal malignancy (or its treatment)

- Hypertrophied calves (Duchenne and Baker muscular dystrophy)
- Long slender fingers (arachnodactyly) may be a sign of Marfan's syndrome
- Café-au-lait spots, axillary freckling in neurofibromatosis
- Haemangiomas may suggest Klippel–Trenaunay–Weber syndrome
- Hairy patches, skin tags or sacral dimples may indicate underlying spinal pathology
- Thickening of the ankles, wrists and knees, rachitic rosary in Rickets
- Feet deformity can be a sign of packing disorder (DDH). A cavus foot deformity is a common feature of diastematomyelia
- Nail abnormalities in nail–patella syndrome

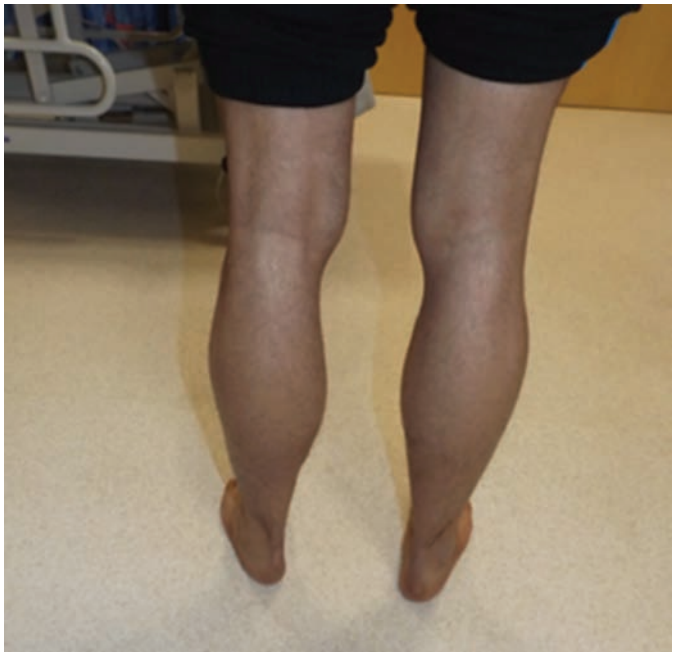

Figure 14.4 Pseudo-hypertrophy in a child with Becker's disease

- Generalized joint laxity is valuable in assessing a flatfoot or patellar dislocation (see Beighton's score, Table 4.1)
- Posture
  - The standing posture
  - The curvature of the spine
  - The level and contour of the shoulders
  - The level of the anterior superior iliac spine (ASIS)
  - Limb symmetry (carrying angle, geno-varum and valgum, muscle girth)
  - For hindfoot alignment – Valgus or varus
  - For evidence of tiptoeing, flat foot or cavus deformity
- Gait
  - Any characteristic abnormal gait (toe-walking, antalgic gait, waddling gait, etc)
  - Foot and ankle movements and positions
  - The knee movements and positions
  - Hips and pelvis movements and positions
  - Head and shoulder movements and positions
  - Upper limb swings

A classic example of the importance of general assessment in exams and real life is finding features suggestive of Down's syndrome in a child who is referred with a dislocated hip. This will impact on preoperative work up (heart and cervical spine) consenting (high recurrence rate) and surgical technique (femoral varus osteotomy).

Specific musculo-skeletal examination

This refers to examinations of individual regions or joints (such as neck, shoulders, elbows, wrists, hands, spine, hips, knees, ankles and foot). These are essentially as in adults (look, feel, move and special tests) with some differences which we will explain in the relevant sections below.

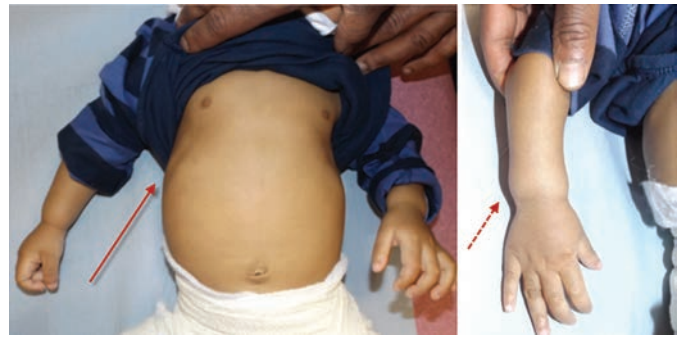

Figure 14.5 Rickets. Thickening of the wrists and rachitic rosary in a child with rickets

As with taking history, examination should be focused on aspects relevant to the main complaint. Although it is good to follow a system of examination, it is impossible (and not advisable) to do every test on a child.

Cerebral palsy

May come up as an intermediate case, it is unusual as a short case. It is possible you will be shown a gait analysis video at the end of the case and asked to describe it. Don't panic, simply describe what you see. If the patient is just walking up and down, say so and try and describe the gait (see below). If the patient appears to have ping-pong balls or some other marker stuck to their joints this is *motion analysis* or a *kinematic study*. If the patient seems to be made to walk over a marked panel of the floor and virtual lines shoot up as they step on it, this is a force plate to measure *kinetics*. If they have trailing wires the chances are they are having electromyography.

When examining the patient make a conscious effort to talk directly to the patient and not through a carer. Minimize patient movement to avoid undue distress. Ask how well the patient can walk before asking them to move. Examine in order:

- General examination
  - General physical signs
  - Posture
  - Walk
- Specific musculo-skeletal
  - Examine relevant aspects of every joint/segment by look, feel, move and special tests

General examination

General physical signs

Comment on presence of wheelchair, walking aids, communication devices and orthosis, e.g. ankle–foot orthosis (AFO), knee–ankle–foot orthosis (KAFO), wrist splints, spinal brace, etc

Look at trunk for gastrostomy or a subcutaneous reservoir for a baclofen pump
Pattern of involvement

Anatomical

Monoplegia: affecting one limb only – Rare
Hemiplegia: affecting one side. Upper limb classically more affected than lower
Diplegia: predominantly lower limbs
Quadriplegia or total body involvement: May well be in wheelchair

Movement disorder

Spastic: increased reflex reaction
Athetoid: linked writhing movements

- Ataxic
- Hypotonic
- Mixed

Dystonia is another form of movement disorder which is described where the pattern of abnormal posture changes seemingly at random

Posture (standing/sitting)

Note posture, any obvious deformities, the position of the upper limbs, trunk alignment, leg length discrepancy and foot position
Comment on spine alignment. Scoliosis is more common the more severe the movement disorder. There are two main patterns of scoliosis in cerebral palsy, an 'idiopathic scoliosis' type curve with vertebral rotation (rib hump) and possible compensatory curve – And a 'long C' or neuromuscular curve which classically describes a single curve from neck to lumbosacral junction

Walking

Observational gait analysis need not be as scary as it sounds – Just describe what you see but in a logical order. Practice on your friends and neighbors
Start with big obvious things – Need for walking aids, orthoses, speed, symmetry. Then describe what happens from the bottom up as the patient walks
Feet: Foot progression angle, initial contact, foot shape and comment on the three rockers of the foot
Lower limbs: Describe the joint positions and movement during gait cycle, ankles, knees and hips
Pelvis: Level or tilted? Lateral movements when walking? Trendelenberg?
Trunk: Posture and movements. Flexion, lordosis, lateral tilt?
Upper limbs: Posture, swinging or held stiffly. The flexed posture of the arm in hemiplegia may not be evident until the patient walks or performs more complex tasks
Shoulders and upper body: Posture, excessive movement?
Head: Is it steady, does it move from side to side or up and down?
Easy!

Specific muscular-skeletal

Examine the patient on the couch (first supine then prone – If patient is able to do so).

Couch

Look, feel, move and special tests– Nothing clever needed.

Hip joint examination

Although hip examination in children is similar to that of an adult, children pelvis is small and can be easily tilted causing inaccuracy when assessing the hip's range of movement (ROM). This is particularly true in children with CP; hence, two people are recommended to conduct the examination. In the exam, do not hesitate to mention this and if one of the examiners offers help, try to utilize him or her (in our clinic we get help from a nurse or one of the parents).

Look for any visible deformity, discoloration and scars. Look and feel for dislocated femoral heads in buttock. Dislocated hips are more frequent in children with total body involvement, significantly less frequent in diplegics and rare in hemiplegics.

Feel for temperature and tenderness.

Move (if the child age and condition allow, check active before passive ROM). Ask the child to flex the right hip (record the active flexion), if it is not full, try to flex it fully (record the passive flexion). Do the same for other ROM. to stabilise the pelvis with one hand whilst assessing the ROM particularly abduction/adduction.

Clinicians often talk about R1 and R2 and it is useful to know of (and even more impressive to demonstrate) them. R1 and R2 stand for range of motion 1 and 2 respectively. Depending on the speed you use to check the range of motion of a limb, you may feel a catch where the limb stops moving but with gentle and slow force it moves again to its final range of motion. R1 refers to range of motion of the limb up to the catch whereas R2 refers to the final range of motions achieved. The difference between R1 and R2 helps differentiate between spasticity and contractures. The higher the difference the more spastic the muscle is and the more likely it responds to Botox.

Special tests

1. Thomas' test to assess the hip flexion contracture. Flex both hips maximally with the knees flexed. Put a hand under the lumbar region to ensure flatting the lumbar lordosis, but also ensure no pelvic tilting. Allow the side being assessed to extend fully. The angle between the couch and the thigh is the degree of flexion contracture
2. Staheli's (prone extension) test is an alternative for Thomas' test with an advantage of being able to measure the actual extension. Patient is placed prone and the legs allowed to dangle over the edge. With the contralateral hip flexed, extend the ipsilateral side to the degree that causes

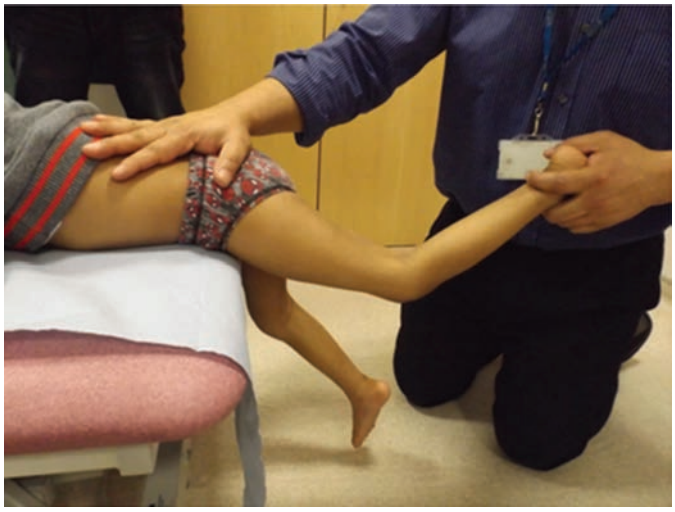

Figure 14.6 Staheli's (prone extension) test. Child is placed prone position, flatten spine by adjusting the pelvis position and assess hip extension. In this child there is 30° of fixed flexion deformity

the pelvis to elevate. The degrees short of full extension equal the degrees of fixed flexion deformity (Figure 14.6)

3. Adductors contractures and Phelp's test. Child is supine, stabilise the pelvis as above. the degree of hip abduction is measured with the hip and knee in extension (normal = 45°). Adductor contracture <30° is a risk for progressive hip subluxation. Repeating the test with the knee flexed to exclude the medial hamstring (MH) which crosses both joints. If there is no MH tightness, the values should be similar (Phelb's test)
4. Ober's test (iliotibial band (ITB) contracture) Child is on the side with the spine straight. The hip to be tested (the upper most) is then flexed to 90° (with the knee flexed to a right angle through the test), fully abducted, and brought into full hyperextension and allowed to adduct maximally. The angle of the thigh and a horizontal line parallel to the examination table represents the degree of abduction contracture. A normal limb will drop well below this horizontal line. If there is abduction contracture, the hip cannot be adducted to neutral position

Then help patient to turn and lie on his belly (prone position) to continue assessment:

5. Duncan–Ely test to assess rectus femoris contractures). Child is in prone and the knee is gradually flexed. The examiner feels the spasticity and resistance of the rectus muscle and observes the elevation of the ipsilateral hemi-pelvis.. The elevation of hemi-pelvis is usually subjectively graded into (+, ++ and +++)
6. Assess the lower limb rotational profile (See in-toeing and out-toeing)

Knee joint examination

Look for any visible swelling, deformity, discoloration, callosities and scars

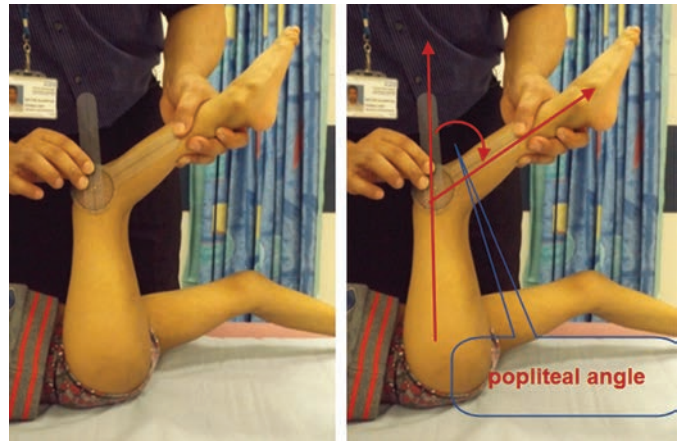

Figure 14.7 Popliteal angle. Foot and ankle examination

Feel for temperature and tenderness. Find the point of maximum tenderness and relate it to a possible pathology. Check for the patellar position (alta or Baja) and for mal-tracking

Move

Knee joint is a sloppy hinged joint allowing mainly with flexion–extension motion (slight rotation and translation is possible but very limited). The normal knee's ROM is 0–150°. The knee extension should be measure with the hip is extended (which relaxes the hamstring muscles) gives the fixed flexion deformity of the knee, which is usually caused by the capsule (or bony deformity).

Special tests

The popliteal angle is performed with the child supine and the hip is flexed at 90° with the contralateral hip is extended (Figure 14.7). The knee is then extended. The angle between the vertical line and the tibia is the popliteal angle (normal <20°). Some authors measure the angle between the femur and tibia as the popliteal angle. Other authors flex the hip to 45° rather than 90° (mimicking the hip flexion in normal gait) and measure the angle. The latter is called the modified or the functional popliteal angle and probably more relevant clinically.

Anterior pelvic tilt may increase popliteal angle by tightening the hamstring muscle, so measuring the difference between the measurements with the contralateral hip extended and those with the hip flexed is the Hamstring shift test. Flexing the hip beyond the hip fixed flexion deformity ensures the pelvis is not anteriorly tilted and gives a more representative measure of hamstring tightness.

SLR is another way to measure the degree of hamstring contracture by raising the limb keeping the knee in full extension (as in spine examination). The angle between the extremity and the table is measured (normal <70°).

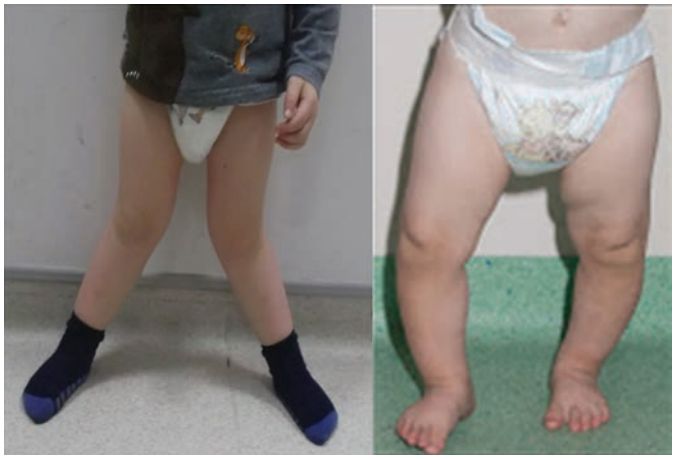

Figure 14.8 Bowed legs and knocked knees

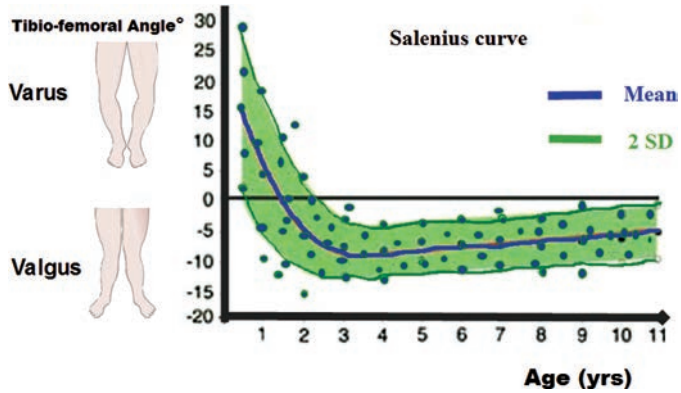

Figure 14.9 Salenius curve

Look

- Gait
  - A normal heel strike/toe-off gait?
  - Is it of normal height? (High stepping gait indicates foot drop
  - Is the gait smooth and symmetrical?
- Any scars, swellings?
- Deformities (flat feet, high arched feet, hallux valgus or varus, toe clawing)?
- Examine the patients shoes – Evidence of asymmetrical wearing may indicate abnormal gait
- Foot/ankle symmetry – Heel alignment – Valgus or varus deformity?
- Achilles tendon – Any obvious swelling, discontinuity/erythema?

Feel for temperature and tenderness. Find the point of maximum tenderness and relate it to a possible pathology.

Move

Ankle plantar flexion (30–40°)
Ankle dorsiflexion (15°)

Subtalar joint motion – Grasp the talar head between thumb and index finger of one hand and heel with the other – Turn sole towards midline (to assess inversion) and outward to assess eversion. No movement is tarsal coalition or joint fusion.

First MTP joint dorsiflexion (65–75°) to allow adequate third rocker.

Special tests

Silverskiold test: Child is supine, the heel inverted to lock the subtalar joint (prevent any dorsiflexion through midfoot). The degree of dorsiflexion is measured with the knee flexed and extended. Flexing and extending the knee relaxes and tightens the gastrocnemius muscles only and not the soleus. This allows finding which muscles are tight. If the tightness involves the gastrocnemius only it can be released selectively.

Look for midfoot break which is common in children with tight gastrosoleus muscle.

Knee deformity (genu varus/genu valgus)

The unusual natural history of coronal lower limb alignment is a popular topic in the exam. It could face you in a clinical or viva station. The candidate is expected to know the natural history of coronal leg alignment and signs of deviation from physiological (Figure 14.8).

Salenius and Vankka[1] studied the coronal alignment (tibio-femoral angle) of lower limb in children and found that children are born with genu varum (bowed leg) of about 15°, which decreases through infancy. The legs become straight around 18 months of age, then progress to valgus (knocked knees) reaching maximum valgus of average 10° at around 3–4 years of age. Valgus then gradually decreases reaching the adult value of 5° around age 8 (Figure 14.9). With a standard deviation of 8°, meant that 95% of newborn children could have a tibio-femoral angle of –1° (i.e. valgus) to as high as 31° and still within normal. Therefore, the trend is more important than a single reading.

It is useful to remember that genu varus is more likely to be pathological if:

1. Present after 2 years
2. Unilateral or asymmetry of >5°
3. Associated with shortening of the limb (or stature)
4. Severe (beyond 2 SD of the mean as per Selenius chart)
5. In child with obesity.

And genu valgus is more likely to be pathological if:

1. Severe (intermalleolar distance >10 cm at 10 years or >15 cm at 5 years)
2. Unilateral

Always consider pathological causes of genu varus and valgus when encountered in clinics or exams (Table 14.3).

Examination

Overall inspection: ask the child to stand. Look for facial dysmorphism, the height and weight of the child and plot

Table 14.3 Causes of genu varus (bowed legs) and genu valgus (knocked knees)

Bowed leg	Knocked knees
1. Physiological	1. Physiological
2. Tumours such as osteochondorma	2. Tumours such as osteochondromas
3. Skeletal dysplasia	3. Skeletal dysplasia
4. Blount's disease	4. Primary tibia valga
5. Infection	5. Infection
6. Trauma	6. Trauma
7. Metabolic (vitamin D deficiency, fluoride poisoning, osteogensis imperfecta)	7. Renal osteodystrophy
8. Focal fibrocartilaginous dysplasia	8. Neuromuscular disorders (Polio) and tight ITB

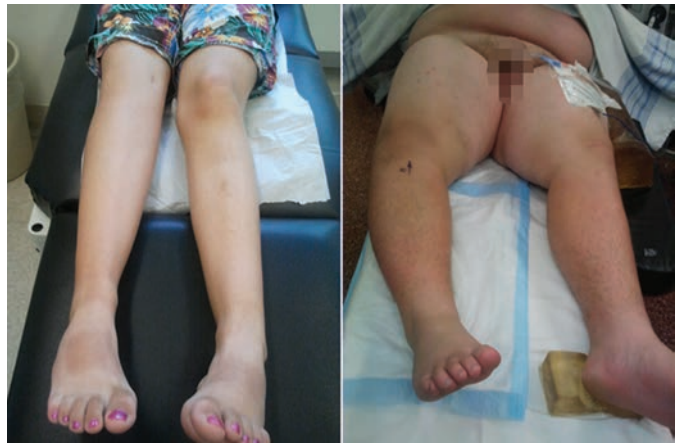

Figure 14.10 Asymmetrical knee deformities. The left picture shows a girl with valgus deformity coming from the femur following hemiepiphysiodesis while the right is Blount's disease with the deformity in the tibia. To ascertain the site bending the knee will correct the tibias alignment on the left but not on the right

them on an appropriate height–weight chart (most centres now use electronic charts).

Children with bone dysplasia or metabolic disease are frequently of short stature.

Examine lower limb alignment quickly from front, side and back. While at the back, quickly check spinal alignment – Tell patient and carer what you are doing. Carefully inspect the limbs for scars, cutaneous markings, dimples, etc. Check leg length.

Assess alignment when patient supine on examination couch. Square the pelvis on the couch. Rotate the legs so that patella points straight upwards. Now look at the leg – Is it in varus or valgus? Is it correctable?

If there is a noticeable deformity, where is it? Is it in the femur, tibia or in the joint? For instance in Blount's disease it is often clear that there is an angulation below the knee, whereas in physiological bowing, the whole leg describes a gentle curve (Figure 14.10). If in doubt, bend the knee, this will dissociate these three places and make the site of deformity clearer. Assess the knee for ligament laxity.

If asked to measure the angulation across the knee, try not to emit any audible signs of distress. Examiners hate that.

Palpate the ASIS and offer to mark the site with a pen. (It would be unwise to actually do so.) Place the goniometer centre on the centre of the patella. Line one limb up on the ASIS and the other on the line of the tibial crest or midpoint of the ankle. This is of course terribly inaccurate.

Intermalleolar distance can be helpful and reproducible when performed by the same clinician and in bilateral symmetrical genu valgus. It may not be accurate if it is unilateral or the patient is obese.

Assess the lower limb rotational profiles (see below) as rotational deformities are often associated with genu varum or valgum. Moreover, they should be considered in any surgical correction plans.

Do not forget to look at parents' legs (and actually ask if there is a similar problem in the family). If this is the case, suspect autosomal dominant diseases such as hypophosohatemic rickets, multiple exostosis and bone dysplasia).

Don't be pushed to offer a surgical plan without radiological assessment. You should ask, if prompted, for standing leg alignment x-rays taken. Radiographs are also useful in documenting the site and size of the deformity and enable the aetiology to be diagnosed in some cases.

Investigations

- Radiological assessment
  1. Tibio-femoral angle as per the Selenius curve
  2. Metaphyseal–diaphyseal angle of Levine and Drennan (normal $<11°$ but abnormal $>16°$)
  3. Metaphyseal–epiphyseal angle (normal $<20°$)
- Biochemical if metabolic diseases are suspected

Treatment

Treat any underlying pathology if they exist.

Observation: Usually the correct first line until a trend has been established

Bracing: although debated it is still practiced, particularly for juvenile tibia vara; may be successful in mild deformity.

'Guided growth': this is the modern and expensive reincarnation of physeal stapling. Hugely popular at the moment and it works. Learn to recognise an eight-plate when you see one (Figure 14.11). The advantage of the eight-plate is the reversible breaking effect on the physis.

Osteotomy: when all else has failed. Choice is between single stage correction with internal fixation or gradual correction using a ring fixator. It is for this reason that Charles Taylor and his Spatial Frame deserve beatification.

Table 14.4 Causes of leg length discrepancy

Causes	Examples
Congenital	1. Longitudinal limb deficiency (hemimelia) 2. Hemihypotrophy or hemihypertrophy (do not forget association with internal malignancy) 3. Dysplasia 4. Metabolic diseases
Acquired	1. Trauma 2. Infective (osteomyelitis involving the physis, gonococcal septicemia) 3. Neoplasia 4. Inflammatory diseases (juvenile rheumatoid arthritis) 5. (In adult, joint replacement)
Neuromuscular disease	1. Cerebral palsy 2. Poliomyelitis (NB. In these conditions, limb equalisation including shoe raises may be harmful)
Apparent	1. Joint dislocation (DDH) 2. Joint contractures

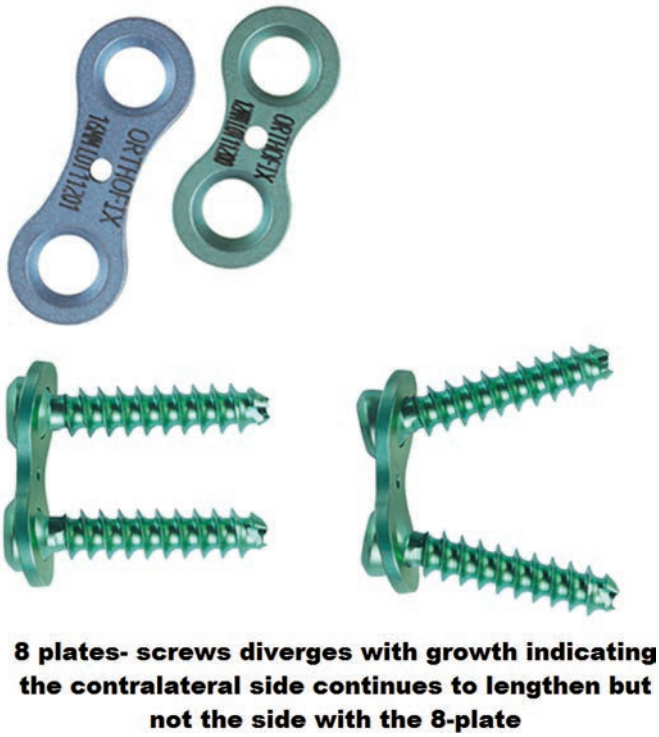

Figure 14.11 The eight-plate

Examination corner

Short case 1

A candidate was to examine the knees of a 9-year-old overweight school girl who was teased at school because of the way she walks. He commented on the weight of the child and the clear valgus of both knees. He also noted bilateral large carrying angles. He asked the patient to walk and noted that she has flat feet on both side.

He examined the knees with the patient standing from the front, side and behind. He could not see any scar. He asked the patient to lie on the couch and started measuring the angles by identifying the ASIS, the patella and mid of the ankle. He initially forgot to measure the intermalleolar distance, but he admitted that he should have checked it when she was standing. The examiner asked *'How would that help you?'* The candidate answered that all signs showed that this girl has a physiological genu valgus and the intermalleolar distance would help me to quantify this. The examiner asked *'Show me how you would measure the distance'*. The candidate asked the patient to stand with both knee caps pointing forward and the knees touching each other, identified both malleoli and measured the distance between them which was 7.5 cm.

The examiner asked *'How would you manage this patient?'* The candidate said that he would like to obtain a radiograph. The examiner was not impressed, *'You just said everything was within normal limit, so do you think the x-ray is justified'?*

The candidate replied that he was not sure, but he felt that he would discharge this patient to GP care with advice to be re-referred if the situation worsens. Hence, he felt that radiograph would provide extra assurance for him as a clinician and to the child and her family that there is nothing serious. He probably would not x-ray her knee if he arranged to bring her back to his clinic.

Leg length discrepancy

This is a very common referral to children's orthopaedics; it can present as an intermediate or short case in a child or adult. History should be directed to find the cause (Table 14.4), the trend (worsening or improving) and any prior treatment and outcomes.

Five objectives of clinical assessment:

1. Size of discrepancy
2. Site of discrepancy
3. Status of the joints
4. Is the discrepancy real?
5. The cause of the discrepancy if not yet available.

General assessment

General physical signs

Look at the patient in general, any dysmorphism, facial and tongue asymmetry (Beckwith–Wiedemann syndrome), skin markings, and signs of previous surgery. Look briefly at the upper limbs for deformity or obvious shortening. Leg length discrepancy is common in spastic hemiplegia. Upper limb length difference may not be easy to evaluate so don't waste time if not obvious.

Posture

Standing: equinus of the ankle on short side or flexed knee on the long side. Look for pelvis obliquity.

Gait

Short leg gait, comment on rhythm and trunk position. Look at the shoes for adjustments and wear pattern.

Specific muscular-skeletal

Leg length evaluation

Patient standing in socks. Sit or crouch in front of the patient – Use a chair if available, it makes it look as if you're used to doing this, is more comfortable and avoids the appalling possibility of losing your balance and toppling into the patients lap.

Ask patient to stand straight with both knees straight. Place thumbs firmly but not painfully on anterior superior iliac spines. Don't tickle. This will tell you which is the long leg and a surprisingly good idea of the discrepancy. Ask for standing blocks, they will be marked in mm. Place your best guess height in blocks under the foot of the short leg and check ASIS again. Adjust block height until the pelvis is level with knees straight. This is the functional leg length difference. With the pelvis level, step back and assess leg alignment. Beware of fixed hip flexion and/or adduction (see next step). Turn patient with back to you, or walk round them. Still with pelvis level and knees straight, inspect from the back and take a quick look at the spine. Patients with hemihypertrophy and other conditions may have leg length discrepancy **and** scoliosis.

Couch evaluation

If more is expected of you, direct patient to initially sit on the couch, look whether scoliosis corrects. Comment on knees and feet position when the patient is sitting, sometimes it is very clear that one side is shorter. Lie patient supine on the couch. Square the patient's pelvis to the couch and comment on any obvious deformity.

Swiftly assess joints (hips, knee and ankles) range of motion to exclude joint contractures as a cause of leg length discrepancy.

True and apparent shortenings are terms are often used in the contest of leg length discrepancy. True shortening is referred to the affected limb is physically shorter than the other leg. It is measured using a tape the ASIS to the medial malleolar tip while both lower limbs are in identical positions and the pelvis is square.

The apparent shortening refers to the sum of the true shortening plus the shortening due to fixed deformities. It is the apparent shortening that matters to the patient. This measurement helps in assessing the extent of natural compensation developed for concealing the actual disparity.

By now, the size of discrepancy is appreciated and would be confirmed by radiograph. The next step is to determine the site(s) of the discrepancy

Table 14.5 Management options for leg length discrepancy

Leg length discrepancy	Options
0–20 mm	Conservative: Nothing, insole or shoe raises
20–50 mm	Consider epiphysiodesis of the opposite side at appropriate age, unless the child is already very short, when you may consider lengthening of the short limb after appropriate consultation After skeletal maturity, acute shortening of the long limb is an option
>50 mm	Offer lengthening of the short limb ± epiphysiodesis of the long limb

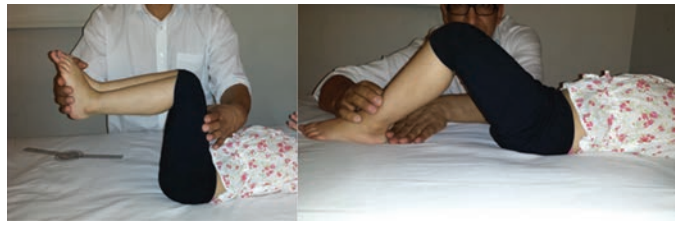

Figure 14.12 Galeazzi's test

Place thumbs on ASIS and middle finger tips on the greater trochanter (GT) simultaneously to assess hip position. The difference in distance between ASIS and GT **suggests discrepancy proximal to GT**. This can be measured more precisely by drawing Bryant's triangle and measure the perpendicular distance between the tip of greater trochanter and another line dropped from ASIS perpendicular onto the bed. This is often called Bryant's test.

Flex hips to 90° and look at heights of the knees (Galeazzi's sign; Figure 14.12) to assess femoral length, place heels together and look at knee height to assess tibial length. Inspect the feet, a dysplastic or traumatized hindfoot may lose you a centimetre or more in height. Offer to use a tape measure to document the various segment lengths using the usual bony landmarks. This is pretty dreary stuff and of little practical use, but your examiner might like watching you do it.

If you suspect congenital femoral dysplasia and/or fibular hemimelia, examine the knee for ligament insufficiency, particularly cruciates.

Management (Table 14.5)

It depends on current leg length discrepancy, predicted leg length discrepancy at skeletal maturity and patient's perception of discrepancy.

- Current leg length discrepancy
  - Clinical
  - Radiological
    - Teleoroentgenography: single exposure hips to ankles over radio-opaque ruler

  - Orthoroentgenography: Separate exposures centred on hip, knee, ankle over a single film with a ruler
  - Parallel beam scanogram: Separate exposures centred on the hip, knee and ankle over a radio-opaque ruler on separate films
  - CT scanogram (becoming most popular)
- Predicted leg length discrepancy at maturity
  - Menelaus rule of thumbs
  - Moseley straight line method
  - Eastwood and Cole method
  - Paley's multiplier method
- Patient's perception of discrepancy
  - How tall the child is? Is he taller or shorter than average?
  - How tall are the parent or family members?

Examination corner

Short case 1

Patient is an adolescent male in shorts. Be friendly and polite. Please don't try to be down/in with the kids – The children hate that.

EXAMINER: Would you please evaluate this gentleman's leg length discrepancy? Talk me through your findings as you go.

CANDIDATE: On inspection the patient looks generally fit and well. He has a visible shortening of the left leg and tends to stand with the right knee flexed. I can see surgical scars on the left thigh and what appear to be pin-site scars consistent with previous external fixator treatment. I see that his left trainer has what looks like a 2 cm build up on the sole.

Would you mind walking to the wall and back? He has a short leg gait, but no obvious discomfort. The left knee is in a little valgus compared to the right and the entire leg seems slimmer. Could you stand with your feet level and your knees straight please? (Assess pelvic tilt by palpating ASIS) There is a significant leg length discrepancy; could you pass those blocks please? (*Adjust standing blocks to level pelvis*) On block standing there seems to be a 4 cm leg length discrepancy, short on the left. Examining the patient from behind, with his pelvis level the spine is straight. Would you please lie down on the couch on your back? His hip movements are good. The majority of the shortening is in the femur on the left though the tibia is a few millimeters short too. Foot and ankle are good. Knee examination demonstrates an exaggerated anterior draw and Lachmann's test on the left consistent with cruciate ligament deficiency.

EXAMINER: Good. Can you guess the clinical background to these findings?

CANDIDATE: These findings would be consistent with a patient with congenital femoral deficiency who has had an episode of femoral lengthening during childhood and is now approaching skeletal maturity with further relative shortening of the left leg.

EXAMINER: Very good. I can tell you he has already had epiphyseodesis of the right distal femur and proximal tibia and is 16 years old on Tuesday. How would you manage this now?

CANDIDATE: I would discuss the options with the patient. He could be managed with a shoe raise if he has had enough surgery. Alternatively the options are to lengthen the left femur again or to shorten the right femur by, say, 25 mm, which would give him a more acceptable discrepancy.

EXAMINER: If he were to opt for lengthening, how would that be done? Are there any particular risks in this patient?

CANDIDATE: The standard method is by using an external fixator, either circular frame or monolateral. I know there are lengthening intramedullary nails available but I understand they are both extremely expensive and essentially experimental. A particular concern in this patient is his cruciate deficiency, one would have to take great care to avoid posterior subluxation of the knee during lengthening, and I would like an x-ray of the hip too before making any further decisions.

Short case 2

In his first short case, a candidate was asked to examine a 5-year-old girl with right leg shortening. She was sitting on the couch with significant shortening of the right leg, mainly the thigh.

- I could see her shoes underneath the couch fitted with a massive shoe raise (probably 8 cm). Quick evaluation of upper limbs revealed no obvious abnormality. After I mentioned these finding, I was asked about the possible diagnosis, I answered proximal focal femoral deficiency (PFFD)
- Discussed the associated conditions, when I mentioned fibular hemimelia, I was asked to assess the patient's fibula. I mentioned I could see five toes and could feel reasonable size and site of the fibular head and lateral malleolus, but it was difficult to assess the shaft, but x-ray may be more accurate to confirm this
- Classification and treatment: We discussed the Aiken classification and the bell rang before starting discussing treatment

Toe deformity

Various toe deformities are common in clinics and exams (Figure 14.13 and Table 14.6). Children with hallux valgus, hallux varus, mallet and curly toes have been commonly featured in the FRCS exam. The usual presentation is cosmoses, although pain or pressure over deformed toes are not uncommon. Some children are asymptomatic, but parents are concerned they may get trouble in the future. Although less common in children, possible underlying causes such as trauma, neuromuscular problems, inflammatory arthritis and diabetes mellitus should be enquired about and noted.

Unless you are specifically asked to examine the feet, start with general examination (general signs, posture and gait)

Table 14.6 Definition of toe deformities

Deformity	MTPJ	PIPJ	DIPJ
Hammer toe	Dorsiflexed or neutral	Plantar flexed	Neutral, hyperextended,
Claw toe	Dorsiflexed	Plantar flexed	Plantar flexed
Mallet toe	Neutral	Neutral	Plantar flexed
Curly toe	Neutral or plantar flexed	Plantar flexed	Plantar flexed

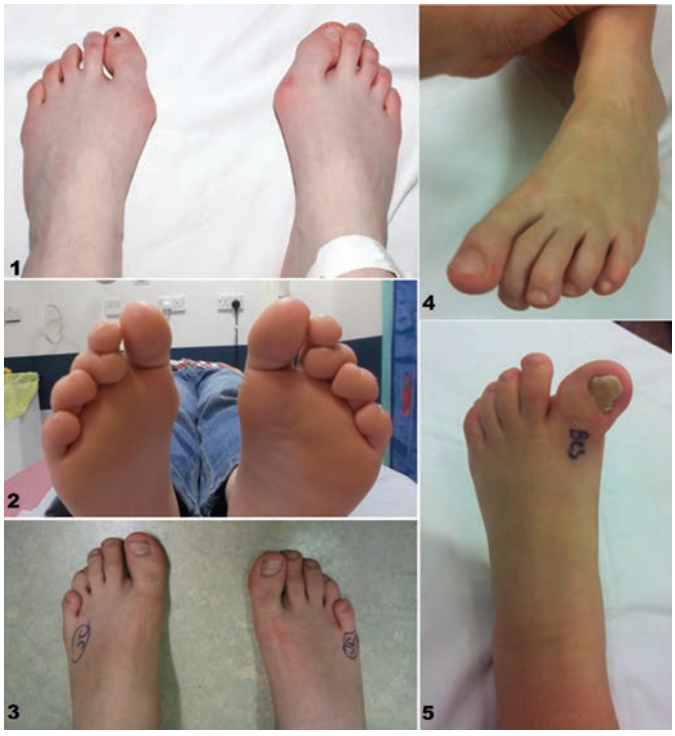

Figure 14.13 Toe deformities. 1: Hallux valgus, 2 and 4: Curly (under lapping) toes, 3: Overlapping toes and 5: Hallux varus

but do not waste a lot of time on them particularly if it was short case.

Look at the shape of the feet (pes cavus, pes planus), describe any deformity. Feel for tenderness and try to be precise about the site of tenderness. There may be two very close but clinically distinctive areas of tenderness.

Assess the flexibility of toe joint. Normally joints are flexible, note any contractures. Do not forget to examine the shoes.

Management

- Manage conservatively if at all possible; strapping, stretching, interdigital spacers, guards, footwear adjustment, orthotics, etc
- Several surgical options are available to deal with toes deformities ranged from tenotomies, capsular release and pinning; tendon transfer, Butler's procedure, various bony osteotomies and fusion. If you do well and time permits, you may get asked about your indications for your procedures of choice and to describe how you do them

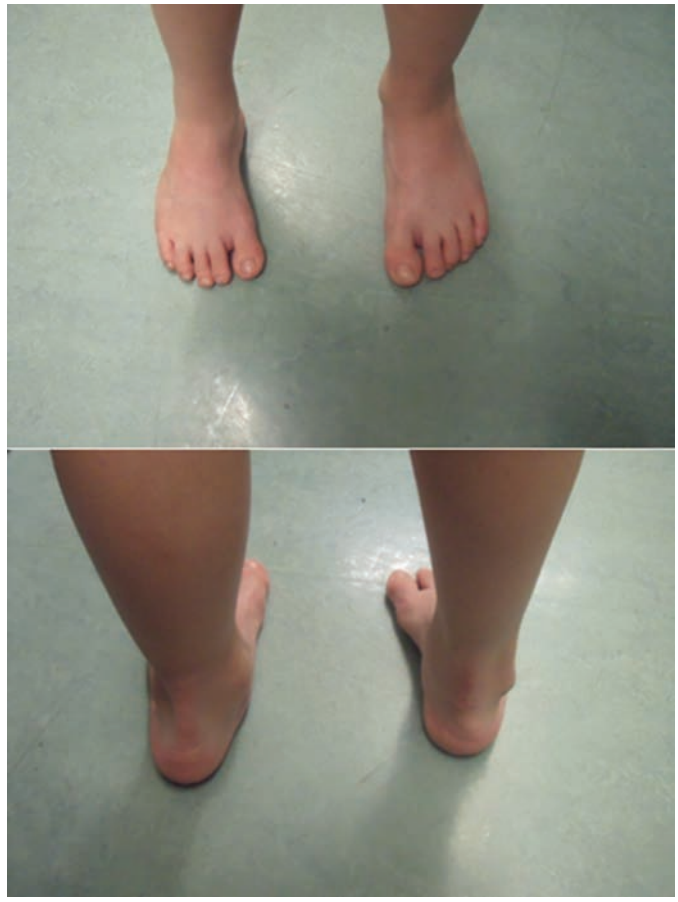

Figure 14.14 Residual (undertreated) clubfoot

Clubfoot

Treated clubfoot is a common short or intermediate exam case, particularly when there is suboptimal outcome such as under correction (Figure 14.14) or over correction. This should be easy viva even for candidates who did not do paediatric orthopaedic training. It is unlikely you would face a newborn child with clubfoot (although you may in the viva section).

Enquire about:

- Age at initial presentation (the earlier the better outcome)
- Treatment before relapse
  - Number of casts (normally around 4–6)
  - Tenotomise (90% needs tendoachillis tenotomy)
  - Length of brace wear. Boot and bar braces such as Denise Browne boots should be worn continuously for 3 months, after which they will be used at nap and night-time for 4 years. Compliance (or tolerance) has been shown to be the most significant risk factor for relapses)

  - Tendon transfer (about 20% needs tibialis anterior tendon transfer)

Start with the general examination unless you are clearly directed by the examiner, e.g. 'Examine this young boy's left foot.' Describe the deformity to the examiner and in particular look for residual metatarsus adductus, heel varus and equinus. You can use Pirani's score to structure your description. Look for secondary deformities (equinus can cause knee recurvatum). Are these deformities correctable?

Assess the range of motion of joints and tendons' strength particularly peroneal and tibialis anterior tendons. If the tibialis anterior tendon pulls the foot into dorsiflexion and supination, it may need to be transferred to the midfoot. Assess the shoes for size, modification and wear patterns.

Investigations

Weight-bearing AP, lateral and Saltzman's views of the foot.

Management

Serial casting can be repeated but the older the child the less likely it would be successful on its own. Surgical intervention often involves combinations of soft-tissue releases, bony procedures and tendon transfers tailored to that individual foot to achieve supple, pain-free plantigrade foot.

- Medial release (almost every medial structure can be released or lengthened but avoid damaging the deltoid ligament)
- Posterior release (tendoachilles and ankle posterior capsule)
- Tendon transfer such as whole or split tibialis anterior tendon transfer
- Bony procedures to correct alignment: Lateral calcaneum slide to correct varus, closing wedge cuboid osteotomy to swing the forefoot around the talo-navicular joint and correct forefoot adduction. Dorsal closing wedge of the first metatarsal to elevate the first ray
- Rarely arthrodesis is needed for severe and uncorrectable deformity.

Fortunately, over-corrected clubfoot has become rare since Ponseti's treatment was introduced. It was common after surgical treatment. The heel is usually in valgus and forefoot is abducted. These require medial calcaneum slide to correct valgus, opening wedge Cuboid osteotomy or calcaneum lengthening to swing the forefoot around the talo-navicular joint and correct forefoot abduction and dorsal openning wedge of the first metatarsal to create the medial longitudinal arch (basically the opposite of what you do for under-corrected clubfoot).

Bony exostosis (osteochondromas)

The most common bone tumours in children may be solitary or multiple. They usually rise from tubular bones metaphysis due to aberrant cortical overgrowth adjacent to the growth plate leading to eccentric bony growth usually away from the joint. They usually continue to grow until the growth plate closes at puberty. There are four common variants of bony exostosis that you may encounter in the exam:

1. ***Solitary osteochondromas***
   They usually present as a painless, slow-growing swelling in children or adolescents, mostly around the knee. They may cause mechanical symptoms depending on the location. There may be a history of trauma, but this is often coincidental. The patient is otherwise healthy.
2. ***Hereditary multiple exostoses (Diaphyseal aclasia)***
   An autosomal dominant (ask for family history when you suspect the diagnosis) condition. Patient is usually short with multiple bony exostosis and asymmetrical growth at the knees or/and ankles. There may be a leg length discrepancy. There may be scars from previous surgeries.
3. ***Multiple epiphyseal dysplasia***
   Mostly autosomal dominant condition characterized by the presence of epiphyseal (periarticular) chondromas of the knees and ankles. Patients with this condition usually present in late childhood. The spine is usually normal. Recessive multiple epiphyseal dysplasia is distinguished from the dominant type by malformations of the hands, feet, and knees and scoliosis.
4. ***Dysplasia epiphysealis hemimelica (Trevor's disease)***
   It is an epiphyseal dysplasia, thus, involving the joint. The lesions are usually restricted to one side of the body, either left or right; hence, the name hemimelica. It usually occurs in infants or young children. The medial side is affected twice as often as the lateral side.

The approach to patients with solitary or multiple bony exostoses include:

- Recognition of the condition and optimum description
  - The nature of swelling (Bony hard, pedunculated or sessile, attached to the underlying structure–bone, does not move with tendon or muscle, non-pulsatile, etc)
  - Is it single or multiple (actively search for other lesions)
  - Is it localized to one limb or side
  - Any family history
- Assess (or predict) the effect of the bony exostosis
  - Pressure affects on muscle, nerve-including spinal cord, vessels, etc
  - Interference with growth-shortening, mal-alignment, arthritis or joint pain (ask for long leg alignment views)
  - Cosmetic appearance
- Be aware of neoplastic potential (mostly to chondrosarcoma)
  - Solitary (1%) versus multiple (probably higher as there are many – controversial).
  - Growth of the swelling after maturity (or cartilage cap. >1 cm in adult)
  - Family history of malignant transformation
  - Neurological compression.

Investigation

1. X-ray: The radiographic appearances are usually characteristic
2. CT scan: Useful in the assessment of osteochondromas in the pelvis, shoulder, or spine
3. MRI is useful for assessing continuity of the parent bone with the cortical and medullary bone in an osteochondroma, cartilage cap size, impingement syndromes, and arterial and venous compromise
4. Angiogram is rarely needed; however, it may give useful information when assessing vascular occlusion, aneurysm and pseudoaneurysm formation

Treatment

1. Solitary osteochondromas; resects if symptomatic or very large (avoid in skeletally immature because severe growth deformity may result)
2. Multiple osteochondromas: It is risky and not feasible to remove multiple bony exostoses. However, excision of the symptomatic ones may be beneficial. Treatment of osteochondroma sequel is often needed
   a. LLD (as above)
   b. Angular deformity
      i. Timed hemiepiphyseal stapling
      ii. Corrective osteotomy
   c. Surgical decompression of neurovascular compression.

Examination corner

I was asked to assess a child with a swelling in the popliteal area. I think this was my lowest point – Struggled to feel it, examiner seemed to be getting a bit impatient by my ability to decide if it was a bony swelling. Finally gave osteochondroma as potential diagnosis and was relieved to be handed an x-ray showing the very same.

Neurofibromatosis

Neurofibromatosis (Nf) is the most common hereditary, hamartomatous condition affecting the peripheral and central nervous systems; hence, it is commonly features in the exam. Most candidates are able to pick the diagnosis of Nf because of distinctive features (Table 14.7).

Table 14.7 Diagnostic criteria for Nf-1 from the National Institute for Health

Signs	Requirement
Café-au-lait spots (Figure 14.15)	6 or more, >5 mm in children, >15 mm in adults
Cutaneous neurofibromas	2 or more
Plexiform neurofibroma	1 or more
Axillary or inguinal freckling (Figure 14.15)	Any
Optic glioma	1 or more
Lisch nodules	2 or more
Distinctive bone lesions	Sphenoid dysplasia, cortical thinning of long bone with or without pseudarthrosis
First-degree relative	Must have documented Nf-1 as per these criteria

If two or more of these signs are present then a diagnosis can confidently be made.

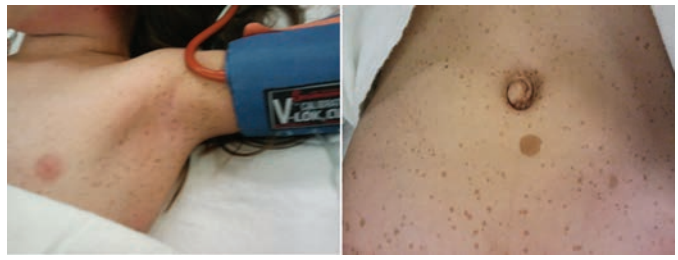

Figure 14.15 Neurofibromatosis. Axillary or inguinal freckling and café-au-lait spots

Elbow gunstock deformity (Figure 14.16)

A common spot diagnosis short case. The candidate is usually asked to assess a child with an elbow deformity. Most candidates do not have difficulty in reaching the diagnosis, but they do not provide optimum anatomical and functional assessment.

Inspect both sides for scars and palpable lumps (can be caused by hereditary multiple osteochondromatosis). You can ask the patient about history of trauma whilst examining. The deformity is clearer when the patient abducts both shoulders to 90° with thumbs up. Measure the carrying angle in the anatomical position comparing both sides. Demonstrate the range of movement and functional movement (can he reach his face, mouth and buttock). Check for signs (and symptoms) of elbow instability. Do not forget neurological assessment. Ask the patient and parent about the real concern and their expectations.

Figure 14.16 Elbow gunstock deformity

Management

- Bilateral varus or valgus elbow deformity in child with chromosomal anomalies usually does not require surgical intervention
- Unilateral, mild with no risk of progression (no growth plate damage), manage conservatively. The parent and child need to be reminded the operation would be associated with scarring and this can be ugly
- Unilateral, severe with no risk of progression, offer closed wedge osteotomy with displacement of distal fragment (to prevent medial or lateral translation associated with simple wedge osteotomy
- Unilateral, severe with risk of progression, delay treatment until skeletal maturity unless the deformity becomes severe or the joint becomes unstable.

Examination corner

Short case 2

EXAMINER: This 9-year-old boy was referred by his GP because his elbow did not look right, would you like to assess his elbow please?

CANDIDATE: He is a slim and healthy looking boy with obvious deformity of his left elbow. I think he has a gunstock deformity following a supracondylar fracture.

(There was a silence for a few seconds – the examiner probably wanted more information.)

EXAMINER: So what is a gunstock deformity?

CANDIDATE: It is a varus deformity of the elbow caused by growth arrest of the medial physis.*

EXAMINER: How would you manage this boy?

CANDIDATE: The deformity does not limit elbow function and most patients seek advice for cosmetic reasons. So if it is mild, I would reassure the child and parents and I offer them a review in 6 months to a year. If it is severe and the child is distressed by it, I offer them corrective surgery.

EXAMINER: What type of surgery?

CANDIDATE: Supracondylar osteotomy.

EXAMINER: What type of supraconylar osteotomy?

CANDIDATE: Lateral closing wedge osteotomy?

EXAMINER: Would you do epiphysiodesis at the same time to prevent recurrence?

CANDIDATE: He is 9 years old and still growing, so I would not do epiphysiodesis.

(Fail)

*Although, gunstock deformity can be caused by growth arrest, the commonest cause is usually mal-union. It is a combined varus and internal rotation of the distal fragment. Few candidates were asked to demonstrate the internal rotation component of the gunstock deformity (Yamomoto's test). The candidate should have assessed the deformity further and he should not have jumped into the diagnosis.

Achondroplasia

If you are examined on a patient with achondroplasia, it is time to score high. Clinical features are characteristics and potential orthopaedic problems are well recognised. Patients have a normal IQ and areusually very cooperative. The features include:

- Head and face
  - Large head with frontal bossing
  - Midface hypoplasia
  - Dental malocclusion and crowding
- Skeletal features
  - Disproportionate short stature: Normal trunk length with rhizomelic shortening of the proximal limbs with redundant skin folds
  - Brachydactyly and trident hand configuration
  - Lumbar lordosis
  - Hyperextensibility of most joints, especially the knees
  - Limited elbow extension and rotation
  - Bowed legs
- Radiographic findings
  - Small skull base
  - Progressive interpedicular narrowing in the lumbar spine region
  - Short pedicles which can cause spinal stenosis
  - Short femoral neck and metaphyseal flaring with inverted V shape distal physis
  - Small sacrosciatic notch, flat-roofed acetabulum

Two sets of orthopaedic problems that are associated with achondroplasia:

1. Spine

   Craniocervical junction abnormalities with subsequent cord compression may cause death in infancy. High risk with contact sport, car accident and intubation in general anesthetics

   Lumbar stenosis with neurological claudication in early adulthood. This responds to decompression

   Kyphosis
2. Limb lengthening

   Very controversial particularly for if it is for cosmetic reasons. It may be justified in very short patients who for example cannot drive nor do their jobs because of very short limbs

Growth hormone treatment is still being evaluated.

Tarsal coalition (rigid flat feet) (Figure 14.17)

This condition has been featured in the clinical exam frequently as a short case, but also a common viva question. The classical scenario is a teenager (10–14 years old) with

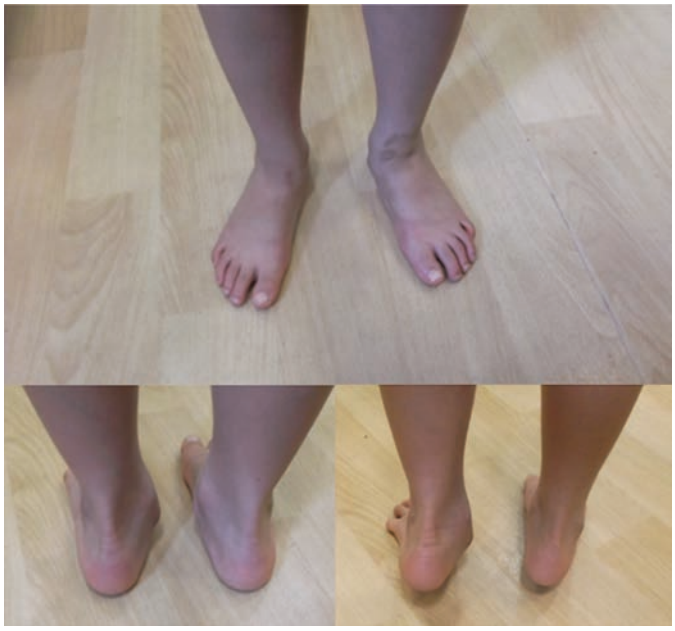

Figure 14.17 Tarsal coalition. Left rigid flat due to tarsal coalition. Note the left heel remains in valgus on tiptoeing

recurrent ankle sprains or fractures or both and the examination revealed rigid flat foot (unmissable!). Another scenario is to assess this child who was referred by his GP because he has flat feet?

Ask the patient to stand up with the feet slightly apart and observe the posture. The heel is usually in valgus and the forefoot is abducted (if adducted, think of skew foot). Inspect the bottom of the feet; you should be able to pass two fingers under the medial arch. If the planter aspect of the foot is convex, consider vertical talus.

Observe the patient walking. Observe him standing on toes while supported to a wall. In flexible (physiological) flat feet, the heels normally move to varus and the medial arch becomes more prominent. In tarsal coalition, the heel remains in valgus (well, not always true) and the arch does not reconstitute. Ask the patient to sit on the couch dangling his feet down. While doing so, ask permission to look at his shoes for wear pattern. If the shoes are new and there is no wear pattern, try to score extra marks by asking how long s/he has had the shoes for. There is more wear over the medial side of the heel.

Ask the patient where the pain is (if there is any). Calcaneonavicular coalition usually causes lateral side pain while talocalcaneal coalition causes medial side pain. Assess for tendoachillis tightness as this may cause stiff flat feet. Then assess the ROM of the ankle and subtalar joint. There is significant stiffness in the subtalar joint movement. It can be difficult to elicit as these patients have compensatory ball and socket ankle joint (by remodeling) and this can be deceptive. Try to use the thumb and index finger to stabilise or at least to check for talus body movement when assessing subtalar joint movement.

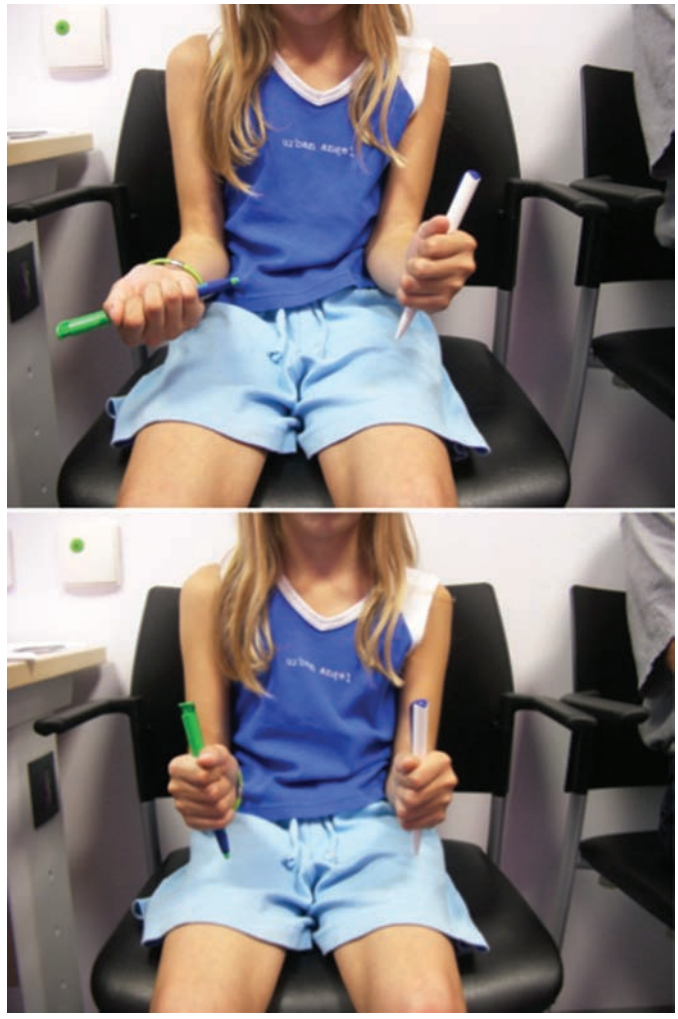

Figure 14.18 Left radioulnar synostosis

Radioulnar synostosis (Figure 14.18)

An extremely common short case. Can be congenital or acquired; partial or complete; fibrous or bony. Congenital bilateral in 60% of cases.

Traumatic radioulnar synostosis is more common and can happen in adult as well as children. It has been classified by Vince and Miller[2] into three types:

Type 1: Distal, located in the distal intra-articular portion of the forearm

Type 2: Diaphyseal, located in the middle and non-articular distal one-third of the forearm, most common

Type 3: Located in the proximal third of the forearm

Predisposing factors for traumatic radial ulnar synostosis:

- Badly displaced and comminuted fractures
- Both fractures at the same level
- Crushing injuries of the forearm
- Open fractures
- Single-incision exposure of both bones
- Fractures with concomitant head injury
- Delayed surgical fixation

Symptoms:

- Difficulty turning doorknobs
- Difficulty buttoning shirts
- Difficulty using eating utensils or writing devices
- Difficulty using or grasping small objects

The traumatic radioulnar synostosis will be covered in more details in the upper limb section.

Congenital radioulnar synostosis may present:

- In isolation (by itself)
- In association with other skeletal abnormalities (about one-third of the time)
- In association with problems of the heart, kidneys, nervous system or gastrointestinal system
- In association with certain genetic syndromes, such as Holt–Oram syndrome (also called hand–heart syndrome) and fetal alcohol syndrome

It has been classified into four types by Cleary and Omer[3]

Type 1: Fused clinically but not radiologically, small but normally developed radial head

Type 2: Similar but with clear bony synostosis

Type 3: Hypoplastic, posteriorly dislocated radial head

Type 4: Hyperplastic, anteriorly dislocated radial head

Th parents usually notice that the child holds his upper limb in funny angles and is not able to rotate his hand. There is usually no pain. Sometime, a trauma brings attention to the problem when a child falls on elbow and was found unable to rotate the forearm and the x-ray confirmed the abnormality.

Management

With the congenital variety there is usually little functional deficit. The forearm is in fixed pronation (30°), neutral or in slight supination. This is generally less disabling than fixed supination. Children may compensate with increased rotatory movements at the wrist, carpus and shoulder. Patients with unilateral synostosis usually do not require surgery unless there is marked fixed pronation.

There is no universal agreement on the best treatment. The current opinions are to rotate the forearm to the optimal position. The optimal position is controversial as well.

In unilateral synostosis, forearm position ranges from 15° of pronation to 35° of supination. Patients with bilateral synostosis may request surgical correction with the dominant limb fixed in 30–45° of pronation whilst the other side is fixed in 10–30° of supination. Other authors suggest the dominant hand is left in 20° of pronation and non-dominant hand in neutral.

Surgery

Rotational osteotomy refers to the realignment of the forearm by distal osteotomy with slight shortening to reduce the tension on soft-tissue structures. In adults both bones are realigned and fixed with compression plating. In children realignment of the radius is the preferred option.

In children osteotomy through the fusion mass with K-wire or Steimann pin fixation has been described but is generally not recommended.

When the deformity is congenital the soft tissues have been contracted from in-utero and the neurovascular structures are, therefore, sensitive to large rotational corrections. Complications of surgery include nerve palsies, vascular compromise, compartment syndrome and mal-union.

Resection of the bony bridge with interposition of fascia lata, plus resection of the radial head and division of the interosseous membrane have been described in the textbooks but they are technically demanding, results are often unpredictable and are generally not recommended although this may change in the future.

Examination corner

Short case 1

A candidate in her first short case was asked to examine the left forearm of a middle-aged male patient. She commented on several well-healed surgical scars present over both the volar and dorsal surfaces of the wrist, which she was told to ignore and to just concentrate on the forearm. There was muscle wasting present over the flexor muscles of the forearm.

The candidate was a little confused as what to do next and was told to examine forearm rotation. The patient demonstrated restricted forearm pronation. Asked what the diagnosis was she mentioned radioulnar synostosis. She was then asked the likely cause, to which she replied, 'trauma'. The candidate was then asked to check the patient's forearm rotation on the right side, which was similarly restricted.

The examiner then asked whether she still felt trauma was the cause and would it not be more likely to be congenital. In retrospect the candidate felt that being asked to examine the forearm had unnerved her and she would have made a better account of the condition if she had been asked to examine the patient's elbow.

'Would you care to examine this gentleman's forearm?'

Short case 2

A candidate was asked to examine the elbow of a young girl aged about 5. The lack of forearm rotation was immediately spotted when the patient was asked 'show me your arms'. A slight loss of full extension was also noted after which the candidate was asked what the probable diagnosis was. The candidate was then shown radiographs, which confirmed a proximal radioulnar synostosis. There followed a brief discussion about management options.

(Pass)

Madelung's deformity (Figure 14.19)

More common in exam than in real life. It can be a spot diagnosis when the deformity is moderate or severe, but it can be very subtle and needs a high level of suspicion. The clues are

Figure 14.19 Madelung's deformity

that the patient is usually female, with bilateral prominent ulnar styloid and restricted supination, dorsiflexion and radial deviation. Flexion and pronation are usually normal.

There are different ways how the examiners may pose the initial question; commonly they are guided by the patient's initial symptoms or current problem. The prominence of the distal end of the ulna is what draws the attention of most patients initially; later pain and loss of function may become an issue. Do not rush to give a diagnosis. Assess the patient, the limb and function fully first, then provide a diagnosis, ideally with a list of differential diagnosis. Usually a patient is a young female. Ask her to expose her upper limbs to the elbows paying attention on how easy or difficult she finds this and comment on this. Describe what you see:

> 'There is a prominence of ulnar head toward the dorsum with the whole wrist deviated ulnar and volar ward. I cannot see any scar, swelling or deformity over the back of hand, wrist or forearm. In particular I cannot see ulnar (or radial) drift of fingers or thumb deformity. "May I ask you to turn your hand over please?" She has difficulty in supination and trying to use her shoulder to compensate for lack of supination, I will assess this fully when I come to ROM assessment, . . . etc.'

Ask the patient about any tender areas and palpate the hand for tenderness. If you are not sure about the diagnosis at this stage . . . start from the fingers and proceed proximally, keep looking at the patient's face to sense any pain or discomfort. There is usually tenderness over the distal radioulnar joint. Gently check how mobile it is.

Assess movement: 'Can you straighten your elbow fully please? And can you bend them fully please?' If there is any restriction in active ROM, try to improve it passively. 'Can you tuck your elbows to your waist and stick your thumbs up?' 'Now, turn your hand up?' 'Let's measure this.' 'Active supination is 20°.' 'Let me see whether I can increase it further by turning your wrist gently.' 'No I cannot without causing you pain' . . . etc.'

Assess the functional profile such as picking up objects, washing face, writing, doing buttons up, and putting trousers on, . . . etc.

There are two rare variants you may need to be aware of them. Reverse Madelung's deformity in which the physeal growth arrest is ulnar and dorsal (rather than ulnar and volar); hence, the radial articular surface tilt is dorsal and the ulnar head prominence is volar. The second is the Chevron carpus in which the physeal growth arrest is ulnar and central. This can be quite difficult to diagnose as there is little or no deformity of the wrist and no instability of the distal radioulnar joint.

Madelung's deformity could be:

1. Post-traumatic (growth disturbance of distal radial ulnar volar physis secondary to trauma-usually repetitive)
2. Dysplastic (multiple hereditary osteochondromatosis, Ollier's disease, achondroplasia, multiple epiphysial dysplasias, and mucopolysaccharidoses
3. Chromosomal or genetic (Turner's syndrome)
4. Idiopathic or primary

Management

Conservative

In mature patients with mild deformity, symptomatic treatment using pain killers, splints and job modification.

Surgery

There are four important considerations:

1. Patient age and the growth remaining in the distal radius
2. Severity of the deformity
3. Severity of the symptoms
4. Clinical and radiographic findings

Operative treatments are divided into:

- Operation to prevent or correct primary deformity (Vickers' physiolysis, osteotomy, epiphysiodesis, radius lengthening)
- Operation to decrease pain and improve range of movement (Sauve–Kapandji operation) – Some authors advocate a Darrach procedure (excision of the distal ulnar); however, there is a risk of carpal instability
- Both (wrist fusion, osteotomy).

Arthrogryposis

Literally means curved joints. Arthrogryposis is a descriptive term and not an exact diagnosis, because there at least 150 possible underlying diagnosis. Hall et al. 1985 considered three main groups:

1. Classical arthrogryposis multiplex congenita; in which the limbs are involved and the muscles are absent or deficient

2. Arthrogryposis associated with neurogenic (brain, spinal cord or peripheral nerve) or myopathic (congenital muscular dystrophy or myopathy)
3. Arthrogryposis associated with other syndromes or anomalies such as diastrophic dysplasia or craniocarpotarsal dystrophy

Arthrogryposis multiplex congenital has a sporadic occurrence. Aetiology is still unknown with several theories advocated. Fetal akinesia (decreased fetal movement) seems to be a common pathway for these theories. Other recognisable patterns of arthrogryposis have an established heritability, the most commonly encountered are the distal arthrogryposes and Freeman–Sheldon syndrome.

Several candidates have been tested on patients with arthrogryposis. Recognition is not particularly difficult. There are classical features that you can rattle off quickly to the examiner:

1. Involved limbs are tubular in shape, with thin and subcutaneous tissue and absent skin creases, particularly over joints
2. Deformities are usually symmetric, and severity increases distally, with the hands and feet typically the most deformed *(I call them the Kangaroo upper limbs).*
3. The patient may have joint dislocation, especially the hips and, occasionally, the knees.
4. The trunk is rarely affected (occasionally scoliosis).
5. Atrophy may be present, and muscles or muscle groups may be absent.
6. Sensation is usually intact
7. Patients have normal IQ in most cases.

A multidisciplinary team approach (paediatrician, orthopaedic, geneticist, neurologist, psycholist, physiotherapist and occupational therapist) is essential for successful outcome in these patients. The basis of orthopaedic assessment and management is to optimize function and to keep these children as independent as possible (and they do).

In the exam situation, you are more likely to be directed to a particular problem to assess. 'This child was born with bilateral clubfeet. He had two courses of Ponseti serial casting but it has not been successful. Would you like to examine him and tell us what you think?' Or 'This 5-year-old child is known to have arthrogryposis. He had bilateral dislocated hip which was reduced when he was 1 years old. He is here for routine follow up appointment. We want you to assess his hips please'.

I simulate arthrogryposis assessment to that of patient with severe rheumatoid arthritis. Although, we are interested in the state and function of individual joint, but the function of the whole limb is far more important.

When you are asked to assess a patient with arthrogryposis in exam, do not panic. Listen to the examiner carefully (often the answer is in the question). Introduce yourself, and then undress them reasonably.

Inspect the upper limb comparing both sides at the same time. Comment briefly on the general signs of arthrogryposis but do not dwell on them. Describe the posture of the limbs. The shoulder is usually internally rotated and adducted, elbow in extension, wrist and finger in flexion. Ask the patient to show you what s/he can do, 'Can you reach your face (for washing and feeding)?' 'Can you reach your buttock (for toileting)?' 'Can you do your buttons?' 'Can you pick up this coin?' 'Can you hold a pen?' 'And can you write?' 'If you imagine my hand as door knob, can you turn around?' 'Can you use computer keyboard?' . . . etc.

If s/he cannot do certain task, assess why and what stop the patient doing so. Is it the shoulder's internal rotation, is it the elbow stiffness or is it the wrist? Do not hesitate to say, I would refer the patient for full OT assessment (this is what we do in clinic). If a problem is identified, suggest a solution or solutions and involve the patient and carer with the solution you suggest ('So if we improve the elbow bending, do you think that would help?'). Start with the safest and most successful solution and proceed to the others. For example, if the elbow extension deformity was the problem, consider physiotherapy before operation, manipulation before tendon transfer and so forth.

Pay attention to the details, particularly signs of previous surgical interventions. It is not a good idea to offer them a surgery that they had already had it.

Deformities of the wrist and elbow and the foot and knee can be addressed in the first weeks of life with a combination of passive stretches and resting splints. In the lower limb this is effectively a modification of the Ponseti regime, with variations depending on the severity of the deformity and the position of the knee joint.

Hip dislocations are common and usually have occurred in utero well before birth, these joints are rarely amenable to closed treatment with a brace and are, therefore, treated either by open reduction in the first year of life, or are best left dislocated.

Orthopaedic interventions can be useful in most joints and regions of the arthrogrypotic child (rarely the shoulder), but this should be carefully planned, staged and timed.

Aim to correct most major deformities within the first 12–18 months of life then concentrate on function.

In later life, surgical interventions are limited to maintaining mobility and optimizing function. Tendon transfer may bring motor power to a joint that has been optimised. The elbow joint is well suited for tendon transfers with good results in selected cases.

Klippel–Feil syndrome

Adult or child: Short webbed neck or no neck appearance with low hairline.

Spot diagnosis: Head on top of the shoulders with restricted range of movement. Full spinal and neurological examination are required.

Associated features:

- Sprengel's shoulder
- Torticollis

- Congenital scoliosis/kyphosis of the cervical spine (high risk of instability)
- Congenital heart disease – Echocardiogram is indicated
- Renal anomalies – Ultrasound is indicated

Feil classified the syndrome into three types:

- Type I – A massive fusion of the cervical spine
- Type II – The fusion of 1 or 2 vertebrae
- Type III – The presence of thoracic and lumbar spine anomalies in association with type I or type II Klippel–Feil syndrome

There is a high risk of instability, especially if the malformation limits mobility at one level; these include atlanto-occipital fusion with C2–C3 block vertebrae, abnormal atlanto-occipital junction with several distal block vertebrae; and a single open interspace between two block segments. Subluxation of the cervical spine can occur with minor injury. Warn patients against contact sport (diving, gymnastics and rugby) and review annually with flexion/extension plain radiographs. Arthrodesis of unstable segments may be required if excessive instability and neurological abnormalities are present.

Klippel–Feil syndrome is a classic spot photograph diagnosis in the paediatric orthopaedic oral examination but it rarely comes up in the clinical section presumably there is more important clinical scenario to cover in a limited amount of time available.

Sprengel's deformity of the shoulder (Figure 14.20)

A complex anomaly that is associated with malposition and dysplasia of the scapula. Although, it is commonly associated with the Klippel–Feil syndrome, it can be a feature of other syndromes such as Poland syndrome and VATER syndrome. Embryologically, the scapula is derived from the neck and normally descends to the thorax by the end of the third month of intrauterine life; any interruption in its descent can result in a hypoplastic, elevated scapula, known as the Sprengel's deformity.

The trapezius, rhomboid, or elevator scapulae muscle may be absent. The Sprengel's deformity usually presents with shoulder asymmetry and restriction of shoulder abduction. The scapula is elevated by 2–10 cm and its inferior pole is rotated medially.

Management

Conservative

Physiotherapy to maintain the range of movement and strength of shoulder girdle.

Surgery

Several surgical procedures have been recommended to improve the cosmetic appearance and the shoulder function when it is severely impaired. These include but not limited to Mears, Konig, Green and Woodward scapuloplasty. For optimum outcome, experts recommend surgery before 8 years.

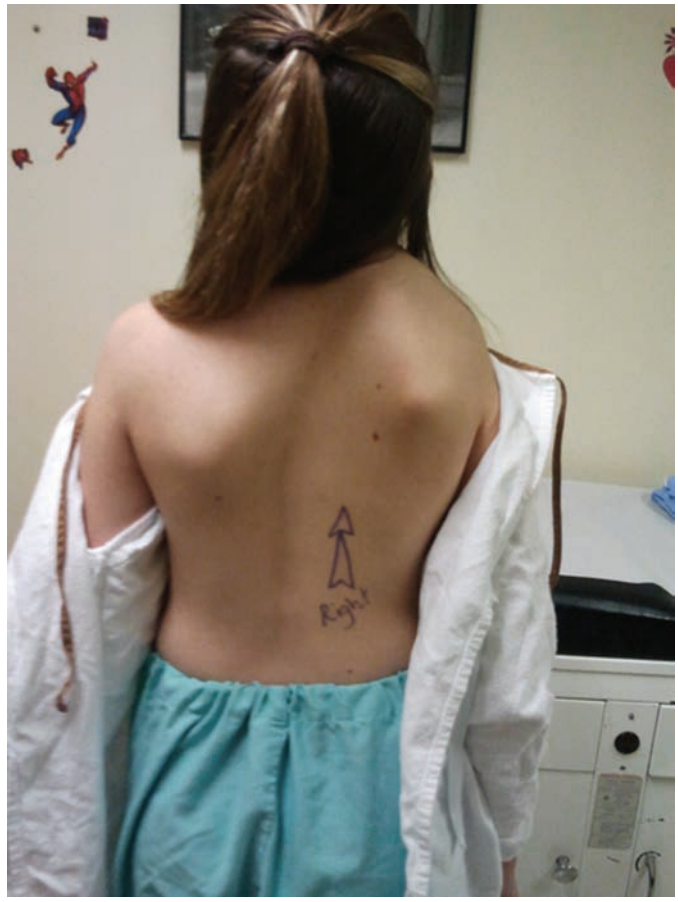

Figure 14.20 Sprengel's deformity

Congenital pseudoarthrosis of the clavicle

The patient will be a child and it will almost always be the right clavicle. If it occurs on the left side it is associated with dextrocardia. There will be a non-tender swelling/lump over the middle third of the clavicle and possibly a gap across the clavicle. The shoulder may hang lower than on the opposite normal side. The clavicle is also effectively shortened.

The pseudoarthrosis is caused by failure of fusion of the medial and lateral ossification centres. Possible aetiology is an abnormally high subclavian artery. Differential diagnosis includes post-traumatic pseudoarthrosis, neurofibromatosis and cleidocranial dysostosis of the clavicle (skull abnormality).

This is a painless condition and produces little functional abnormality. There are parental concerns about the unsightly lump. The opposite ends of the clavicle fragments are enlarged just lateral to the midpoint of the clavicle. The larger sternal fragment is pulled upwards by the sternocleidomastoid and lies slightly superior and in front of the shorter acromial fragment. The shoulder droops and is rotated forwards.

Management

Conservative

Observe; leave it alone, especially if the patient is asymptomatic, because of the risk of possible complications from surgery.

Surgery

Excision of the pseudoarthrosis, curettage of the bone ends and fixation with plate and screws. A bone graft (tri-cortical iliac crest) is sometimes required to reconstruct the length and shape of the clavicle. A recon plate is then contoured and fixed with screws. Surgery can be carried out by the age of about 4 years although opinion varies as to the appropriate age for surgery. Brachial plexus neurapraxia has been reported following resection and fixation of the pseudoarthrosis. With skeletal growth the lump and instability at the pseudoarthrosis site increase. The overlying skin becomes atrophic and the deformity can become cosmetically unsightly. The affected shoulder droops. The patient may complain of mild pain and weakness in the shoulder. Surgery is generally advised, 'My preferred option would be to fix it'.

Examination corner

Short case 1

Classic short case: spot diagnosis – the candidate should recognise the diagnosis immediately on inspection.

EXAMINER: Would you like to examine this young girl's shoulder and describe what you are doing as you go along?

She is a young girl about 5 years old and, therefore, it is vital to smile at the child to put her at ease no matter how stressed you may feel. Introduce yourself to both mother and child. Crouch down so you are at her eye level.

CANDIDATE: On inspection from the front there is an obvious swelling over the middle of the right clavicle. Is it painful? Can I touch it? On palpation the swelling is bony, hard, non-tender, not attached to skin, its surface is uneven, its edge is distinct and it is non-pulsatile. There is a suggestion of a small amount of painless mobility between the two ends. The swelling is probably bony in origin and very suggestive of pseudoarthrosis of the clavicle. Can you swing your arm outwards (demonstrated whilst talking)? She has a good range of shoulder abduction, which is not painful.

EXAMINER: Good, let us look at her x-rays.

CANDIDATE: This confirms a pseudoarthrosis of the right clavicle with smooth sclerotic enlarged deformed bone margins characteristic of the condition.

EXAMINER: How are you going to manage it?

CANDIDATE: My preferred option would be to fix it. It's an unsightly deformity that tends to worsen when the child grows. It can also be painful and the shoulder tends to droop down.

EXAMINER: Is there any place for conservative treatment?

CANDIDATE: That's certainly an option if it's painless and causing no undue problems and the parents are not concerned about its appearance.

EXAMINER: Why are they always right sided?

CANDIDATE: That is because it is thought that the subclavian artery interferes with fusion of the medial and lateral ossification centres. They can occur on the left side with dextrocardia.

Congenital radial head dislocation

A classic short case. Possibly a radiographic spot diagnosis in the paediatric oral with discussion about its differentiation from a traumatic or paralytic dislocation. May occur as an isolated entity or associated with several generalized skeletal malformation syndromes.

Developmental dislocations of the radial head may occur with diaphyseal aclasia or hereditary multiple osteochondromatosis as a result of retarded ulnar growth. In paralytic disorders the muscle imbalance between the supinators and pronators can result in radial head dislocation.

Be familiar with the various management options available and their indications.

Memorandum

'On examination of the left/right elbow there is a suggestion of a mass present posteriorly around the lateral epicondyle. The attitude of the left/right elbow suggests there is a loss of full supination.'

'I would like to confirm this clinically. There is both a restriction of full supination and loss of full extension of the elbow. Flexion and pronation appear full. The swelling itself is non-tender and bony hard.'

Background

The radial head may be dislocated anteriorly, laterally or posteriorly. Posterior dislocations are nearly always congenital. Congenital anterior dislocations are nearly always associated with other congenital conditions. The primary defect is thought to be capitellum dysplasia. The anteriorly dislocated radial head is rounded, often with a deficient capitellum and a long radius. The posterior border of the ulna is concave instead of convex. The posteriorly dislocated radial head is thin and elongated, and the posterior border of the ulna is markedly convex.

Relocation is usually not successful. The condition predisposes to osteoarthritis of the ulnohumoral joint in later life. It can be bilateral.

Clinical features

A completely dislocated radial head is usually pain free with little loss of function. Pain may develop in adolescence or adult life if the radial head is subluxed. An anterior dislocation will usually have restricted flexion and supination due to a mechanical block. Posterior dislocation usually causes limitation of extension and rotation of the forearm. Lateral dislocation of the radial head tends to cause cubitus valgus. The radial head can usually be easily palpated and may produce an ugly prominence on the lateral side of the elbow. Associated conditions include arthrogryposis, Ehlers–Danlos syndrome, diaphyseal aclasis and nail–patella syndrome.

Management

Conservative

- If there are no symptoms and if the limb function is satisfactory then leave it alone

- In childhood, it is best to leave alone; surgery should preferably not be carried out because it may cause longitudinal growth disturbances
- In adulthood again preferably to manage conservatively especially if detected co-incidentally

Surgery

- Open reduction of the radial head with shortening of the radius. This is difficult and recurrent dislocation is common. The flat or convex radial head and flat capitellum make relocation very difficult
- If necessary, excision of the radial head in the adult can be done to relieve pain and improve appearance; however, the ROM is not significantly altered. This is not recommended in children as proximal migration of the radius and inferior radioulnar subluxation can develop as normal growth of the ulna continues
- In some conditions a short ulna predisposes to radial head dislocation, e.g. multiple osteochondromatosis. The radial head will fall back into position if the ulna is lengthened to restore the normal relative lengths of the radius and ulna.

Examination corner

Short case 1

Young boy about 9 years old with mother

EXAMINER: Would you like to examine this young girl's elbow for me?

CANDIDATE: *(I immediately thought about congenital radial head dislocation or radioulnar synostosis. I began with a simple description of how the elbow appeared)*

On general inspection the elbow is held in about 20° of flexion and almost full pronation. There is an obvious swelling on the posterolateral aspect of the elbow

The ulna appears more prominent at the wrist. I would like to go on and palpate the swelling. On palpation the swelling is non-tender, 2 cm by 2 cm, bony hard consistency non-pulsatile, non-mobile, not attached to skin.

Examining elbow movements there is normal flexion but loss of the final 20° of extension. Pronation and supination are both mildly restricted with loss of about the final 20° of each movement.

EXAMINER: So what do you think the diagnosis is?

CANDIDATE: Congenital dislocated radial head.

EXAMINER: What are the other causes of a dislocated radial head?

CANDIDATE: Trauma, diaphyseal aclasia, radioulnar synostosis

EXAMINER: How do you treat it?

CANDIDATE: If it is not painful and causing minimal functional difficulties I would treat the condition conservatively, advice with reassurance. Surgery can be difficult.

Trigger thumb (Figure 14.21)

Rarely shown in the clinical exam because most patients are operated on by the age of 4. It is usually asymptomatic and the patient is brought in by parents after noticing a fixed posture of the thumb's IPJ.

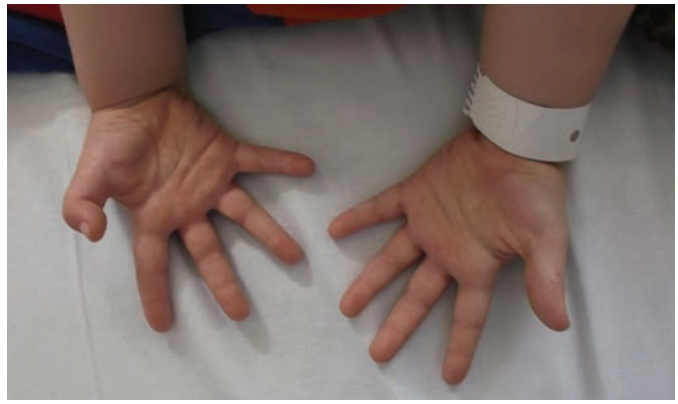

Figure 14.21 Trigger thumb

Examination corner

Short case 1

A candidate was asked to examine the thumb of a 3-year-old child who was sitting in his mum's lap. He hoped it was a trigger thumb, so he did not jump in to examine the child's hand but he started talking to the parent (and the child) and observing the deformity. He commented that the child looked healthy and active; both thumbs IPJ seemed to be flexed. He could not see any other abnormal features such as scars or congenital abnormalities.

CANDIDATE: My initial thought is that this child has trigger thumbs and I would like to confirm my initial impression.

EXAMINER: How would you do this?

CANDIDATE: In trigger thumb, it is usually bilateral, the IPJ can be flexed further but the extension is limited and there is usually a mobile, palpable lump at the mouth of A1 poly which moves as I flex and extend the IPJ.

EXAMINER: How would you treat him?

CANDIDATE: My preferred option in this age group is surgical release of the A1 poly, but in infants, I would advise on regular gentle stretching as this can be successful. Failing this, surgery would be advocated.

EXAMINER: This child is 3 years old now, would you wait till he is older and stronger?

CANDIDATE: I usually recommend surgery when the child is between 2 and 4 years of age. Before 2, there is a higher risk from anesthetic and surgery; after 4 years, the child may develop permanent structural changes leading to fixed flexion deformity.

(Pass)

Other paediatric upper limb conditions

Clinodactyly, camptodactyly, Kirner's deformity, thumb duplication, radial club and ulnar club hands have been featured in the clinical exam as short cases of spot diagnosis and further management. These have been covered in the hand core topic.

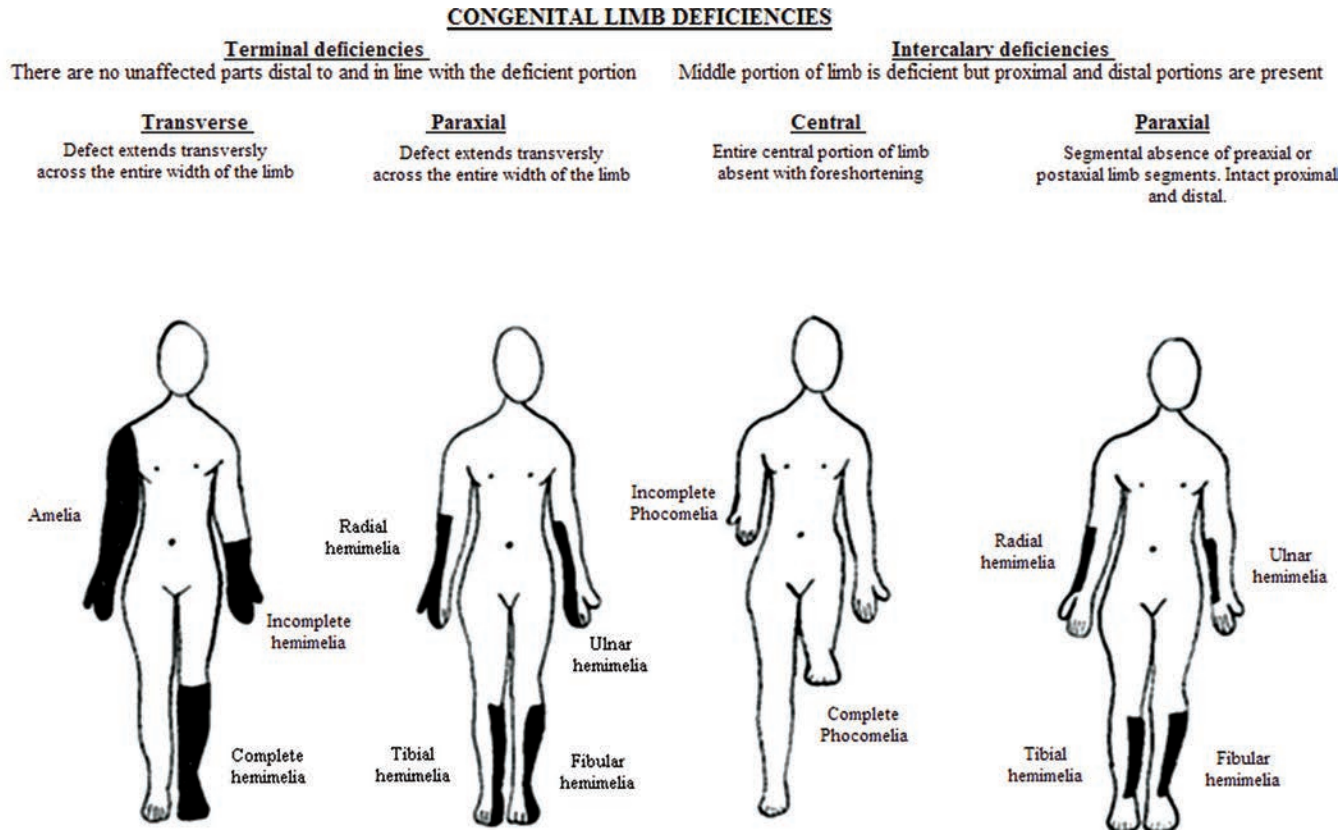

Figure 14.22 Congenital limb deficiencies

Congenital absence of limbs or part of a limb (Figure 14.22)

You may encounter a patient with congenital anomalies of the extremity as a short case and the examiners want to see a reasonable understanding of the classification and potential treatment options.

Three important issues are to be explored in these types of scenarios: The functional limitation; the cosmetic appearance; and the psychological impact on child and parent.

International Federation of the Hand Classification[4]:

1. Failure of formation
2. Failure of differentiation
3. Duplication: May apply to whole limb, mirror hand, polydactyly
4. Overgrowth: Includes conditions such as hemi-hypertrophy and macrodactyly
5. Congenital constriction band syndrome
6. Miscellaneous
7. Generalized skeletal abnormalities such as arthrogryposis, congenital dislocation of the radial head and Madelung's deformity

Failure of formation

1. Transverse arrest – It can be at any level, shoulder to phalanx (congenital amputation)
2. Longitudinal arrest
   - Preaxial – varying degrees of hypoplasia of the thumb or radius
   - Central – divided into typical and atypical types of cleft hand
   - Postaxial – varying degrees of ulnar hypoplasia to hypothenar hypoplasia
   - Intercalated longitudinal arrest – various types of phocomelia

Failure of differentiation

1. Soft tissue – syndactyly, trigger thumb, Poland syndrome, camptodactyly
2. Skeletal – various synostoses and carpal coalitions
3. Tumorous conditions – include all vascular and neurological malformities

Child with a popliteal cyst (Figure 14.23)

EXAMINER: This 7-year-old boy was brought by his parent as they noticed a swelling at the back of his knee. Would you like to examine him and tell us what you think?

CANDIDATE: *(Introduces himself and asks permission to examine the child knees)* . . . The child looks healthy and active and I could see he is not in discomfort when he moves his knee. Is this the swelling that's concerned you?

MOTHER : Yes.

CANDIDATE: Is it painful? Or has it caused any symptoms?

EXAMINER: *(Interrupting)* We just want you to examine the swelling and describe your findings as you go along.

CANDIDATE: There is ill-defined swelling at the posteromedial aspect of the knee, it measures about 2 × 3 cm. *'Can I feel it please?'* It is not particularly warm and has ill-defined margin. It is not tender even when I press hard on it.

The knee is not swollen or hot and I can bend it fully without any discomfort. I would like to check for transillumination, but I am sorry I do not have a torch.

EXAMINER: Here is a torch.

Candidate demonstrated positive transillumination of the cyst. He offered to examine the groin for any swelling and the foot for any unexpected pathology.

EXAMINER: What would you tell this boy and his parent?

CANDIDATE: This is likely to be a popliteal cyst and it is a benign condition that rarely causes symptoms and usually disappears as the child gets older.

EXAMINER: The parent wanted to know how quickly the swelling will disappear.

CANDIDATE: I am not sure but it could take several years. As long as it does not cause symptoms or gradually gets bigger, I would not worry about it.

EXAMINER: How would you investigate?

CANDIDATE: The diagnosis is clinical and further tests are just to rule out unexpected pathology, so if the swelling is associated with pain, increasing size, I would consider MRI scan or ultrasound.

(Pass)

In straightforward cases like the above two, most candidate pass easily, but we advise to aim more than just a pass by quoting supporting evidence. For example, the candidate would have scored higher if he mentioned 'This is likely to be a popliteal cyst and it is a benign condition that rarely causes symptoms and usually disappears as the child gets older. Dinham in his classical paper[5] reviewed the natural history of over 100 popliteal cysts and found most popliteal cyst disappeared spontaneously within 5 years.

Nail–patella syndrome

Uncommon autosomal dominant disease usually presented in late childhood or early adulthood with knee pain or recurrent dislocation of the patella. Four features are very characteristic for this syndrome: Fingernail dysplasia, absent or hypoplastic patellae, the presence of conical iliac horns and hypoplasia of radial head. Forty per cent of patient developed immune related nephropathy which is the most serious complication. Most patients do not need any surgical intervention; however, patients with recurrent or permanent patellar dislocation should be treated with surgical stabilisation.

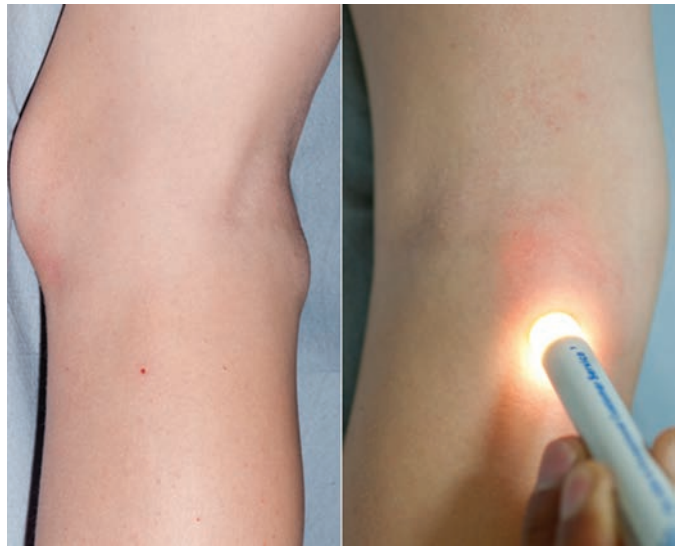

Figure 14.23 Popliteal cyst

Examination corner

Short case 1

EXAMINER: Would you like to examine this young lady who complains of gradually increasing pain in her knees?

CANDIDATE: *'Can I ask you to stand up please?'* There is bilateral knee swellings and puffiness; I cannot see any scar or deformity. *'Can I see you walking please?'* She walks normally and the knees bend appropriately with walking. *'Can I ask you to lie on the couch please?'* I cannot feel any hotness. She seems to have tenderness around her knee cap on both sides. *'Can you straighten your knee out please?' 'And can you bend it fully?'* Now, I am checking the knee stability. I think this girl has patellofemoral chondromalacia and early arthritis.

EXAMINER: Why do you think so?

CANDIDATE: She is young, female with bilateral anterior knee pain and tenderness. This is a classical presentation.

EXAMINER: Then would you like to examine her elbows as well?

CANDIDATE: *'Can I ask you to stand up please?' 'And if you turn your hands forward please.'* There are no visible scars, or swelling. *'Can you straighten your elbow any further please?'* OK, there is a loss of about 15° of full extension. *'Can you bend it fully please?'* They are almost symmetrical with a loss of 15°. *'Please, tuck your elbows to your waist with the thumbs pointing up.'* I can see she cannot supinate her hands fully. *'Can you turn them down please?'* Again, she has restricted pronation.

EXAMINER: Do you still think it is classical patellofemoral chondromalacia?

CANDIDATE: No, may be juvenile rheumatoid arthritis affecting her knees and her elbows.

EXAMINER: Do you want to examine her dad's elbows?

CANDIDATE: *(more puzzled) 'Can I ask you to straighten your elbows with hands facing forward?'* I cannot see any scar and the elbows lost at least 30° of full extension and I cannot straighten them any further. There is significant restriction of pronation and supination.

EXAMINER: Can you guess the underlying pathology?

(Pass)

In retrospect, the candidate feels that he had failed that particular short case but he did well in other stations and managed to obtain the pass mark. He'd never seen or read about the nail–patella syndrome, but the scenario was of classical nail–patella syndrome.

Pes cavus

Pes cavus is a deformity in which the longitudinal arch of the foot is high and does not reduce on weight-bearing. It is a spectrum including cavo-varus foot, Plantaris, calcaneus and calcaneo-cavus foot deformities. A child with pes cavus can be the subject of intermediate or short case. It is not surprising that many candidates do not like this topic. This is partly because there is a wide spectrum of clinical problems and presentations, which may need different types of treatments. Moreover, there are various underlying conditions in cavus foot and these conditions merit special attention and assessment. Here we try to provide a simple and logical approach to a patient with pes cavus. We strongly recommend reading the topic in more depth in one of the textbooks as well.

Rang's tripod theory depicts the foot as a balanced tripod (the calcaneum, the first and the fifth rays); with all three points resting on the ground (Figure 14.24). Muscle imbalance causes one or more of these structures to assume abnormal posture around their joints increasing the height of medial arch. For example, cavo-varus is usually caused by the calcaneum moved into varus (stronger tibialis posterior) pushing the foot into supination. To compensate and balance the tripod, the first ray has to flex more. This increases the height of the medial arch. The same could happen when there is an excessive planter flexion of the first ray (strong peroneus longus).

The pattern of muscle imbalance varies according to the underlying condition, hence, the type of pes cavus. In Charcot–Marie–Tooth disease (HSMN), for example, the tibialis anterior is weak or paralysed while peroneus longus is normal. The head of first metatarsal is depressed owing to unopposed action of peroneus longus. The peroneus brevis is weak while tibialis posterior is normal leading to varus hindfoot. Because the weakness of the tibialis anterior, patient tries to compensate by using toes extensors to provide enough dorsiflexion to clear the floor causing hyperextension at the MTPJs and subsequent clawing. It also tightens the planter fascia (windless mechanism) and the arch of the foot is accentuated further.

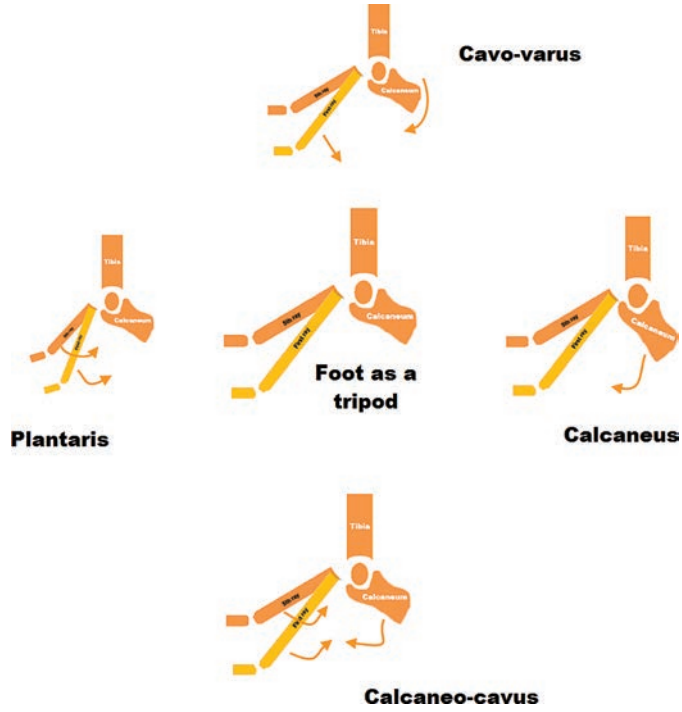

Figure 14.24 Tripod theory to explain pes cavus. Pes cavus is a spectrum of high arched foot deformities including cavo-varus foot, plantaris, calcaneus and calcaneo-cavus foot deformities

In spina bifida and poliomyelitis, there is a weakness of the triceps surae leading to calcaneus deformity due to unopposed action of ankle dorsiflexors and reciprocally plantarflexed forefoot.

Varus heel (subtalar inversion) locks the midtarsal joints causing a rigid foot. Excessive pressure may fall under the head of metatarsals leading to painful callosities.

When faced with a patient with pes cavus, the clinical picture is usually clear, but there are key questions to answer:

1. What actual problems does the patient have now?
2. What might happen in the future?
   (a) Is it progressive?
   (b) Risk of ulceration or infection
3. Is there an identifiable neurological cause?
4. What are the patient (and parent) expectations?

What is the problem?

Pes cavus can produce a wide spectrum of symptoms. It can cause infective ulceration threatening limb or life; on

the other hand, many patients have quite limited problems and little or no disability requiring no treatment. Common problems in cavus foot:

1. Toe deformity rubbing on shoes
2. painful calluses under the metatarsal heads caused by forefoot plantar flexion and fixed toe deformity
3. Lateral foot pain and painful calluses on the lateral foot border due to hindfoot varus
4. Walking difficulty due to foot deformity or foot drop
5. Foot wear problems due to deformity
6. Ankle instability due to hindfoot varus and peroneus brevis weakness
7. Worries about progression.

It is valuable to assess the impact of these individual problems on patients. How does it affect you? Does it interfere with your work? Sport? Walking, etc.? Children may not complain of pain, but they fatigue easily.

Is it progressive?

1. The length of history (When did you notice the deformity? Patient may be born with it. Is it getting worse? Is the other foot normal? Do you have problem with bowel or bladder? Any weakness in your hands or shoulder?)
2. Any previous history of ulceration or infection? How was it treated? Is it getting more frequent? Or more difficult to treat?

Parents or carer may provide valuable information.

Is there an identifiable neurological cause?

Are there any problems with your hands, back, vision, etc? Has anybody else in the family had a similar problem? Is there any family history of neurological diseases?

Causes of pes cavus

1. Congenital
   (a) Idiopathic
   (b) CTEV
   (c) Arthrogryposis
2. Acquired
   (a) Trauma
   (b) Neuromuscular
      (i) Muscular dystrophy
      (ii) HSMN
      (iii) Polio
      (iv) Spinal cord disorders (spina bifida, spinal dysraphism)
      (v) Friedrich's ataxia
      (vi) Cerebral palsy

What are the patient (and parent) expectations?

'How can I help you? What do you expect from your visit? Is it the pain or the ulcer that really bothers you?'

As in history, the clinical assessment has two objectives

1. Describe the deformity and the effect of the deformity
2. Search for any underlying condition. This includes full neurological assessment

Inspection (patient is standing – If able)

- Are the leg muscles wasted?– Quick glimpse on the hands
- Is there a high medial arch (can you pass two fingers underneath)? Is it bilateral? And symmetrical?
- Is the heel in varus, neutral, valgus (coronal plane)? Is it in equinus or calcaneus (in the sagittal plane)?
- Is the whole forefoot plantarflexed (plantaris) or is the first ray most plantarflexed?
- Is there toes clawing? Callosities, ulceration?
- Shoes and walking aids
- Watch the patient walking

Palpation

- Are the deformities correctable or fixed? How easy correctable?
  - Hindfoot

    Varus – Coleman's test (Figure 14.25)
    Equinus – Can be brought down. Check for TA tightness (Silverskjőld's test)

  - First ray – Can be brought level with the other rays? Secure the hindfoot with left hand in neutral position and look at the rays from front. Are they level? If not, can they be brought level?
  - Is the toe deformity correctable? (Blackburn grading)
- Assess for tenderness at the callosities
- Assess the sensation – Usually gloves and stocking type in HSMN and dermatomal in spinal disorders
- Assess foot circulation as it may have impact on surgical intervention.

Movement

- Assess active movement of each joint, paying particular attention on the powering muscle. Common pattern is the foot dorsiflexion powered by the toes flexor rather than tibialis anterior
- If the active movement is not full, try passive movement to achieve the full range
- Full neurological assessment to identify the cause.

Investigation

- Radiology
  - X-rays
    1. Weight-bearing lateral (calcaneal pitch – Normal $<30°$ and lateral Meary's angle – Normal 0–5°)
    2. PA (Meary's angle – normal 0°)
    3. Coby's view (calcaneotibial angle $<5°$)
  - MRI (spine and brain when indicated)

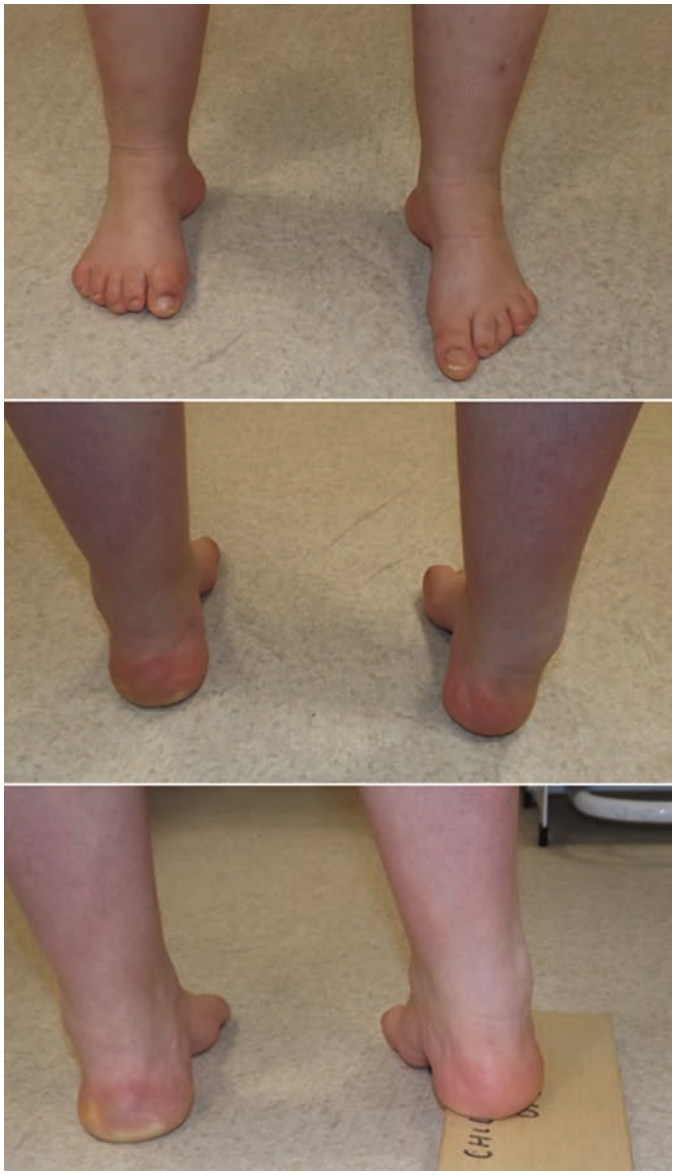

Figure 14.25 Pes cavus and Coleman's test. Coleman's test shows the heel varus fully corrects indicating the primary deformity is the hyperflexion of the first ray. Surgery to elevate the first ray will improve the apparent heel varus and there is no need for calcaneum osteotomy

- Bloods such as muscle enzymes and genetic screening
- Neurophysiology may be indicated in assessing underlying neurology

Treatment

Conservative

- Physiotherapy: Tendoachilles stretching or strengthening exercises, muscle strengthening may improve muscle imbalance
- Orthotics and accommodative

Operative

Operative intervention may be indicated when the child becomes symptomatic, and when orthotics is ineffective, but before the feet become stiff. The aim of surgery is to achieve pain-free, plantigrade, supple but stable foot. There are various types of operations that may be beneficial depending on the condition of that particular cavus foot. It is recommended to know the basic principle of these operations. These can be summarized:

1. Release of the plantar fascia.
2. Closing-wedge dorsiflexion osteotomy of the first (± second) metatarsal
3. Calcaneum sliding and closing-wedge osteotomy
4. Transfer of the peroneus longus into the peroneus brevis at the level of the distal fibula
5. Achilles tendon lengthening
6. Clawing of the toes is improved by flexor-to-extensor transfers and extensor tendon lengthening or tenotomy
7. Jones' procedure
8. Triple arthrodesis

In-toeing/out-toeing

Another standard in children's clinics and something you should be able to rattle through quite swiftly.

General points

There is a wide range of normal values for rotational alignment in children and adults. Pathology should be suspected when there is:

- Pain
- Limp
- Length discrepancy
- Asymmetry
- Rapid change in rotational profile

Examination (Table 14.8 and Figures 14.26 and 14.27)

Ask about height and weight, think metabolic bone disease/bone dysplasia.

Stand patient in front of you, assess leg lengths and Trendelenburg's. In an adolescent, think slipped upper femoral epiphysis (SUFE)! Quick look at back – Never hurts.

Ask the patient to walk the longest distance you have available. Assess the foot progression angle (FPA) (Figure 14.26). This is the angle subtended between the straight line along which the patient is walking, and lines drawn through the long axes of the footprints. This can be measured with some accuracy if the patient is made to step in chalk powder before walking or with video gait analysis. In the clinic the foot progression angle is usually eyeballed. Normal FPA ranges from –5° to 20°. In-toeing of –5° to –10° is mild, –10° to –15° moderate and more than –15° is severe.

Direct the patient to the examination couch and ask them to lie prone. Put a pillow under the chest with arms flexed and hands under the chin – This is comfortable and avoids squirming. Flex the knees and start assessing the rotational profile from feet upward or from hip downward. My preference form: Feet upward!

Table 14.8 Summary evaluation of rotational profile of the lower limb

Measurement	Normal (°)	Significance
Foot progression angle	−5 to +20	< −5 in-toeing; > 20 out-toeing
Foot lateral border (Some use heel bisector line)	Straight	Curved inward, metatarsus adductus
Transmalleolar thigh angle	0–40	Outside the range indicates tibial torsion
Thigh foot angle	0–20	Outside the range indicates tibial torsion
Prone hip internal rotation	20–60	Outside the range indicates femoral torsion
Prone hip external rotation	30–60	Outside the range indicates femoral torsion
Femoral anteversion	40 at birth 20 at 5 years 16 at 16 years	Higher values indicates persistent femoral anteversion

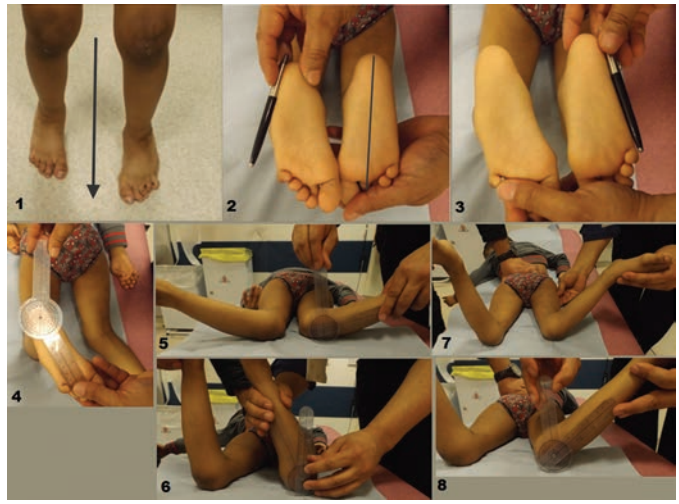

Figure 14.27 Assessment of rotational profile of the lower limb. 1: Foot progression angle; 2 and 3 lateral border of the foot and heel bisector line; 4: Foot thing angle; 5 and 6 prone hip internal and external rotations respectively; 7 and 8 femoral anteversion

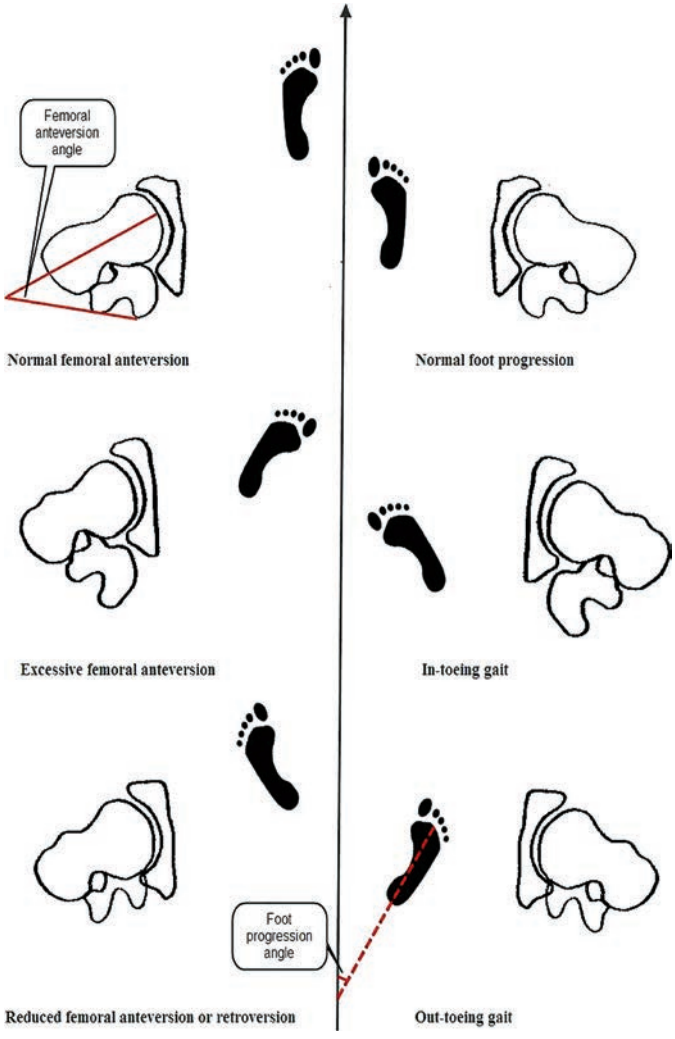

Figure 14.26 Foot progression angle and relationship to femoral anteversion

Describe the shape of the foot in general terms. Someone with significant metatarsus adductus or a skew-foot may appear to have in- or out-toeing simply due to the position of the foot itself.

With the knee flexed at 90°, kneel up on the couch and peer down the axis of the tibia in order to assess the foot–thigh angle (FTA). This is the angle subtended between the line of the thigh and that good old imaginary line through the middle of the foot. An unscrupulous clinician can of course twist the foot to give just about any angle desired and the interobserver error in this measurement is alarming. If one earnestly wishes to take a reproducible measurement, the trick is to gently hold the foot in neutral dorsiflexion and try and do the same each time. Normal FTA ranges between 0° and 20°. This angle measures the tibial and hindfoot rotational status. The transmalleolar thigh angle (TMA) is a measure for tibial torsion only and with practice, you can assess it relatively easily. Normal value of TMA is 0–40° and a value <0° suggests internal tibial torsion.

Hip rotation: Flex the knees to 90° and use the tibias as goniometers to measure hip internal rotation (<70°) and external rotation (<30°) on each side.

Measure the femoral anteversion (Gage's test): find the greater trochanter and palpate gently with one hand while rotating the femur as above with the other. Judge when the lateral prominence of the trochanter is at its greatest and record the degree of hip rotation that corresponds with that. This measurement appears rather improbable when written down; in practice it is surprisingly convincing. Practice on a friend in advance and don't waste time over it in the exam. Normal femoral anteversion is about 40° at birth, 20° by the age of 9 and reaches the adult value of 16° by the age of 16 years.

Try to use the correct terms. Version when the rotational profile is within normal and torsion if it is abnormal. So if the version of the femur is >40° at 9 years, this is called internal femoral torsion.

Do not overlook the fact that muscle tone/pull in patient with cerebral palsy (CP) may cause in-toeing or out-toeing.

The written description above seems rather complex, but in reality and with some practice the whole procedure can be done in well under a minute. If you don't have a compliant friend or partner to practice on, the sequence of examinations can be usefully rehearsed using a doll or large soft toy. Really.

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Chapter 15

General viva guidance

Abhijit Bhosale and Stan Jones

Having passed the part 1 every candidate should have a reasonable knowledge of orthopaedics but further refined preparation is essential for the part 2.

The part 2 section of the examination is clinically oriented. The first day involves examination of patients:

- Intermediate cases (an upper and lower limb) each lasting 15 minutes and candidates are expected to take an appropriate history, examine in a structured and friendly manner, discuss the relevant investigations and formulate a treatment plan for the respective patients/cases
- Three short upper limb cases lasting 15 minutes in total
- Three short lower limb cases also lasting 15 minutes

The short cases involves examination of patients to demonstrate relevant clinical signs and, thus, make a diagnosis. Candidates may be required to discuss management of the respective patients.

The second day is dedicated to viva examinations. Candidates will be examined across four viva stations, i.e. paediatric/hands, adult pathology, trauma and basic science. In each of the stations, the candidates will be examined for 15 minutes by each of the 2 examiners and are marked by each examiner separately. During each 15 minutes section each candidate will be examined on 3 separate clinical scenarios each lasting 5 minutes. At the end of the 15 minutes a bell is rang and the candidate will then be examined by the second examiner. Each examiner scores the candidates for each of the respective 5 minutes sections.

How to prepare for the viva

The key to success in the viva examination is to have a broad knowledge of the curriculum, listen to the examiner, answer the questions asked and do so in a structured and confident manner.

Many candidates prepare seriously for the clinical section of the examination only after passing part I. This is not advisable as there may not be adequate time between the two parts of the examination to enable one to cover the breadth of the curriculum and, hence, be fully prepared. Remember failure is costly financially, mentally and physically.

Preparation for the viva examination involves assimilation of further knowledge by reading standard textbooks such as Postgraduate orthopaedics and Miller's textbook of orthopaedics. Many examiners also use these books. Knowledge gained is then reinforced by viva practice.

Practice can be gained by having mock viva sessions:

- With registrar colleagues who may also be preparing for or have just passed the examination
- With consultant colleagues (who may be examiners) in their offices or clinics
- At the local teaching programme
- During local trauma meetings

It is better to make mistakes at these sessions and learn from them than to do so at the main examination as that can lead to failure. In addition, further knowledge can be gained at these sessions.

We suggest candidates do not prepare for the viva examination in isolation. It is good to be part of a revision group. Some candidates may find it beneficial to revise with others of similar knowledge while others may find it stimulating to work with others who have more knowledge and, thus, stimulate them.

Courses

Examination revision courses are a useful preparation tool, but these courses can be expensive and not all are really worth the expense, time off work, traveling to the venue and hotel costs. Courses come in different formats, i.e. clinical, viva or lecture or a combination of these. A clinical-oriented course is a must.

It is advisable to go on at least one course, but we recommend that you inquire from colleagues who have just passed the examination which courses they found most beneficial. Courses are also a good forum to gather some useful tips from other colleagues or the lecturers who may also be examiners. Be careful however that any info is accurate and bonafide and hasn't been exaggerated third or fourth hand by candidates. Be also careful with candidates downplaying the exam. Misleading information can seriously damage your well being!

Structured questions

As of November 2014 viva questions have been pre-set by the examination board. Examiners meet the evening before the clinicals and for each session of the viva (morning/afternoon) chose from a set list of questions. The examiners conducting the viva examination will choose six questions from a total of

nine questions. Some questions may be ambiguous and, therefore, need to be clarified or discarded. Other questions may include laminated clinical photos that are unclear or of poor quality such that they are amended or discarded. Examiners will generally avoid questions that fail to differentiate between good and badly performing candidates. Sometimes the biomechanics or statistics question that no one expects to be chosen is snapped up by examiners who may actually enjoy vivaing on the topic. Some questions may be ambiguous and, therefore, need to be clarified or discarded. Other questions may include laminated clinical photos that are unclear or of poor quality such that they are amended or discarded. Each question has a model answer and the subsection examiners around a table discuss and agree on a minimum level of knowledge required to safely pass a viva question with a score 6. They reach a consensus opinion usually fairly quickly.

Scoring

Score 8. Generally a candidate will keep on talking and the examiners may occasionally run out of scripted questions to ask. Can quote the literature to support an answer

Score 7. Doing well for most of the oral but some gaps in places. Occasional prompts

Score 6. Score 6 covers a large difference in performance. This will be from a well-polished answer which has been generally very good except for a couple of silly mistakes that has dragged the mark down to a scrapped through score 6 where the candidate was a whisker away from failing and scoring a 5. Sometimes a candidate is absolutely fine with basic questions but just can't raise their game when more difficult questions are asked. With each topic the examiner usually gives a candidate a couple of opportunities to go further with their answer and score a higher mark.

Score 5. Big gaps in knowledge. Large chunks of the topic unanswered.

Score 4. The candidate has said something unsafe or dangerous. Does not understand the question or where it is going. Unfamiliar with topic. Can't keep the question moving forward. Like pulling teeth out for the examiners

Examiners' etiquette

Examiners have been encourage to make full use of the marking system from 4 to 8. Each pair of examiners marks independently and are not allowed to discuss their score with each other until after awarding their marks. Any major discrepancy in marks (2 marks or more) needs to be fully justified and investigated. They have to remain attentive, be articulate and encourage candidates to perform to the best of their ability. They should be objective, open minded, unbiased and be able to move a question forward appropriately. An examiner should not keep hammering on a point especially if a candidate doesn't know the answer.

Examiners should put the candidate at ease at the beginning of the viva and introducing themselves and making sure the candidate is aware of what viva they are involved with. Examiners are not allowed to discuss any candidate's performance or marks with any other team of examiners at any time of the exam. Examiners should give neutral feedback and avoid negative or positive comments. They should allow the opportunity for candidates to answer the question and remain fairly quiet during the viva, at maximum contributing 20% of the discussion. Examiners are there to assess candidates not to teach. The viva exam is a summative exam and enough information needs to be gleaned by the examiners to confidently give the candidate an appropriate mark. If a candidate is failed in general more copious notes are made by the examiners to provide available evidence to justify their decision.

The co-examiner should make notes whilst the other examiner is asking questions. They should not interrupt, leave the examination table to answer their phone, appear bored or disinterested or fall asleep.

Candidates' etiquette

A small number of candidates perhaps through intense 'nerves' or 'stress' may behave in a bizarre dysfunctional manner. This may involve invading an examiners personal space, appearing odd with strange visual expressions or just being all over the place. This behaviour should have been spotted and dealt with by intensive professional coaching and feedback before a candidate even had a chance to sit the exam. Just as serious is arguing, being rude, disrespectful or arrogant. Examiners are advised to ignore this type of behaviour but we live in the real world and think it is likely to subconsciously affect a candidate's mark. Viva courses that mimic the real exam are important as they allow a candidate the chance to dry run their performance. Good courses should involve experienced exam-focused consultants who can provide appropriate feedback. Poor body language and eye contact can be addressed at this stage before it becomes too late. A hesitant and anxious performance can be practiced and honed in on until a candidate comes across as articulate and confident in their viva approach. This is one of the main advantages of joining a group to revise for the exam.

Tips about the viva examination

- Listen to the question being asked and answer appropriately.
- Maintain eye contact with your examiners and smile.
- If asked how you would manage a condition start by saying 'I would'. Always refer to yourself
- When asked about a topic, it is legitimate to keep talking until you are stopped and it is advisable not to invite any leading questions that you do not have answers for.
- Do not argue with examiner, even if you know you're right! In case of controversy, quickly move on as smoothly as you can.
- If an examiner says, 'Are you sure?' It suggests your answer is not correct. Take the hint

- If you do not know the answer to a question do not stay mute but let the examiner know you have no idea so he/she can move on to another question.
- If you do not understand the question being asked you are justified in asking the examiner to repeat it. Don't keep asking each question to be repeated however.
- Do not keep thinking about previous poorly answered questions but move on.
- Do not start an answer quoting references, unless it is very obvious (e.g. Baumgartner's paper on tip apex distance)
- If asked to comment on a fixation, don't criticise the fixation straightaway, always start by saying that the case may have been difficult. Give your balanced opinion on it. It creates a bad impression if you are very critical or condescending of a poor fixation as it suggests you may end up being an unsupportive colleague in the future.
- Practice drawing figures such as the Selenius graph and brachial plexus, etc. and be sleek.
- It is not necessary to know a long list of references to pass the viva. At the same time candidates are unlikely to score an 8 with no idea at all about the literature.
- In between vivas do not discuss your questions with your colleagues

Good luck!

Chapter 16

Hip oral core topics

Sammy A. Hanna and Paul A. Banaszkiewicz

Anatomy of the hip

Surgical approaches

These are common and important surgical approaches. A thorough knowledge and understanding of the five main approaches to the hip joint (medial, anterior, anterolateral, lateral and posterior) is required. Do not just stick to just the posterior or Hardinge approach as the medial approach (in the paeds viva) and anterior approach (recent renewed interest) may sneak into a viva discussion. That said the posterior approach to the hip joint is probably the most commonly asked surgical approach in the whole FRCS (Tr & Orth) exam so you would be absolutely crazy not to learn this approach inside out and back to front[a].

When asked about an approach, we suggest you structure your answer as follows:

- Indications for the approach
- How to set up the patient
- Anatomical landmarks and location of incision
- Internervous planes (if any)
- Extensile measures (if any)
- Structures at risk
- Limitations of the approach

Colour atlas pictures

Candidates may be asked to identify structures labelled in a blank manner on a colour atlas picture. Make sure that you practice identifying relevant anatomical structures around a hip joint in a colour atlas textbook before the exam. The examiners tend to lift these pictures from the more popular colour atlas textbooks or CDs on the market. Hardcore candidates should consider using these particular atlas books for revision to enhance chances of success in the FRCS (Tr & Orth) exam, although this may be taking things just a little too far. These are quite straightforward viva questions and candidates would be expected to identify the vast majority of anatomical structures correctly. That said some of these anatomical pictures can be quite detailed and complicated.

Blood supply of femoral head

This is a favourite question in either the trauma, basic science or adult pathology oral. This may lead into a discussion about avascular necrosis (AVN) of the femoral head. The blood supply has three sources:

1. The medial circumflex femoral artery (MCFA) is the most important supply; it is a branch of the profunda femoris artery
2. The lateral circumflex femoral artery (LCFA) supplies the inferior portion; it is a branch of the profunda femoris artery
3. The artery of the ligamentum teres, a branch of the obturator artery or occasionally the MCFA. Forms the medial epiphyseal vessels. Usually unimportant with only small amount of the femoral head supplied from this artery

The main contribution stems from the MCFA deep branch, the lateral epiphyseal artery. This supplies the majority of the head and neck. The LCFA supplies the anterior inferior head. At the base of the neck the ascending branches of the medial and lateral circumflex arteries form an **extracapsular arterial ring** with minor contributions from the superior and inferior gluteal arteries. The extracapsular arterial ring gives off **ascending cervical arteries** that travel upwards under the hip capsule and along the femoral neck deep to synovial membrane continuing toward the femoral head as **retinacular arteries**[1]. The retinacular arteries are divided into three groups

- Posterior inferior and posterior superior (from medial femoral circumflex artery)
- Anterior (from lateral femoral circumflex artery)

At the margin of articular cartilage on the surface of the neck of femur the retinacular arteries form a **subsynovial intracapsular arterial ring**, that supplies the head through multiple ascending epiphyseal arterial branches that go on to enter the head of the femur (lateral epipyseal most important) (Figure 16.1)[b].

[a] The surgical approach part of a viva topic can sometimes be the differentiating section of a viva, where if you answer it reasonably well you can scrap through an otherwise ordinary viva performance, but if you are unconvincing and lack confidence you may end up with a disappointing marginal fail.

[b] Have a mental picture of Figure 16.1 in your mind to revise from – It's much easier.

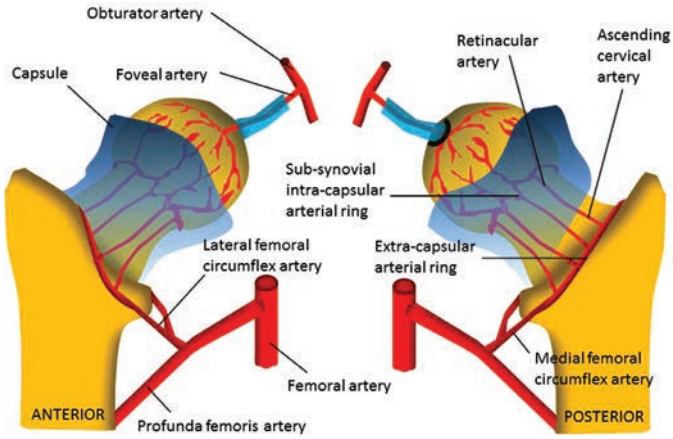

Figure 16.1 Blood supply of the femoral head

Epiphyseal blood supply: tenuous supply from:

- Branches of the subsynovial intra-articular ring that enter the head posterosuperiorly (lateral epiphyseal arteries)
- Vessels from medial epiphyseal artery entering through the ligamentum teres
- Anastomoses with metaphyseal arteries

Metaphyseal blood supply: excellent supply from:

- Extracapsular arterial ring
- Branches of ascending cervical arteries
- Branches of the subsynovial intra-articular ring
- Intramedullary branches of the superior nutrient artery

Examination corner

Basic science oral 1: Blood supply femoral head

Second question after initial discussion on the position a leg assumes after a traumatic posterior and anterior dislocation of the hip.

EXAMINER: What is the blood supply of the femoral head?

CANDIDATE: The blood supply to the femoral head is derived from the medial and lateral femoral circumflex arteries; these are branches of the profunda femoris artery. They form an extracapsular arterial ring around the base of the trochanter. Ascending cervical arteries are given off this ring, which then branch into retinacular arteries, which form a subsynovial intracapsular arterial ring.

The candidate was stopped in mid sentence by the examiner, who was satisfied with the answer and wanted to move on to another question. This led on to a discussion of AVN of the hip and types of total hip arthroplasty (THA) to use in a young patient.

Basic science oral 2: Anatomy of the posterior thigh

The examiner had a colour-laminated photocopy of the whole of the posterior thigh from an atlas.

- Identification of bare labels on it. All muscles had to be identified with attachments and nerve supply
- Identification and surface anatomy of the sciatic nerve
- Popliteal fossa: Anatomy, approaches and the neurovascular structure arrangement
- Posterior approach to the hip: Structures going above and below the piriformis muscle, anatomy of the superior and inferior gluteal nerves and arteries. Identification of pudendal nerve and nerve to obturatus internus beneath the piriformis muscle
- Safe zones for acetabular screws
- Femoral head blood supply

Basic science oral 3: Anatomy of posterior hip and thigh

- Surface markings of the sciatic nerve
- Causes of superior gluteal artery injury
- Hamstring origin
- Approaches to the hip joint

Basic science oral 4: Hemipelvis anatomy

- Laminated picture of a hemipelvis with outline of muscle origins. Candidates were asked to identify five or six muscle origins

Basic science oral 4: Anatomy posterior aspect hip

- Name the structures on the posterior aspect of the hip. A colour figure was provided with bare labels

Basic science oral 5: Gluteal muscles

- Anatomy of the gluteal muscles and pelvis
- Surgical approaches to the hip joint; complications of the direct lateral approach

Trauma oral 1: Surgical approaches

Neck of femur fracture in a 45-year-old patient. How to reduce closed – Essentially asked to describe the Ledbetter's technique – Flexion/Traction/IR/Ext/ABD:

- The hip is fully FLEXED
- and slightly ADDUCTED as TRACTION is applied
- Full INTERNAL ROTATION is then applied
- Leg is then CIRCUMDUCTED into slight abduction while the rotation is maintained
- then brought into EXTENSION

Trauma oral 2: Surgical approaches

- Anteriorly or posteriorly dislocated hip. Failed closed reduction Describe open reduction and approach used

Osteonecrosis of the femoral head[c]

Definition

Osteonecrosis (ON) is the death or necrosis of bone secondary to loss of its vascular supply or more simply 'death of bone from ischaemia'.

Causative factors

A variety of aetiological associations with ON have been demonstrated (Table 16.1).

[c] The term osteonecrosis is preferred to 'avascular necrosis' or 'aseptic necrosis' because it does not imply a specific aetiology.

Table 16.1 Causative factors of AVN

• Idiopathic
• Trauma
• Steroid therapy
• Alcohol
• Caisson's disease
• Sickle cell disease and other haemaglobinopathies
• Post irradiation
• Gaucher's disease[d]
• Hyperlipidaemia
• Systemic lupus erythematosus (SLE)
• Renal transplantation
• Burns
• Endotoxin reactions
• Pancreatitis
• Nephritic syndrome
• Inflammatory bowel disease

Vascular disturbance of the femoral head

Aetiological factors in AVN are usually related to underlying pathological conditions that alter blood flow, leading to cellular necrosis and ultimately femoral head collapse. This damage can occur in one of five vascular areas around the femoral head:

Intravascular

1. **Extraosseous arterial factors** are the most important. The femoral head is at increased risk because the blood supply is an end-organ system with poor collateral development. Blood supply can be interrupted by trauma, vasculitis (Raynaud's disease), or vasospasm (decompression sickness)
2. **Intraosseous arterial factors** may block the microcirculation of the femoral head through circulating microemboli. These can occur in sickle cell disease (SCD), fat embolisation or air embolisation from dysbaric phenomena
3. **Intraosseous venous factors** affect the femoral head by reducing venous blood flow and causing stasis. Obstruction of venous drainage. Conditions such as Caisson's disease or SCD

Extravascular

4. **Intraosseous extravascular factors** affect the hip by the increasing the pressure, resulting in a femoral head compartment syndrome. Fat cell hypertrophy after steroid administration, lipid deposits in marrow extravascular space or within osteophytes can create an elevation of intraosseous extravascular pressure. There is encroachment on intraosseous capillaries resulting in decreased intramedullary circulation
5. **Extraosseus extravascular (capsular) factors** involve the tamponade of the lateral epiphyseal vessels located within the synovial membrane. This can occur after trauma, infection or arthritis causing a hip effusion

Pathophysiology

The pathophysiology is still controversial and uncertain. Several theories have been put forward[2].

- **Intraosseous hypertension theory** (compartment syndrome bone) Increased pressure in a closed rigid osseous compartment of bone increases intraosseous pressure. Blood flow through the intraosseous compartment is inversely proportional to the bone marrow pressure; thereby, any condition that causes an increase in this pressure will produce a decreased blood flow to bone in that area resulting in secondary ischaemia and AVN
- **Abnormality of extraosseous blood flow.** There may be significant differences in the regional vascular anatomy between individuals that predispose them to AVN
- A **fat emboli** phenomenon in subchondral arterioles. This results in intraosseous coagulation, which leads to generalized venous thrombosis and retrograde arterial occlusion
- One hypothesis considers AVN is caused by **fat cell hypertrophy** in which fatty marrow overload leads to increased bone marrow pressure inside the femoral head resulting in sinusoidal vascular collapse and AVN
- Other authors believe the condition is caused by a direct **cytotoxic effect** on osteocytes (alcohol)
- Several studies have demonstrated **clotting abnormalities** such as deficiencies in protein S, protein C and antithrombin III in patients with ON. A few studies have shown the presence of both hypofibrinolysis and thrombophilia in patients with ON. Both hypofibrinolysis and thrombophilia are associated with an increased incidence of thrombotic events that may contribute to the pathogenesis of osteonecrosis

[d] When mentioning causes of ON stick to the most common ones first. If you start with Gaucher's disease you are inviting trouble. It isn't the most obvious cause of ON and may irritate the examiners enough for them to switch the topic. Gaucher's disease is a cause of ON but it is small print and should be mentioned near the end of your list after the more obvious causes have been discussed (trauma, alcohol, steroid use, etc).

EXAMINER: How common is Gaucher's disease as a cause of ON?

CANDIDATE: Not very common

EXAMINER: But since you've mentioned it, tell me you all you know about Gaucher's disease.

The examiners will think your reasoning is poor, you have no sense of order of priority in your answer and you have not demonstrated higher order thinking.

Table 16.2 Ficat classification of AVN

0	Silent	Preclinical and pre-radiographic Diagnosis suspected in one hip when the other hip has definite disease Bone marrow pressure studies abnormal and core biopsy would reveal characteristic histological patterns. This stage was not described in original classification. No clinical symptoms. Normal radiographs. MRI non-diagnostic
1	Preradiographic	Radiographs usually normal or at most minor changes such as subtle loss clarity with poor definition or blurring of trabeculae pattern. Diagnosed with a positive MRI or bone scan. Earliest clinical manifestation of the syndrome. Usually presents with sudden onset of ischaemic hip pain in the groin with or without radiation down the front of the thigh
2A	Precollapse (Before flattening of head or sequestrum formation	This extends over several months with clinical symptoms and signs persisting or worsening. Radiographs demonstrate osteopenia/sclerosis femoral head
2B	Crescent sign	Curvilinear subchondral radiolucent line due to subchondral fracture
3	Collapse Bone sequestrum broken off	Segmental flattening and collapsed femoral head. Worsening pain, limp, limited range of motion in all planes
4	Osteoarthritis	Terminal phase, secondary degenerative change superimposed on a deformed femoral head

- Jones has suggested that a combination of three factors result in microcirculatory thrombus leading to ON: Stasis, hypercoagulability and endothelial damage[3]. Many mechanisms are overlapping and mutually supportive and all eventually lead to a final common pathway of vascular occlusion and ischaemia leading to both marrow cell and osteocyte necrosis

Clinical features

Usually non-specific with insidious-onset hip pain, which is worse with weight-bearing, often present at rest and eventually at night. It is associated with a decreased or painful range of hip movement, limp, muscle weakness and antalgic gait.

Radiology

- **Radiographs.** AP and frog-leg lateral radiographs. Normal in the early stages of the disease
- **Bone scan.** Poor sensitivity especially with early disease
- **MRI scan.** Generally accepted as the gold standard for confirming a suspected clinical diagnosis of ON and asymptomatic contralateral disease

ON is diagnosed when a peripheral band of low signal intensity is present on all imaging sequences, typically in the superior portion of the femoral head, outlining a central area of marrow. This peripheral band is most apparent on T1-weighted sequences. On T2 sequences, the inner border of the peripheral band shows a high signal in 80% of cases. This is called the 'double line' sign and is pathognomonic for ON.

Differential diagnosis

- Transient osteoporosis of the hip: A self-limiting condition, MRI demonstrates oedema into the femoral neck and metaphysis, which is not common with ON
- Femoroacetabular impingement: Symptoms tend to be more mechanical and do not usually occur at night. Positive impingement test
- Acetabular labral tear: Patient describes snapping and 'clicking' in the groin. Symptoms also mainly mechanical
- Sports hernia: Can be associated with a lump or 'fullness' not seen in AVN

Classification systems

Ficat

The original classification in 1964 did not include stage 0. Stages 0–II are described as early stages and Stages III and IV are classified as late stages. Stages II and III represent the distinction between precollapse and collapse. This classification system established the premise of staging osteonecrosis and subsequent classification systems[4] (Table 16.2).

Steinberg (University of Pennsylvania)

Seven-stage system (0–6)[e]. It is considered more useful than Ficat because it grades the severity and extent of the involvement, both of which affect prognosis (Table 16.3).

Mitchell MRI staging classification of AVN[5]

The Mitchell classification may be useful in grading lesion acuity, as infarcted bone will tend to progress through the classes of signal intensity over time. However, this progression is not always consistent, and more than one class of signal abnormality may be found in a single lesion (Table 16.4).

The prognostic value of MRI is more dependent upon the size and location of the lesion. Small lesions confined to the medial anterosuperior portion of the femoral head tend not to collapse. AVN that does not contact the subchondral margins tends to have a good prognosis regardless of lesion size.

[e] Easy to mix up and say that the Steinberg classification is a six-stage system because you remember the number 6. It's not, as there are seven stages, from zero to six. Some clever examiner will pick you up on this.

Table 16.3 Steinberg classification of osteonecrosis adult hip

0	Normal x-ray, bone scan and MRI, diagnosed on histology
I	Normal x-ray, abnormal bone scan or MRI findings (minimal pain)
II	Sclerosis and/or cyst formation in the femoral head
III	Subchondral collapse (crescent sign) without flattening
IV	Flattening of the femoral head without joint narrowing or acetabular involvement
V	Flattening of the femoral head with joint narrowing and/or acetabular involvement
VI	Advanced degenerative change

Notes:
Volume head involvement (VHI): Minimal (<15%), moderate (15–30%), extensive (>30%).
Surface collapse and dome depression: 2 mm, 2–4 mm, >4 mm.
Location: Medial, central, lateral.

Table 16.5 Management of ON

• Pre-collapse	
Prevention	Alcohol
Non-operative	Protective weight-bearing Pharmacological: bisphosphonates, iloprost, statins Hyperbaric oxygen Pulsed electromagnetic field
Joint-sparing procedures	Core decompression Vascular fibular graft
• Post collapse	
Osteotomy	Varus/valgus Rotational
Joint arthroplasty	Bipolar hemiarthroplasty Cemented THA Uncemented THA
Arthrodesis	

Classic reference

Ficat RP. Idiopathic bone necrosis of the femoral head. Early diagnosis and treatment. *J Bone Joint Surg Br*. 1985;67:3–9.

Toulouse

Ficat and Arlet proposed the original classification of avascular necrosis in 1964 before the advent of MRI. It consisted of stage I through to stage IV and did not include stage 0 previously identified by Hungerford in 1979.
In 1985 the Ficat classification added a stage 0, also known as 'silent hip'. Stage 0 is both preclinical and preclinical with the diagnosis suspected in one hip when the other hip has AVN. This was diagnosed on a positive functional exploration of bone.
Despite Mont et al. identifying at least 16 classification systems in use to grade and describe avascular necrosis , the Ficat system continues to be the most widely used system. One significant change is that

Table 16.4 Mitchell MRI staging classification of AVN

• Class A	Bright on T1	Intermediate on T2 (fat)
• Class B	Bright on T1 and T2 (blood)	
• Class C	Intermediate on T1	Bright on T2 (fluid and/or oedema)
• Class D	Dark on T1 and T2 (fibrous tissue)	

histological examination of the hip (biopsy) is no longer required for a diagnosis of AVN. MRI is the most sensitive and specific diagnostic method.

Prevention

- Identification and elimination of risk factors. Applicable to alcohol intake and steroid administration
- Adherence to established safety guidelines for divers and those working under hyperbaric conditions

Management (Table 16.5)

Goals of management are to relieve pain, improve function, minimize morbidity and maintain options for secondary procedures. Four radiographic finding are routinely used when formulating a management plan: (1) Is the lesion precollapse or post-collapse; (2) size of necrotic segment; (3) amount of femoral head depression; (4) acetabular involvement with signs of osteoarthritis.

Non-operative

Observation (protective weight-bearing)

Not a good option as most patients do poorly. Collapse of the femoral head was noted by Ohzono et al.[6] to occur in 80% of patients within 4 years of onset of hip pain (success rates for Ficat stage 1, 35%; stage 2, 31%; stage 3, 13%). Observation may be indicated in those with very limited disease or if the patient is not fit enough for surgery. Start with non-weight-bearing with progression to full weight-bearing when clinical symptoms and signs demonstrate that the hip is less irritable. Radiographic and clinical follow-up is essential until the hip pain subsides.

Non-operative pharmacological management

Lipid-lowering agents, statins, anticoagulants and bisphosphonates have all shown promising results but require further research and clinical reports regarding their efficacy. Agarwala et al.[7] reported on the efficacy of alendronate in the medical management of ON. They demonstrated improvement in symptoms (walking time, standing time, pain and disability), retarded progression of the disease and reduced rate of collapse of the femoral head. Mode of action is inhibition of osteoclastic activity, which reduces oedema, and the rate of remodeling in the femoral head. This then increases bone mineral density

and as such delays the progression of bone collapse. At a mean follow-up of 4 years, 364 hips (92.2%) had a satisfactory clinical result and had not required any surgery. Failure requiring THA occurred in 4 of 215(2%) Ficat stage 1 hips, 10 of 129 (8%) Ficat stage 2 and in 17 of 51 (33%) Ficat stage 3 hips.

Iloprost is a vasoactive compound used in the treatment of vascular occlusion, vasculitis and pulmonary hypertension. It acts on the terminal vascular bed by inducing vasodilatation, reduction of capillary permeability and inhibits platelet aggregation[8]. Promising early clinical and radiographic improvements at 1 year have been reported[9].

Animal studies have shown that statins reduce bone marrow adipocyte size and, therefore, potentially reduce intraosseous pressure within the femoral head. They have pro-osteoblastic and anti-adipogenic effects on bone marrow stromal cells. These effects protect against corticosteroid-induced osteonecrosis.

Electrical stimulation

Only a few short-term studies have been published in peer-reviewed journals and, whilst they report encouraging early results, most orthopaedic surgeons remain skeptical and this management option has not proved widely acceptable. More long-term studies are required and, therefore, it remains experimental and requires further evaluation as part of a RCT[f].

Hyperbaric oxygen (HBO) therapy

One small study[10] reported beneficial effects for stage-1 AVN. Daily HBO therapy was given for 100 days. Overall, 81% showed a return to normal on MRI as compared with 17% in an untreated group. A recently published randomised trial by Camporesi et al.[11] has also shown encouraging results. More detailed studies are needed to evaluate this treatment modality. Mode of action is reversal of cellular ischaemia by increasing the oxygen concentration of extracellular fluid and by reducing oedema by inducing vasoconstriction. Drawback is the prolonged course of treatment required.

Joint-preserving methods

The Kerboull necrotic angle is calculated by adding the area of necrosis on the AP and frog-leg lateral views. Patients with a Kerboull angle >200° more commonly have poor results with certain joint-preserving procedures.

Mesenchymal stem cells

Cultured stem cells are injected under fluoroscopic guidance into the necrotic lesion following percutaneous core decompression. A K-wire (diameter 2.7 mm) is used to perforate the interface between the necrotic bone and healthy tissue. The stem cells stimulate neogenesis and new bone formation using the necrotic tissue as a scaffold. This is experimental, and needs further research.

[f] Play it safe. This is what the examiners want to hear.

Core decompression

The principle is to relieve pressure, increase vascularity and stimulate a healing response. This in turn is believed to reduce pain and halt the progression of the disease. Modern percutaneous techniques are simple, safe and particularly effective in the treatment of small lesions at an early stage of disease. There is minimal morbidity and recovery time.

There is some controversy as to the effectiveness of the procedure but a reasonable body of evidence supports its use. Mont et al.[12] published a meta-analysis of patients treated with core decompression covering 1206 hips. Survival rates reported for Ficat stage 1 were 84%; stage 2 65%; and stage 3 47%. Approximately two-thirds do well (half if you exclude centres of excellence with the most experience). Selection of patients is important as if the head is too severely involved the procedure is unlikely to be successful (Figure 16.2).

Core decompression and porous tantalum rod implant

This functions as a structural graft to provide mechanical support and possibly allows bone growth into the avascular femoral head. The operative technique is much simpler than a vascularized free graft. It avoids the morbidity associated with autogenous bone harvesting and the risks of disease transmission with allograft bone. There is some concern about ease of removal and large amounts of metallic debris if there is conversion to THA. One series[13] reported a 15% failure rate, with retrieval analysis demonstrating limited ingrowth response and insufficient mechanical support of subchondral bone.

Non-vascularized bone grafting

Non-vascularized cortical strut grafting using either the fibula or tibia placed into a core tract into the femoral neck combines the effectiveness of core decompression with that of providing mechanical support to the femoral head, thus, retarding its tendency to collapse. The graft provides a source of mechanical

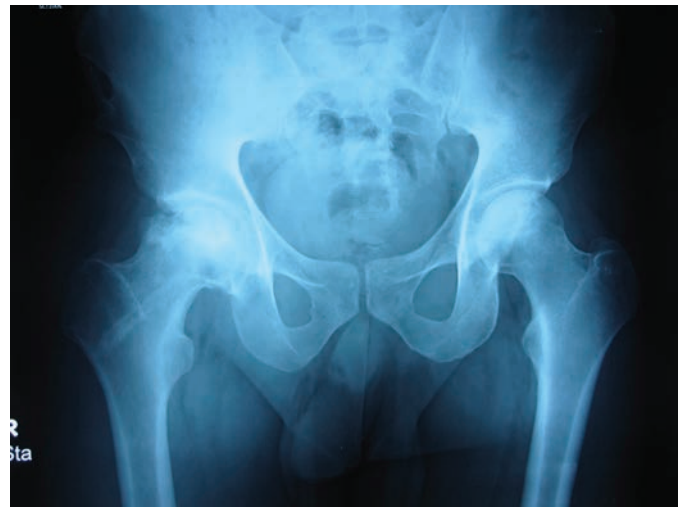

Figure 16.2 Failed core decompression for AVN

support for the articular surface of the femoral head during the healing phase and stimulates neovascularization.

Vascularized bone grafting

Results suggest superior clinical results than non-vascularized grafts. The procedure is technically difficult, time-consuming and requires special equipment and microvascular surgical techniques. Possible option in young patient (<50 years) with precollapse lesion. Consider the diagnosis, patient's age and extent of disease progression.

Trapdoor procedure

Indicated for precollapse (Ficat stage 2). The break in the articular cartilage is exposed following hip dislocation and is opened like a trapdoor. Necrotic bone under the flap is excavated and then removed with a power burr to expose bleeding bone. The defect is then filled with cancellous bone graft. Mont et al.[14] reported encouraging results with Ficat grade 3 (24) and 4 (6), with 73% good to excellent results at 5 years, but hips with Kerboull angles >200° did poorly. Further studies with longer follow-up are needed to assess the usefulness of this procedure.

Muscle pedicle bone grafting

This attempts to preserve the viability of bone graft. Donor sites include the insertion of quadriceps femoris (posterior) tensor fascia lata muscle (anterior) and sartorius. Core decompression performed along with muscle pedicle bone grafting.

Proximal femoral osteotomies

This attempts to shift most of the involved portion of the head medially. There are two general types of osteotomies: Angular intertrochanteric (varus and valgus) and rotational transtrochanteric. They are usually indicated for Ficat stage 2 or 3. The results are best with age 45 or younger, unilateral disease, idiopathic or traumatic aetiology, small to medium area of infarction, no joint narrowing, a combined necrotic angle <200° and a 20° arc of intact lateral femoral head to act as a weight-bearing support. Only applicable to a small number of carefully selected patients and it is difficult to convert failed cases to THA. Avoid if steroid- or alcohol-induced AVN. Reported success rate of 70–80% in stage II–III hips.

Valgus flexion osteotomy indicated for small anterolateral lesions with or without collapse. Corrects adduction deformity common with anterolateral segment collapse with valgus realignment and with the addition of flexion transfers the load to the posterior articular surface. In a small number of patients the necrotic lesion occurs in the medial aspect of the femoral head in which case a varus intertrochanteric osteotomy is indicated. Precise location of the necrotic segment, either anterior or posterior, determines whether flexion or extension is added to the osteotomy.

The Sugioka transtrochanteric rotational osteotomy shifts the diseased portion of the head medially, inferiorly and posteriorly. This is a technically demanding procedure and, although Sugioka et al.[15] reported good to excellent results in 229 of 295 hips (78%) at a mean of 11 years (range 3–16 years), postoperatively these results have not been matched in Europe and the USA.

In summary, a PFO is a reasonable joint-preserving procedure when done by an experienced hip surgeon with osteotomy training in a patient younger than 45 years who has a Kerboull angle <200°.

Joint replacement

Bipolar hemiarthroplasty

Possibly best indicated in an elderly patient whose ON resulted from chronic alcohol abuse and who would be non-compliant with THA precautions postoperatively. Protrusio and erosion of the acetabulum may occur. It is not popular as a high failure and complication rate. Long-term results are not encouraging. Better options in this situation such as dual motion

Hybrid THA

Studies of patients undergoing cemented THA for advanced ON have indicated a high incidence of loosening of the acetabular component. The combination of an uncemented acetabular component and cemented femoral stem offers a different alternative for this difficult problem, with at least one study reporting good medium-term results.

Limited femoral head resurfacing arthroplasty[16]

Main candidates would be Ficat stage III disease, a combined necrotic angle of >200° or >30% involvement, femoral head collapse of >2 mm, and no evidence of damage to the acetabular cartilage. Mixed results reported and initial enthusiasm for use has diminished.

Hip resurfacing arthroplasty (HRA)

With advanced ON it is relatively contraindicated as failure rates may be high. Although early results for AVN in young active patients have been encouraging great concern has arisen for the complications of metal-wear debris and adverse reaction to metal debris (ARDIS). One recent long term study by Amstutz et al[17] (FU 10.8 years, 99 hips) reported excellent results.

Uncemented THA

Although the optimal method of fixation of hip implants in 'dead' bone remains open to debate, some recent studies have reported excellent results using uncemented implants. Cheung et al.[18] reported the outcome of 182 total hip arthroplasties (in 144 patients, 117 AVN hips and 65 non-AVN hips) performed using hydroxyapatite-coated femoral stems. Mean age was 51 years and mean follow-up 14.7 years (range 9.7–19.1 years). Four stems were revised because of aseptic loosening, three in the AVN group and one in the non-AVN group. The 19.1 year survival using revision for aseptic loosening as an endpoint for AVN and non-AVN patients were 97.1% and 96.2% respectively. Stable boney ingrowth was present in 99.5% hips. Johannson et al.[19] in a systematic review found that patients

with sickle cell disease, renal failure and/or transplant, and Gaucher disease have significantly higher revision rates. There was a positive influence on survivorship with the newer designs of cementless THA in ON.

Cemented THA

Historically poor results have been reported for cemented THA in ON. Chandler et al.[20] reported their experience with THA in patients <30 years. They found that 61% of their 'problem hips' were in patients with ON. Stauffer, in 1982, found aseptic loosening of the femoral component in 50% of patients with ON[21]. These results are historic and are based on using first-generation cementing techniques, dated implants and young age of the patient.

In 2002 Fyda et al.[22] reported their results with 53 cemented THA done between 1970 and 1984 in patients with ON. They noted a 28.6% prevalence of femoral loosening and a 29.2% prevalence of acetabular loosening in patients surviving 10 years since the surgery.

Garino and Steinberg[23] reported a 96% survivorship in 123 hips for ON. All femoral components were cemented with second-generation techniques. Seventy-one acetabular components were cemented, and the rest were press-fit and porous-coated. Kim et al.[24,g] analyzed the survival of hybrid and cementless metal on polyethylene (MoP) THAs in young patients <50 years average follow up 9.3 years. They found no significant difference in Harris Hip Score (HHS) and both groups had an overall revision rate of 2%. Although there was no aseptic loosening of the components both groups had high rates of polyethylene (PE) wear and osteolysis.

Cemented THA for ON is an excellent choice in an older patient with low functional demands. With modern cementing techniques it is reasonable option in a younger patients but an uncemented THA may be the better choice and there is more evidence to support uncemented implant use in ON particularly from North America.

Arthrodesis

Arthrodesis may be indicated in young patients with unilateral disease, e.g. trauma. Many cases are bilateral. It is more of a theoretical option for advanced disease than a practical one.

Asymptomatic ON

This is becoming more topical as MRI scans are detecting more asymptomatic early lesions. Mont et al.[25], in a systematic review of untreated asymptomatic ON, found a high (84%) risk of progression of large lesions and a substantial (25%) risk of progression of medium-sized lesions. However, small medially located lesions had a more benign course, with a low risk of progression of <10%. The natural history of asymptomatic medium-sized, and especially large, osteonecrotic lesions was progression in a substantial number of patients. For this reason they recommended joint-preserving surgical treatment in asymptomatic patients with a medium-sized or large, and/or laterally located, lesion.

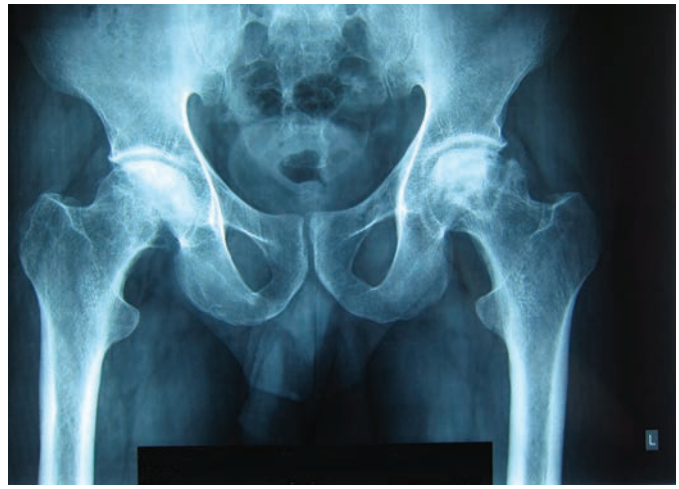

Figure 16.3 AP pelvis showing AVN left femoral head

Nam and Kim[26] demonstrated that the rate of disease progression was related to the extent of necrotic lesion, being 5% for small lesions (<30% femoral head), 46% for medium-sized lesions (30–50%) and 83% for large necrotic lesions. The development of pain is related to the occurrence of femoral head collapse.

Classic reference

Mont MA, Hungerford DS. Non-traumatic avascular necrosis of the femoral head. *J Bone Joint Surg Am*. 1995;77:459.

Review article of published studies of AVN. Meta-analysis of 21 studies (818 hips) with average follow up of 34 months. Methods of treatment were often based on small numbers of patients with different aetiologies and stages of the disease.

Examination corner

Adult elective orthopaedics oral 1: ON hip

Typical GP lead-in letter is a middle-aged male with a short history (4–6 weeks) of severe unilateral or bilateral (less likely) hip pain. Candidates may be given a further clue with the aetiology such as 'has been taking steroids for asthma' or, 'has a history of sickle cell disease', although this makes the diagnosis perhaps too easy. 'There is no history of trauma' may also be included in the introduction.

Candidates will then be shown a radiograph of N and asked to comment (Figure 16.3). ON of the hip is not always obvious on a plain AP radiograph. It is important that candidates do not miss the diagnosis as it is very difficult to recover the viva afterwards and you put yourself on the back foot. The

[g] There are very few direct comparisons of cemented THA vs uncemented THA for ON. This study is slightly confusing as hydrid (uncemented cup/cemented stem) vs uncemented.

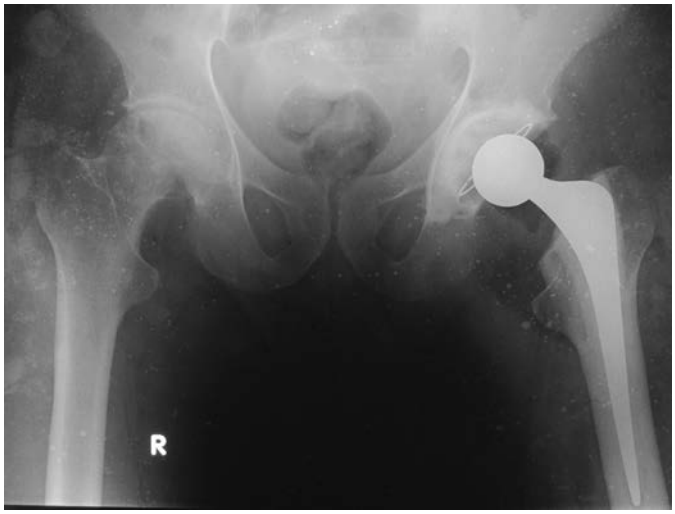

Figure 16.4 Postoperative cemented THA for ON hip

viva question becomes a bare 6 pass needing a nearly flawless performance thereafter.[h]

This is pattern recognition so just keep looking in the orthopaedic books or on various web links to familiarize yourself with the radiographic appearance of early ON. Learn and practice how to describe the radiographic and MRI appearance of ON

The next couple of minutes should be fairly predictable. With aetiology a list of causes should be generated starting with common conditions first.

Pathophysiology is slightly more difficult as you could either discuss the five potential areas of vascular disturbance in ON or go straight to the various theories of ON put forward.

A 7 or 8 candidate would be able to answer in greater detail the specific pathological feature of perhaps steroid induced AVN, alcohol, Caisson's disease and sickle cell disease if probed[i].

Classification should be relatively straightforward[i]. Candidates should really know this in detail – It is reckless not to do so.

Although Ficat et al. is the classic paper the examiners may go for the more detailed Steinberg classification as it is more detailed, has more prognostic value and stretches you a bit further.

With management briefly mention prevention before structuring your answer into conservative, joint-preserving and joint-replacement surgery management.

Recent UK NJR data at 10 years for males <**55 yrs** (all diagnoses) reports a 7.54% revision rate with a **cemented** THA **MoP** bearing surface compared to a **3.57%** revision rate with a cemented THA **CoP** bearing surface. Therefore an acceptable non-controversial answer would be a cemented THA (cemented Exeter stem) and a cemented second generation highly cross-linked PE cup with a ceramic on polyethylene bearing surface (Figure 16.4). Another option especially if the patient is <50 years is an uncemented THA with either a ceramic on ceramic (CoC)- or MCP-bearing surface. Recent UK NJR data at 10 years for males <55 yrs (all diagnoses) reports a **6.35%** revision rate with an **uncemented THA MoP**-bearing surface, 4.50% revision rate with an **uncemented THA CoP**-bearing surface and a 3.47% revision rate for **uncemented THA CoC**-bearing surface. Some examiners may prefer to avoid using an uncemented option in ON because of concerns of a higher failure rate. Therefore candidates will need to justify an uncemented THA choice with some published clinical results specific for ON.

A metal on metal (MoM) hip resurfacing procedure is something candidates should avoid mentioning nowadays. It is a controversial option that could end up with you digging a big hole for yourself and dragging yourself down rather than scoring any extra points.

EXAMINER: What are the published results of hip arthroplasty in patients with AVN?

CANDIDATE: There are conflicting reports regarding the outcome of THA for AVN. Historically, THA had poorer results in AVN compared to osteoarthritis. Other studies have refuted this and report an equally successful outcome after THA. Suggested reasons to explain this observed discrepancy include pooling together of patients with different associated risk factors for AVN and that many reported studies used either first-generation cementing techniques or first-generation uncemented prosthetic designs. This contrasts with more current studies using contemporary prosthetic designs and modern cementing techniques. Patient age may also be an issue, with AVN typically affecting a young age, which means that the outcome of AVN can be expected to be suboptimal. Patients with sickle cell disease, renal failure and/or transplant and Gaucher disease have significantly higher revision rates.

Michael Mont has extensively studied AVN. In one paper he reviewed various studies and reported wide differences in failure rates of THA of between 10% and 50% at 5 years[27]. Furthermore, in his systematic review of 27 published series, all except two studies reported a higher rate of failure in patients with AVN than in age-matched patients with other disorders[28].

EXAMINER: Yes, you are forcing cement into dead bone. This gentleman is young, at 40 years of age. How will you manage him?

CANDIDATE: I would perform an uncemented THA on this patient with a ceramic on ceramic bearing surface telling him it will last between 15 and 20 years before requiring revision.

Although there has been a recent trend in performing more cemented THA as NJR data suggests across all ages better early survival rates at 11 years he is still only 40 years old and I would have concerns with long-term risks of loosening in a young active male and prefer to use an uncemented hip arthroplasty. Despite slightly better results from NJR data using an uncemented CoC bearing surface at 10 years for males under 55 yrs I have concerns regarding sqeaking, catastrophic ceramic fracture and cost and

[h] Whilst we hesitate to say it is a pass/fail issue the examiners may think you may miss this diagnosis in the clinic and be unsafe in your practice.

[i] If you had to chose or bet on one causative agent being probed go for steroids.

[j] We come across a number of candidates near to the exam in practice vivas who apart from knowing the name 'Ficat' do not know any classification systems used for ON.

therefore would opt to use a CoP bearing surface. I would want to make sure the polyethylene is a second generation highly cross linked variety. Newer manufacturing techniques involve either sequential irradiation and annealing (X3) or infusing Vitamin E into irradiated PE to stabilise free radicals and prevent oxidative degradation

Adult elective orthopaedics oral 2

A cut section of a femoral head showing subchondral collapse was presented (Figure 16.5). This is another popular lead picture into AVN hip.

The following were discussed:

Radiographic findings in AVN – Differentiating features between precollapse and collapse

The grading of AVN – 'YOUR' management for this grade

The role of fibula grafts, decompression

There was a very superficial discussion throughout

Adult and pathology oral 3

Osteonecrosis:

- Classification
- Causes
- Management

Basic science oral 1

- Causes of ON
- Classification
- Pathophysiology of ON, specifically steroid-induced AVN
- Discussion of some new theories and treatments proposed
- Management of ON

Basic science oral 3: AVN following fractured neck of femur: blood supply femoral head

What happens to the bone in AVN? Histological changes?

How does repair occur?

Inflammatory cascade. Fibrous vascular in growth in the regions of cell death. Primitive mesenchymal cells differentiate into osteoblasts and osteoclasts. Immature woven bone is deposited throughout the network of dead trabecular bone. The nonviable trabecular bone is slowly resorbed by the process of creeping substitution. Newly deposited bone does not attain the previous mechanical strength and structural integrity of the femoral head leading to subchondral collapse with weight-bearing.

Adult elective orthopaedics oral 4: ON

Femoral head with ON resected at surgery for THA (Figure 16.6).

- AVN (Ficat classification and management)
- Principles of classification.

The lack of a universally accepted classification system for AVN makes it difficult to compare and analyze data published from different centres. The Ficat classification system has a low interobserver reliability and only modest intraobserver reliability. The validity of a classification system reveals the accuracy with which it describes the true pathological process. Validity has not been formally assessed for any of the classification systems for osteonecrosis of the femoral head.

Adult and pathology oral 5

Clinical and MRI differences between AVN and bone marrow oedema syndrome (BMES):

- BMES presents with similar symptoms to ON with disabling hip pain without any history of trauma
- With BMES standard radiographs are normal or demonstrated non-specific osteopenia
- With MRI a heterogenous bone marrow oedema pattern would be seen in the affected femoral head, neck and trochanteric region. There is decreased signal intensity on T1-weighted images and increased signal on T2-weighted sequences
- The main differentiating feature from ON is the lack of focal defects or subchondral changes on T2 MRI images
- All patients with BMES recover completely over a period of 6–12 months without the need for surgical intervention. Treatment is usually symptomatic including

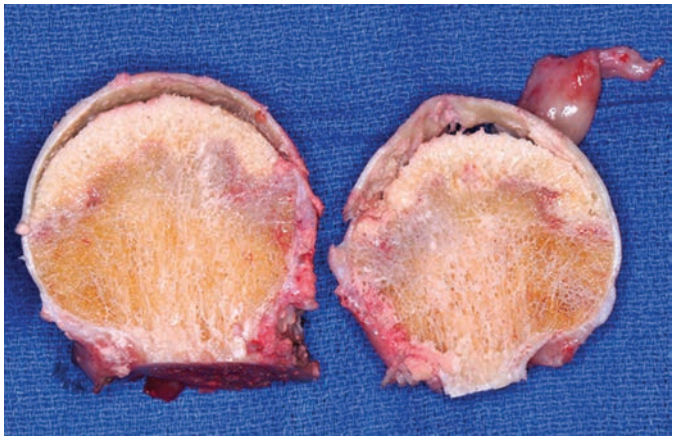

Figure 16.5 Cut section femoral head demonstrating ON with subchondral collapse

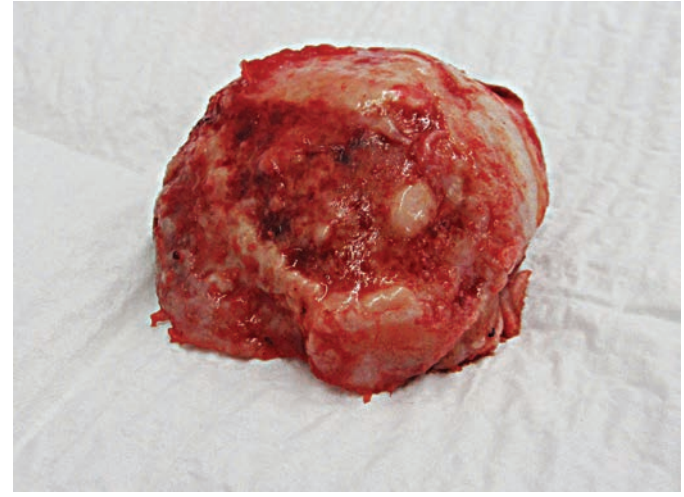

Figure 16.6 ON femoral head resected at surgery for THA

Table 16.6 Grading of protrusio acetabuli according to distance between the acetabular line and ilioischial line

Grade	Men	Women	
I: Mild	3–8 mm	6–11 mm	5–10 mm protrusion
II: Moderate	9–13 mm	12–17 mm	10–15 mm
III: Severe	>13 mm with fragmentation	>17 mm with fragmentation	>15 mm protrusion

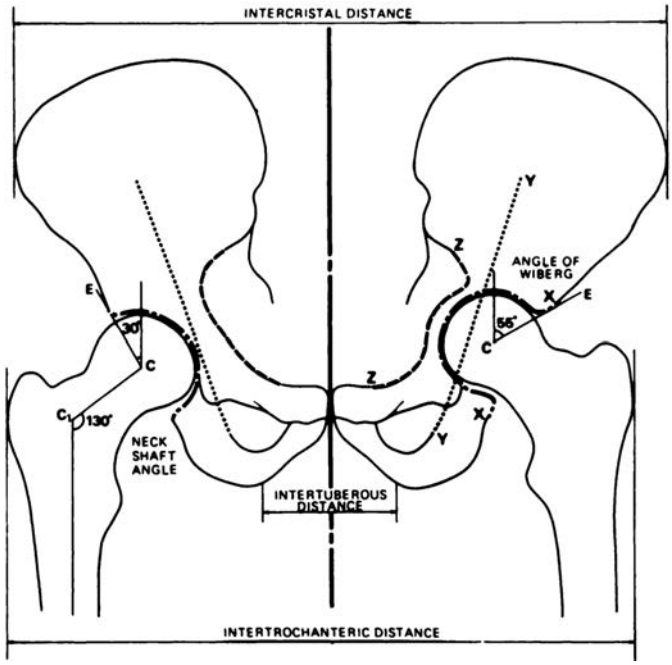

Figure 16.7 Radiographic view of the pelvis showing normal appearances on the left and those of protrusio acetabuli on the right. X–X, acetabular line, Y–Y, ilioischial line, Z–Z, iliopectineal line

analgesia and NSAIDs, physiotherapy and protected weight-bearing to maintain strength and mobility of the hip

COMMENT: This viva question is more challenging than the usual ON hip viva question. It is similar to the 2 for 1 supermarket offer in that the examiners can switch between the two topics. Although BMES is a rare disorder compared to ON, both conditions may not be completely distinct and separate entities but related to a common cause which remains unclear.

Trauma oral 1

Post-traumatic AVN (fractured neck of femur, managed with AO cannulated screws)

- Stage using ARCO (Association Research Circulation Osseous), Ficat
- Management options – Cemented or hybrid THA, possible role for arthrodesis if very young or at least the option discussed

Uncemented THA for AVN post-fractured neck of femur is perhaps not the best option as bone quality is likely to be osteoporotic, there may be issues with osseointegration, femoral stem sinkage, LLD and dislocation. A cemented THA is a safer choice.

Protrusio acetabuli

Definition

Acetabular protrusio is the proximal and medial migration of the femoral head through the medial acetabular wall into the pelvis[29]. It is defined radiographically as migration of the femoral head medial to Kohler's line[k] (a line from the lateral border of the obturator foramen to the medial border of the sciatic notch).

Classification Hirst grade I–III

Radiological classification based on plain radiographs of the pelvis. Armbuster et al.[30] found that in adult men the acetabular line is on average 2 mm lateral to the ilioischial line, but in women it is 1 mm medial. They considered protrusio to be present if the medial wall of the acetabulum is >3 mm or more medial to the ilioischial line in a male or >6 mm medial to it in a female (Figure 16.7 and Table 16.6).

Some authors use the centre-edge angle measurement to diagnose protrusion. An angle >40° is considered diagnostic. Others consider violation of the teardrop as a diagnostic criterion. The teardrop is the most consistent landmark and is a useful way to assess and track progression of protrusion. The acetabular roof angle is negative.

Aetiology[l]

The primary idiopathic form of protrusio is termed Otto's pelvis or disease (arthrokatadysis). It is more common in females (10 : 1), develops after puberty, involves both hips

[k] Also known as the ilioischial line.

[l] This is classic adult elective orthopaedic oral material. An AP radiograph of the pelvis demonstrating obvious bilateral protrusio is shown. After preliminary discussions concerning the grading/classification of protrusio the examiners follow on to ask about possible aetiological causes of protrusio. This generally leads on to technical difficulties of performing a THA. As soon as 'bone grafting' is mentioned, the oral will turn 90° and the examiner will start to discuss the principles of bone grafts. Some examiners consider protrusio as only the prop to lead into a discussion about bone grafting.

and progresses to osteoarthritis in mid-adult life. Clinical presentation includes pain and limitation of leg abduction, loss of hip extension (flexion contracture) and a hyperlordotic posture. Varus deformity of the femoral neck and arthritic changes are common. A degree of protrusio is present in approximately 5% of all cases coming to THA surgery, with 50% cases occurring in patients aged between 61 and 70 years. It has been reported as being present in approximately 22% of rheumatoid patients requiring arthroplasty. The deformity may progress until the femoral neck impinges on the side of the pelvis.

Associations[m]

Decreased bone density

- Osteoporosis
- Osteogenesis imperfecta
- Osteomalacia
- Rickets
- Rheumatoid disease (19%)
- Marfan's disease
- Ankylosing spondylitis

Normal density

- Osteoarthritis
- Otto's disease (idiopathic, 75%)

Increased density

- Hypophosphatasia
- Paget's disease (4%)

Aetiology of secondary protrusio acetabuli

Infective: Staphylococcus, streptococcus, *Mycobacterium tuberculosis*
Inflammatory: Rheumatoid arthritis, ankylosing spondylitis, psoriatic arthritis, Reiter's syndrome
Metabolic: Paget's disease, osteogenesis imperfecta, osteomalacia hyperparathyroidism
Genetic: Sickle cell disease, Marfan's syndrome Ehler–Danlos syndrome
Neoplastic: Neurofibromatosis, metastasis (breast, prostrate most common) radiation-induced osteonecrosis acetabulum
Trauma: Iatrogenic fracture during surgery acetabular, fractures, osteolysis following THA

Symptoms

Clinical features fall into three categories:

1. Symptoms due to the anatomical abnormality (deeping socket): stiffness
2. Symptoms due to secondary osteoarthritis: pain, limp, all movement becomes progressively painful and limited especially abduction as the trochanter starts impinging on the superior acetabular margin
3. Symptoms due to causative disease: rheumatoid arthritis, osteomalacia, etc

Investigations

Standard AP and lateral radiographs of the pelvis will confirm the diagnosis and will permit staging. Judet views or a CT scan will evaluate defects in the posterior and medial wall and help in planning placement of the acetabular cup. MR arthrogram to access the status of the articular cartilage if joint preservation surgery complicated. Exclude metabolic and rheumatological causes of the condition

Management

- Surgical triradiate cartilage closure combined with valgus intertrochanteric osteotomy (VITO) for skeletally immature patients
- Protrusio joint preservation surgery for adolescent and young adult patients[31]. In cases without cartilage degeneration, open surgical dislocation with osteochondroplasty of the acetabular rim and femoral neck. With early cartilage degeneration VITO, reverse periacetabular osteotomy (PAO) or combined reverse PAO and VITO. The latter leads to more cranial directed forces at the hip, reducing pressure on the floor of the acetabulum. In addition, it reduces impingement at the superior acetabular margin
- Management of symptomatic protrusio is THA with non-operative measures reserved for patients unfit or unwilling to undergo surgery. There is merit in delaying THA surgery in young individuals with minimal symptoms

Principles of THA reconstruction

- THA may be technically demanding due to associated significant medial and proximal migration of the joint centre, deficient bone medially and reduced bony support to the acetabular component peripherally
- Template preoperatively to avoid offset and leg length discrepancies. Placing the hip centre back into the correct anatomical position is essential to restore proper joint biomechanics and to lower reactive forces
- The medial wall of the acetabulum is typically thin, and does not usually need reaming. The general principle is to bone graft the floor and lateralize the cup
- Because of medial migration of the femur, the sciatic nerve is often nearer the joint than normal and should be identified early and protected
- Hip dislocation can be difficult due to the excessive depth of the acetabulum and medial displacement of the femoral head. Perform a controlled hip dislocation avoiding excessive force as this may result in fracture of the posterior wall of the acetabulum or proximal femur.

[m] Remember O^5R^2MAP.

Perform an extensive capsular incision. Consider in situ neck ostotomy. In severe cases a trochanteric osteotomy may be required for adequate exposure

- When the femoral head has protruded into the pelvis, an hourglass constriction is created, and the walls of the periphery of the acetabulum diverge. Peripheral reaming will create a new rim with convergent walls wide enough to support the acetabular component. It is important to achieve a good rim press fit (if using a cementless shell) as the thin or deficient medial wall is not relied on to prevent recurrent deformity. Femoral head bone autograft should be placed on the medial wall, especially if there are significant cavitatory and central segmental bony defects. Sections of femoral head can be used
- Current trend is to use cementless fixation. The shell is 1–2 mm larger in diameter than the last reamer and because of the presence of bone graft should have an excellent fit and is quite stable. The use of additional screws fixation is recommended
- If cementing, avoid excessive cement medially as it may be difficult to pressurize into the acetabulum. When a cup is inserted too medially and too high in moderate and severe protrusio, the neck will impinge against the acetabular rim, reducing the primary arc of motion, thus, causing instability and predisposing to loosening
- In cases with a severe deformity, a reconstruction cage may be required
- If coxa vara is present, standard femoral component position (approx. 1 cm above lesser trochanter) may result in limb length discrepancy (LLD). Careful preoperative templating and more distal femoral component placement is required to avoid limb lengthening

The standard treatment of mild protrusio with cement alone has been called into question. The thin medial wall protruded acetabulum is often osteoporotic so that adequate fixation by cement alone is uncertain. Cancellous bone grafting can provide a biological buttress after incorporation and also reduce the effect of thermal necrosis and osteolysis that may follow the exothermic curing of cement. One study reported better results in hips with moderate and severe protrusio reconstructed with bone graft than in mildly affected hips in which cement alone was used[32]. Reconstruction with bone graft enables a better anatomical positioning cup, and its use has been suggested in hips with only a minor protrusion.

The adequacy of correction deformity correlates with long-term prosthetic survivorship. Bayley et al.[33]. found loosening of 50% of acetabular components at long-term follow-up when the centre of rotation of the hip was not corrected to within 10 mm of the anatomical location. Loosening occurred in only 8% of acetabular components in which the hip centre was restored to within 10 mm of the anatomical.

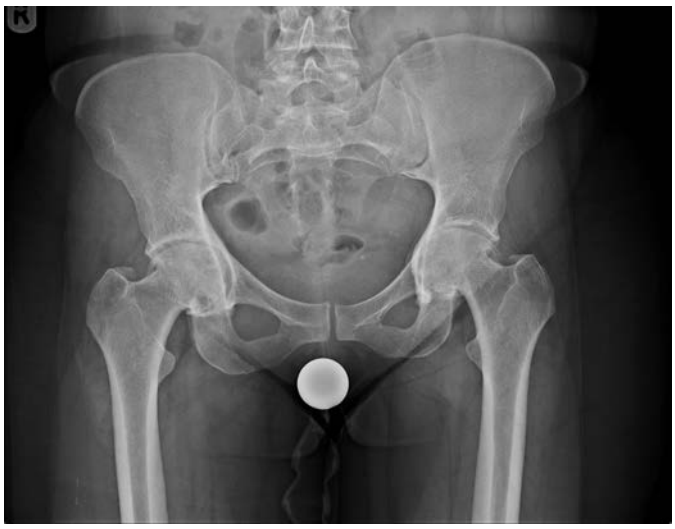

Figure 16.8 Radiograph of bilateral protrusio

Examination corner

Adult elective orthopaedics oral 1: Idiopathic protrusio (Otto's pelvis)

- Indications for surgery
- Approach
- Use of bone graft
- Cementing technique

Adult elective orthopaedics oral 2: Bilateral protrusio

Radiograph shown of bilateral protrusio (Figure 16.8).

EXAMINER: These are the radiographs of a 58-year-old woman who presented to the orthopaedic clinic complaining of bilateral hip pain (Figure 16.8). Would you like to comment on them?

CANDIDATE: This is an AP radiograph of the pelvis, which demonstrates a bilateral grade III Hirst protrusio.

EXAMINER: What is protrusio?

CANDIDATE: If the femoral head is medial to Kohler's line or the centre-edge angle is >40°, then protrusio is present.

EXAMINER: How do you grade protrusio?

CANDIDATE: Mild, moderate and severe based on the distance of the medial wall of the acetabulum to Kohler's line. Mild is 5–10 mm, 10–15 mm is moderate and >15 mm is severe.

EXAMINER: What are the causes of protrusio?

CANDIDATE: Protrusio can be classified as primary, attributed to incomplete or delayed triradiated cartilage ossification (chondrodystrophy), or secondary, attributed to diseases which weaken the medial acetabular wall, such as rheumatoid arthritis, ankylosing spondylitis, osteoarthritis, chronic renal osteodystrophy, osteoporosis, etc. The principle joint reaction force vector is directed more medially than normal with resultant medial migration of the hip centre.

EXAMINER: What are you going to do for the patient?

CANDIDATE: I would initially attempt conservative management but the deformity is quite marked and if symptoms of stiffness and pain were severe enough I would offer her an uncemented THA

with a ceramic-bearing surface and bone grafting to the medial wall.

EXAMINER: Any difficulties you may encounter at surgery?

CANDIDATE: The neck may need to be cut in situ as it may be difficult to dislocate and you could end up with a fracture if you are not careful. The medial wall of the acetabulum is usually thin or may be partially membranous, and should not be penetrated. I would use supplementary acetabular screws as the acetabular shell is not well supported by host bone medially. Failure to restore normal lateral offset may cause the greater trochanter to impinge off the anterior edge of the acetabulum, which may lead to posterior instability. A femoral stem design with increased offset reduces the risk of femoral-pelvic impingement especially if a short neck length is used to equalize leg length.

EXAMINER: What complications can occur?

CANDIDATE: Intraoperative complications include acetabular fracture, neurovascular injury, and visceral injury. Penetration of the medial wall may place intrapelvic structures such as the bladder, ureter, bowel and external iliac artery at risk. Sometimes the posterior soft tissue envelope of the capsule and external rotators will not reach the posterolateral trochanter for repair. The most common postoperative complications include loosening and medial migration of the acetabular component. Others include dislocation, infection and LLD.

EXAMINER: This is her postoperative (AP pelvic) radiograph following right THA (Figure 16.9). What are the results reported for THA in protrusio?

CANDIDATE: Baghdadi et al.[34] from the Mayo clinic in 2013 in *CORR* retrospectively reviewed 162 hips undergoing THA for acetabular protrusio. They reported survival from aseptic cup revision at 15 years of 89% for uncemented compared to 85% for cemented cups. The risk of aseptic cup revision significantly increased by 24% for every 1 mm medial or lateral distance away from the native hip center of rotation to the prosthetic head center.

Acetabular dysplasia in adults[n]

Acetabular dysplasia describes an underdeveloped or shallow, upwardly sloping acetabulum, which may occur with varying degrees of deformity of the proximal femur such as excessive femoral neck anteversion, coxa valga or femoral neck cam deformity[35]. It is an important cause of hip pain, often affecting young women. Symptoms may be experienced for many years before osteoarthritis develops. Patients describe a sharp activity related groin pain that increasingly affects their lifestyle. Symptoms may be exacerbated by rising from a seated position, climbing in or out of a car, going downstairs or sudden rotational movements.

[n] This is a different viva question to DDH. Interest in acetabular dysplasia in adults has increased as its surgical treatment has advanced and the understanding of young adult hip disorders has grown.

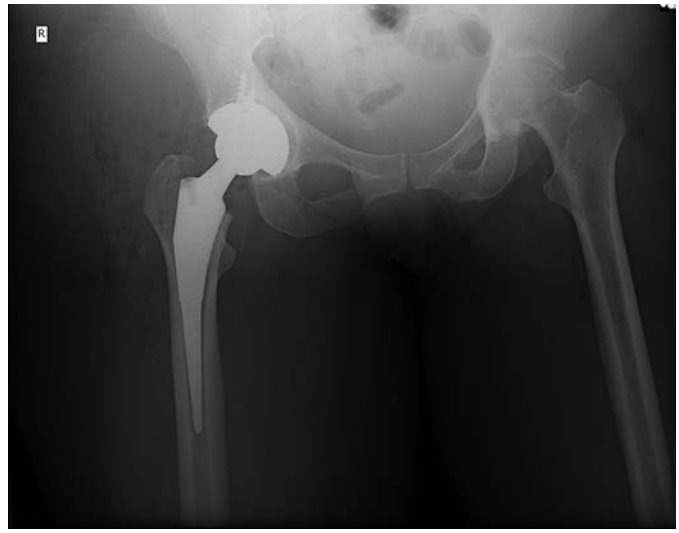

Figure 16.9 Postoperative (AP pelvic) radiograph following right uncemented THA

Imaging

A number of hip angles should be measured[36]:

1. **Lateral centre-edge angle of Wiberg (LCEA).** This assess the superolateral coverage of the femoral head or more simply femoral head lateralisation on an AP pelvis radiograph. It is obtained by measuring the angle between two lines: (a) a vertical line through the centre of the femoral head; and (b) a line from the centre of the femoral head to the superolateral aspect of the acetabular sourcil. An angle $<20°$ is diagnostic of dysplasia whereas values above 40° may indicate pincer femoroacetabular impingement (FAI).

2. **Anterior centre edge angle of Lequesne (ACEA).** This measures anterior dysplasia on the false profile radiographic view that provides a true lateral view of the acetabulum. It is the angle between two lines: (a) a vertical line through the centre of the femoral head; and (b) a line from the centre of the femoral head to the most anterior point of the acetabulum. It is a measure of anterior coverage of the femoral head. An angle $<20°$ is suggestive of anterior subluxation or deficiency.

3. **Tönnis angle (acetabular inclination).** This measures the angle of the weight-bearing surface or sourcil. It is the angle between two lines: (a) a line from the most inferior point of the acetabular sourcil to the lateral margin of the acetabular sourcil; and (b) a horizontal line running through the most inferior part of the sourcil.

A normal Tönnis angle is between 0° and 10°. A decreased Tönnis angle can lead to a pincer form of FAI whilst an increased Tönnis angle may indicate structural instability.

4. **Acetabular angle (of Sharp).** This measures acetabular inclination or opening. It measures the intersection between the following two lines: (a) a horizontal line from the inferior aspect of one teardrop to the other; and (b) a line from the inferior aspect of the teardrop to the superolateral margin of

the acetabulum. A normal angle is between 33° and 38°. Higher angles imply dysplasia in adults.

Investigations

CT scan. This will identify early degenerative changes, cysts and acetabular/femoral version. Three-dimensional CT reconstructions can be useful in identifying cam on the anterior femoral neck.

MRI arthrogram. To detect chondral/labral pathology and extracapsular abnormalities such as avascular necrosis.

Management

Diagnostic local anaesthetic hip injection with examination under anaesthesia (EUA) to confirm the intra-articular origin of pain

Hip arthroscopy for management of chondro/labral pathology and excision of a cam impingement lesion. Occasionally when hip arthroscopy is used to treat labral pathology this may lead to a worsening of symptoms, as the stabilising effect of the labrum may be lost.

The mainstay of surgical management is the Ganz periacetabular osteotomy (PAO). The acetabulum is reoriented to enhance coverage of the femoral head. The aim of surgery is to achieve congruity, stabilise the hip joint, medialize the hip joint center and to reduce contact pressures. This will relieve pain, improve function and prevent further overload of the labrum, cartilage and soft tissues, thereby delaying the onset of osteoarthritis.

Advantages include posterior column remains intact leaving the pelvis stable and allowing immediate partial weight-bearing, minimal internal fixation, extensive mobilization of the acetabular fragment is possible, the blood supply of the acetabulum is unaffected and the dimensions of the true pelvis are maintained.

Indications include:

- Symptomatic acetabular dysplasia with persistent pain
- A centre edge angle of <25°
- A congruent hip joint
- Maintained range of motion with hip flexion>110°
- Preoperative osteoarthritis corresponding to Tonnis grades 0–1

Congenital hip dislocation/subluxation

The hallmark of the dysplastic hip is lack of coverage of the femoral head, whether it is subluxed or dislocated.

Anatomical features

Acetabulum

- Shallow, anteverted, deficient anteromedial wall, small and poor bone quality

Femur

- Small deformed head, short anteverted valgus neck, small and posteriorly displaced greater trochanter and narrow, straight tapered femoral canal. Decreased neck/shaft angle

Soft tissues

- Hamstrings, adductors and rectus femoris muscles are usually shortened and contracted. Abductor muscles horizontal and the hip capsule is elongated and redundant. Psoas tendon hypertrophied. Sciatic nerve shortened susceptible to stretching. The femoral nerve and artery are forced laterally by a high riding femur and are more vulnerable to injury

Classification

Crowe I–IV

Crowe classified dysplasia radiographically into four categories based on the proximal migration of the femoral head (Table 16.7 and Figure 16.10). This classification system is simple to use, reliable, reproducible and relates to the outcome of THA in patients with dysplasia. Although no measure of reliability was included in the original article later studies have documented high levels of interobserver and intraobserver reliability.

Hartofilakidis 1–3

Some surgeons prefer the Hartofilakidis' classification (Table 16.8 and Figure 16.11) system, as they believe it is more practical and simpler to use and may predict the clinical outcome of THA more accurately, since it yields a more precise description of the acetabular pathology.

Management

Conservative

There is certainly a role for conservative treatment of the older patient with neglected bilateral developmental dysplasia of the hip (DDH) who has minimum hip pain. Many patients

Table 16.7 Crowe classification of acetabular dysplasia

Grade I	<50% subluxation or proximal dislocation <10% or 0.1 of pelvic height
Grade II	Subluxation between 50% and 75% or proximal dislocation of 10–15% or 0.1–0.15 of pelvic height Usually do not have leg length inequality or loss of bone stock
Grade III	Subluxation between 75% and 100% or proximal dislocation of 15–20% or 0.2 of pelvic height Complete loss of superior acetabular roof Possibly thin medial wall Anterior and posterior columns are intact
Grade IV	Dislocated or proximal dislocation of >20% or 0.2 of pelvic height True acetabulum is deficient but remains recognisable

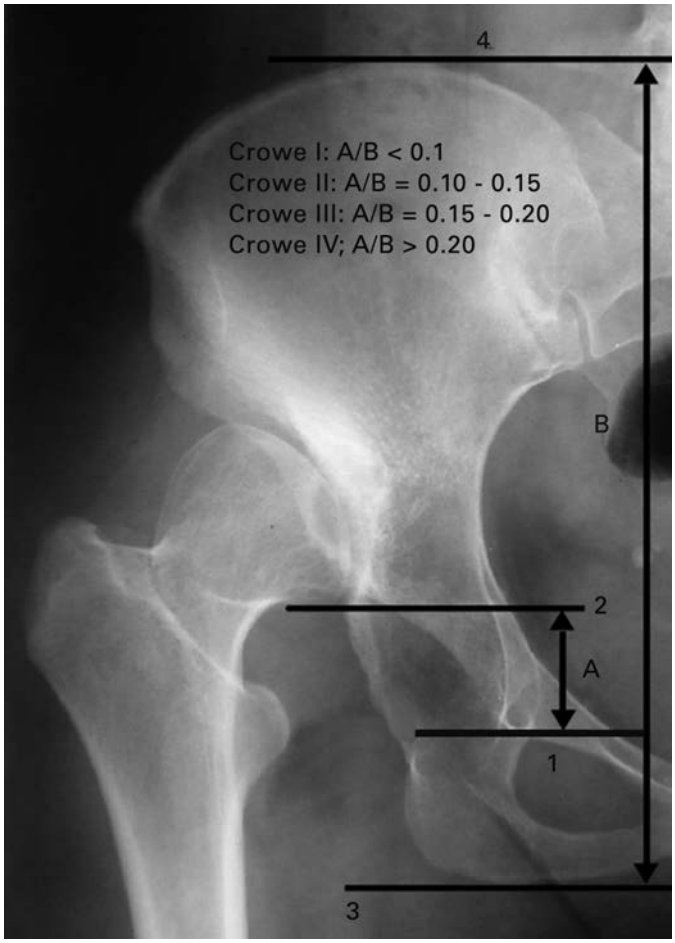

Figure 16.10 Radiograph showing measurements for the Crowe classification system (A, vertical distance between the reference interteardrop line (line 1) and the head-neck junction (line 2); B, vertical distance between the line connecting the ischial tuberosities (line 3) and the line connecting the iliac crests (line 4)

with this deformity function well until later life. Unilateral disease particularly in a young patient is more problematic to treat conservatively even with minimal hip pain. Difficulties with LLD and low back pain (LBP) can tip the balance towards surgery.

Consider shoe-raises, simple analgesics and steroid injections.

Arthroscopic hip debridement

Especially if labral tears are present. However, it is important to point out that this may cause secondary instability due to the lack of bony coverage.

Realignment osteotomy

This is possibly indicated for a young adult in their early 20s. A periacetabular osteotomy is usually performed. This offers a three-dimensional correction and improves acetabular depth, which would make a future THA technically easier, unlike a proximal femoral osteotomy.

Table 16.8 Hartofilakidis' classification

Type 1	Dysplasia in which the femoral head is still contained within the true acetabulum
Type 2	Low dislocation in which the femoral head articulates with a false acetabulum, the inferior lip of which is in contact with or overlaps the true acetabulum
Type 3	High dislocation. The false acetabulum has no contact with the true acetabulum. The femoral head is completely out of the true acetabulum and migrated superiorly and posteriorly to a varying degree

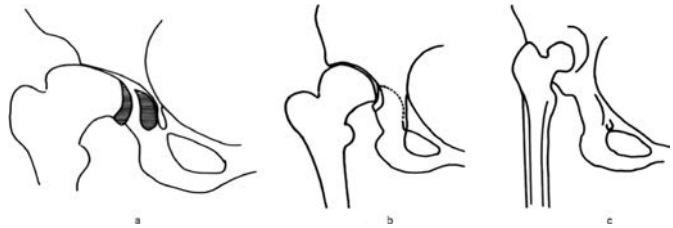

Figure 16.11 Hartofilakidis' classification. Diagram of the three main types of congenital hip disease in adults showing (a) dysplasia, (b) low dislocation and (c) high dislocation

Arthroplasty

End-stage arthritis is generally treated with THA.

Arthrodesis

If unilateral disease is present, arthrodesis for end-stage arthritis is certainly an option worth considering in a young patient with high activity levels. However, it is difficult to convince these patients to undergo arthrodesis with the known excellent early results of THA.

Total hip arthroplasty for DDH

Surgical approach

The surgical approach must allow for exposure and identification of the true and false acetabulum, identification of the sciatic nerve and lengthening of the leg. A conventional posterior or lateral approach may be adequate for mild dysplasia whilst in more severe cases a trochanteric osteotomy may be needed (there is a risk of postoperative trochanteric migration or non-union). Whatever surgical approach is used, the sciatic nerve should be identified, particularly where previous surgery has been carried out as the nerve may be densely adherent to the site of a previous femoral osteotomy. Some surgeons would debate this point, particularly if the degree of lengthening were <2 cm.

Leg length discrepancy

If the leg has been lengthened too much, the femur is usually shortened by a subtrochanteric osteotomy. Shortening by resecting more proximal femoral bone avoids the problems associated with an osteotomy but involves resection of metaphyseal bone that is needed for stabilisation of the uncemented implant and osseous ingrowth.

Placement and coverage of the cup

Anatomical position (low hip centre of rotation)

- Advantages: Facilitates lengthening, better hip function, best available bone stock, diminishes joint reaction forces
- Disadvantages: Difficult surgery, a femoral shortening procedure may be necessary

Non-anatomical position (high hip centre of rotation)

A hip centre located at least 35 mm proximal to the interteardrop line.

- Advantages: Technically easier than the anatomical position; allows the component to be more completely covered by native bone and so may avoid the need for bone grafting and also decreases the need for a concomitant shortening femoral osteotomy
- Disadvantages: Increased shearing forces may lead to early loosening, a higher rate of dislocation than the anatomical location, further revision surgery is difficult as bone stock is not restored, affords a limited amount of leg lengthening, can only use a very small acetabular component with a thin polyethylene cup as the ceramic bearing surface is often not possible because a bigger acetabular shell would be needed

Acetabular coverage

When there is a large segmental defect in the superior wall of the acetabulum, consider using a bulk femoral head autograft held with screws to restore superior coverage of the acetabular component. The main long-term concern with bulk femoral head allografts is loosening of the socket, with variable rates from 0% to 25% reported in the literature. Another option would be to use tantalum augments.

Adequate acetabular cup coverage is required (at least 70%, medialize if necessary down to the inner table) to prevent early loosening. Avoid reaming any more of the superior roof than necessary and lateralisation of the acetabular component.

Intentional controlled fracture of the medial wall (cotyloplasty) to allow for medial advancement of the socket is not universally accepted. There is concern regarding long-term problems of protrusio, cup loosening and failure to restore bone stock making revision surgery difficult.

A shallow dysplastic acetabulum may only accept a small size acetabular component. Small head size is needed to preserve adequate PE thickness but increased dislocation risk. Consider an alternative bearing surface such as highly cross-linked PE, CoC.

The use of an oblong socket has been suggested to restore the hip centre without the use of structural graft. The oblong part of the cup fills in the deficient segment and allows further stabilisation with screws. There is concern regarding the failure to restore bone stock with this device.

Insertion of the cup at the correct level with cement to fill the superior defect and without bone grafting has been associated with poor long-term results.[15] Bone grafting allows the cup to be placed in the correct anatomical position, provides bone stock for future revision surgery and restores leg length.

Linde et al.[16] found that the incidence of loosening with the cup in the true acetabular position was 13% at 15 years compared to 42% loosening at 15 years if placed more proximally.

Technical considerations of the femur in DDH

A narrow femoral canal may make femoral reaming difficult.

Marked anteversion of the femoral neck may be misleading and make component positioning difficult (there is a tendency for anteversion).

Derotation with subtrochanteric osteotomy may be necessary to place the component in the proper orientation (consider if anteversion $>40°$). Another option would be to use modular femoral stem systems that allow to dial-in the desired version (S-ROM®) or the use of custom-made femoral stems, which are also able to address the excessive anatomical anteversion.

If the greater trochanter impinges, it may require osteotomy and lateral displacement. There is a possibility of iatrogenic deformity of the proximal femoral shape from previous osteotomies. Retained metalwork can be extremely difficult to find and remove, and removal creates stress risers. Uncemented fixation with optimal fit and fill of the canal, initial stability and adequate bone ingrowth are not easily achieved in a narrow femoral canal with a thin cortex.

Subtrochanteric shortening

This is indicated when there are several centimetres of shortening or if derotation osteotomy is required. It is performed as an oblique or step cut osteotomy. A CT of the pelvis and lower limbs is essential to measure the femora accurately and, thus, the true LLD. Advantages include preservation of the metaphyseal femoral region (provides most rotational stability of the implant) and allowing concomitant correction of angular and anteversion deformities. It is technically difficult and there is a risk of non-union. Osteotomy can be secured with vascularized onlay autograft fixed with one or two cerclage cables or locking plate fixation.

Complications--Increased risk of complications including nerve injury, vascular injury, deep infection, dislocation and LLD.

Results--Krych[37] reported on a series of 28 Crowe IV hips managed with uncemented THA with subtrochanteric shortening. There was an improved HHS from 43 points to 89 points. Twelve (43%) of 28 hips had an early or late complication or re-operation: 2 subtrochanteric non-unions, 1 femoral component loosening, 1 acetabular component loosening, 1 liner disengagement and 4 hip dislocations.

Examination corner[o]

This can be either an intermediate case or an elective adult orthopaedic oral topic[p]. Once the preliminaries of the radiographic description of the condition and Crowe's classification are out of the way, discussion will turn to management. A large part of the discussion will probably centre on the technical issues in performing a THA in this type of hip.

Adult elective orthopaedics oral 1: Young arthritic patient with DDH
Radiograph of a 23-year-old woman complaining of severe arthritic left hip pain secondary to DDH.

- Discuss management options including the role of pelvic osteotomy?

Adult elective orthopaedics oral 2: Painful THA
Patient who had a right THA at 30 years of age for DDH. THA has now failed.

- Discuss the management.

This question is a double take of both the principles of revision hip surgery and difficulties with DDH surgery. The patient is only 30 and so may require repeated revisions in the future.

Adult elective orthopaedics oral 3: Unilateral DDH
Radiograph shown of a 43-year-old woman with a deformed arthritic left hip secondary to DDH (Figure 16.12).

- Outline your management of this hip?
- How do you preoperatively plan for DDH surgery?

Preop work up would include a full history and clinical examination. Investigations would include AP pelvis AP and lateral radiographs left hip and CT scan.

On the acetabulum side the position of the true acetabulum should be identified and a decision made whether to restore the acetabulum to its true position or not. The degree of anteversion of the acetabulum should be defined as well as the adequacy of bone stock for satisfactory cup fixation and coverage.

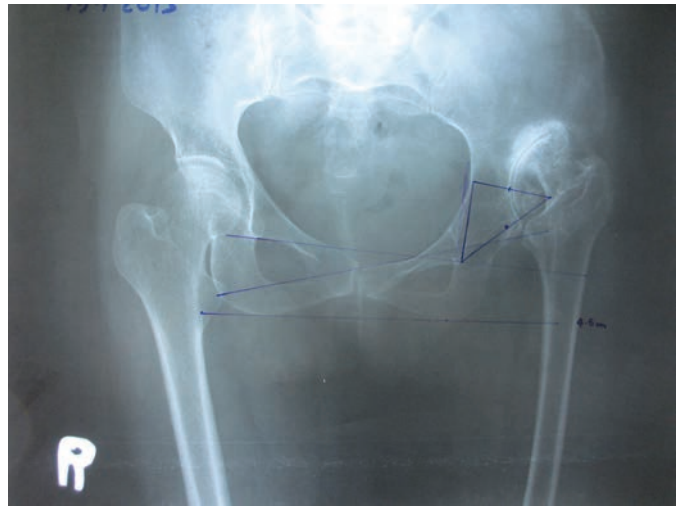

Figure 16.12 AP pelvis radiograph left Crowe 3 hip

Preoperative planning would also include an estimation of the acetabular component size, the preferred method of fixation (cement/uncemented) and need for bone graft. Cemented acetabular reconstruction has fallen out of favour because of reported revision rates up to 37%. Uncemented cups are generally preferred as they are more versatile

On the femoral side the size of the femoral canal and the need for special or custom implants should be assessed.

The need for femoral shortening should be made preoperatively. If there is any doubt subtrochanteric shortening should be performed but it increases the surgical complexity and potential for complications. Up to a maximum of 4 cm lengthening without shortening can be performed in THA but this involves complicated soft-tissue releases. More experienced surgeons are likely to go to subtrochanteric shortening earlier.

The method and amount of femoral shortening needs to be worked out beforehand. Ideally leg lengths should be equalized postoperatively unless there is a bilateral deformity and future surgery is planned for the opposite side.

Preoperative planning should also include the surgical approach to be used, solutions to deal with the hypoplastic acetabulum and femur, management of LLD and restoration of abductor function.

Radiograph shown post-THA surgery (Figure 16.13). Subtrochanteric shortening had been performed for Crowe 3 dysplasia. Preferable to avoid subtrochanteric shortening if possible but facilitates reduction, helps equalize limb length and protects the sciatic nerve. This is usually required with Crowe 3 and definitely with Crowe 4 hips.

In depth discussion of how to calculate the amount of LLD and how much shortening needed.

Although the femur is shortened during the operation, the distalisation of the femoral head to sit in the true acetabulum often results in an overall leg lengthening. The transverse subtrochanteric osteotomy allows shortening and derotation to be performed. When required, osteotomies can be re-cut which compares favourably to Chevron and step cut

[o] Additional information on the website www.postgraduateorthopaedics.com.

[p] This is quite a popular viva topic. There is a lot to talk about and the viva can progress in many different directions depending on how well a candidate is answering the questions.

osteotomies which require careful preoperative templating and planning since they are less amenable to alteration.

Adult elective orthopaedics oral 4: Bilateral Crowe 4 DDH
Radiographs shown of bilateral Crowe 4 DDH (Figure 16.14).

- Outline your management of this patient?

Discussion of the technical details of performing THA
Adult elective orthopaedics oral 4: Bilateral Crowe 4 DDH
With figure 16.14b there was a long discussion on how you would know the sex of the patient. Female as intrauterine contraceptive device seen on AP radiograph. Not quite a pass/fail question but not far away from this. We are unsure why this much importance was placed on this particular point

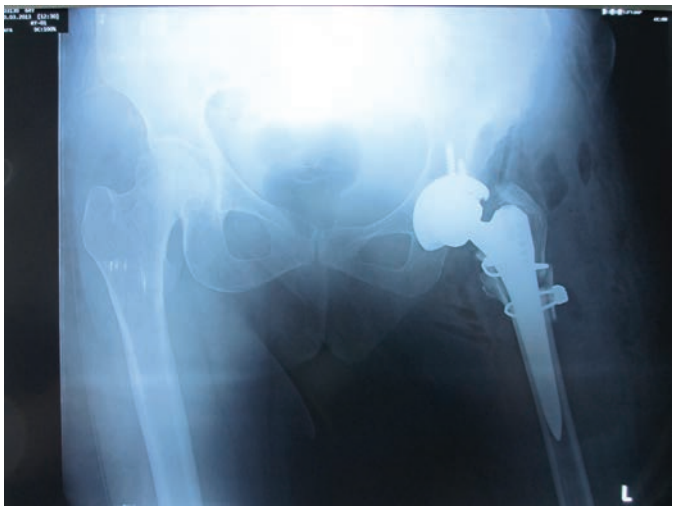

Figure 16.13 Postoperative AP pelvis radiograph left Crowe 3 hip with uncemented THA and subtrochanteric shortening

Primary THA

Indications

Disabling hip pain refractory to conservative management, severely affecting the patient's quality of life.

Contraindications

Absolute

- Active infection (local or distant)

Relative

- Neuropathic hip
- Progressive neurological disease
- Systemic co-morbidity factors
- Inadequate vascularity
- Psychiatric illness
- Non-ambulators
- Severe abductor muscle loss
- Obesity may also be a relative contraindication to THA although this is controversial. Several studies have shown a higher anaesthetic risk and operative complications, including serious cardiovascular and respiratory events, venous thromboembolic disease, infection, component mal-alignment, longer hospital stay and poorer functional outcomes and poorer 5-year survival. Additionally, obese patients tend to have more co-morbidity factors such as diabetes, hypertension, etc. Other studies have supported THA in obese patients.
  The best exam answer would be to say that obesity itself is not a contraindication to surgery:

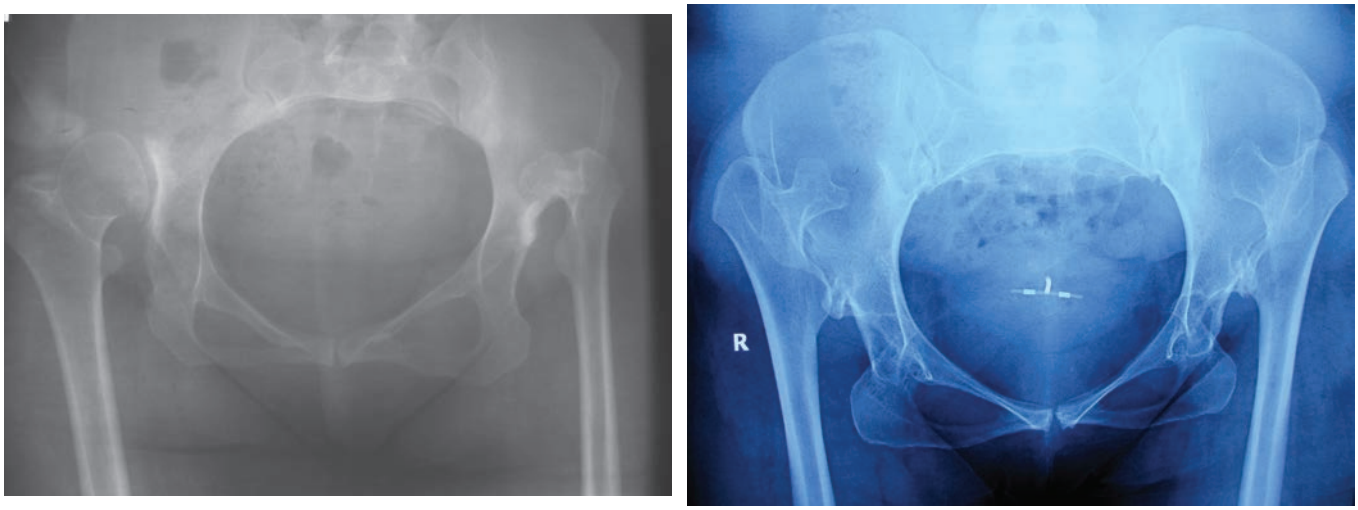

Figure 16.14 AP pelvis radiographs bilateral Crowe 4 hips

'I would ask the patient to attempt to lose weight, make them aware of the increased risks, associated complications and poorer outcomes and let theatre know so that they can arrange for a large orthopaedic table to be in place as well as deep Charnley retractors and a strong assistant. If the patient was morbidly obese I would ask one of my hip surgeon colleagues to review the case, as it might not be a case I would want to take on myself.'

Technical goals THA surgery

- Obtain correct offset
- Equalize leg length
- Restore the centre of hip rotation
- Correct positioning of implants in the patients primary arc range

Complications[q]

Local risks

- Dislocation (2–3%)
- Infection (0.5% osteoarthritis, 1% rheumatoid arthritis)
- LLD (15%)
- Nerve injury 1–2% primary THA, 3–4% revision THA, 5–6% THA for DDH. Possible causes include overlengthening, compression from haematoma, extruded cement or acetabular screw laceration
- Vascular injury
- Aseptic loosening (10% at 15 years)
- Periprosthetic fracture

Systemic risks

- Death (<0.5%)
- Deep vein thrombosis (2%)
- Non-fatal pulmonary embolism
- Fatal pulmonary embolism
- Cerebrovascular accident (CVA) (0.2%). Perioperative CVA greatly increases the risk of in-hospital mortality or discharge to a medical or chronic-care facility (as opposed to home) and increases the duration of hospital stay. Risk factors include advanced age, history of CVA, coronary artery disease, atherosclerotic disease and atrial fibrillation
- Myocardial infarction
- Urinary tract and chest infection

Informed consent

Discuss in terms of the

- Goals of surgery
- Reported success rate of THA
- Alternatives to surgery
- The buzz sentence is that "It is a shared care decision to procedure to surgery with the patient"

[q] Discuss in terms of local and systemic risks and also common, less common and rare complications.

Surgical approach

Trochanteric osteotomy (Charnley approach)

NJR data (10th Annual report) confirms used much less these days for primary THA (1% 2012)[r]

Advantages

- Hip easy to dislocate
- Excellent acetabular and proximal femur exposure
- Cement easy to insert
- Better femoral component alignment
- Useful in revision hip surgery

Disadvantages

- Increased blood loss
- Increased operating time
- Technically difficult to reattach trochanter
- Possibility of trochanteric non-union or wire breakage
- Trochanteric bursitis and non-specific trochanteric hip pain
- Technically challenging approach for inexperienced surgeon

Hardinge direct lateral approach

NJR data 2012 reports use in 40% cases for primary THA.

Advantages

- Compared with the posterior approach; decreased rates of dislocation and sciatic nerve injury with preservation of posterior soft tissues
- Familiarity as trainees mainly use this approach for hip hemi-arthroplasty for fractured NOF

Disadvantages

- Possible superior gluteal nerve injury if the gluteus medius division is extended >5 cm above the greater trochanter
- Damage to abductor musculature leading to a Trendelenburg limp post surgery
- Increased risk of heterotopic ossification
- Limited acetabular exposure
- Unsuitable if a large amount of femoral lengthening necessary
- Inability to adjust trochanteric tension
- Some concern regarding the security of the reattachment of the abductor muscles
- Tendency to insert femoral component within the femoral canal angled from anteriorly to posteriorly

[r] Opponents of the Charnley approach regard it as old fashioned, too invasive and there are better approaches to use. It is still used in Wrightington and other some specialist hip units but less and less these days.

Posterior approach

2012 NJR data confirms increase in use for primary THA, 54% cases.

Advantages

- Preservation of abductor muscles/function
- Avoids the complications of trochanteric osteotomy
- Lower incidence of heterotopic ossification
- Easy exposure, faster rehabilitation, diminished operating time compared to the Hardinge approach for primary THA
- Ability to deal with associated pathology, e.g. posterior column acetabular plating.

Disadvantages

- Increased risk of posterior dislocation
- Increased risk of sciatic nerve injury
- Increased risk of infection

Anterolateral (Watson Jones) approach

This was originally described for open reduction with internal fixation (ORIF) of femoral neck fractures. It exposes the interval between gluteus medius and tensor fascia lata (both of which are supplied by the superior gluteal nerve). Rarely used for THA as often requires additional division of gluteus medius and minimus that lie over the anterior capsule for adequate exposure which may lead to a Trendelenburg gait.

Disadvantages

- Exposure of the acetabulum depends on heavy retraction of the soft tissues and can be associated with damage to the femoral vein, artery and nerve
- Exposure is difficult in obese or very muscular patients
- Access to the femur is restricted and possible only with strong lateral rotation, adduction and flexion so that orientation of the femoral component may be difficult

Anterior (Smith–Peterson) approach

Exploits the plane between between sartorius (femoral nerve) and tensor fascia lata (superior gluteal nerve) superficially and gluteus medius (superior gluteal nerve) and rectus femoris (femoral nerve) deeply.

Mainly used in paediatric cases for open reduction DDH and washout of septic joint.

Modified approach can be used for THA

MIS variation of this exposure for use in THA has gained popularity in recent years but technically difficult and steep learning curve.

Advantages

- True internervous plane
- Avoids cutting the abductor muscles
- Good exposure of the acetabulum
- Lower incidence of heterotopic ossification
- Faster rehabilitation and decreased hospital stay

Disadvantages

- May be difficult to access the femur especially in obese patients and those with wide pelvises
- Risk of injury of the cutaneous nerve of the thigh

Technical tips for primary THA

Acetabular preparation

Failure to ream up to the true acetabular floor will have three negative effects:

- Lateralisation of the acetabular cup (increasing joint reaction forces)
- Uncoverage of the superior acetabular cup or inappropriate abduction of the cup to achieve coverage
- Positioning of the cup in an area of suboptimal vascularity

Avoid excessive cup medialisation. Decreasing the offset by >1 cm will weaken the abductors, increase joint reaction forces and may lead to THA instability. Consider using extended offset stems and lateralized liners when appropriate.

Cup orientation

Generally accepted values are 30–50° acetabular abduction and acetabular anteversion of between 0° and 30°. Charnley recommended 45° abduction and 0° anteversion. Lewinnek et al.[38] recommended a safe zone of 40 ± 10° abduction and 15 ± 10° anteversion. Implants outside this range are four times more likely to dislocate.

Classic reference

Lewinnek GE, Lewis JL, Tarr R, Compere CL, Zimmerman JR. Dislocations after total hip-replacement arthroplasties *J Bone Joint Surg Am*. 1978;60:217–220.

Lewinnek et al. report a 3% dislocation rate in a series of 300 THAs. Anterior dislocations were associated with increased acetabular component anteversion. The authors describe a safe range (5–25° anteversion and 30–50° abduction) to position the cup. The dislocation rate for implants outside this range was four times higher than for those within the range (6.0% vs 1.5%). Significant factors affecting dislocation included acetabular component orientation, surgeon experience and a history of previous surgery. Despite being a highly cited article the study is significantly limited by flawed methodology that weakens the study's conclusions.

Femoral offset

This is the perpendicular distance between the long axis of the femur and the centre of rotation of the femoral head. Increased offset:

- Increases the range of motion
- Decreases the incidence of impingement

- Increases stability by improving soft-tissue tension
- Too small an offset will reduce the movement arm of the hip abductors and cause a limp. Too large an offset will result in an increased bending movement arm during weight-bearing, which produces increased stresses within the stem that may lead to stem fracture or femoral loosening

Examination corner

Basic science oral 1

Several femoral prostheses were set out on the table:

- Discussion of uncemented femoral stems
- Methods of porous coating of the stem
- What is stress shielding in the context of THA?

Loss of proximal femoral bone density as a result of the load bypassing the area. According to Wolff's law, bone remodels according to the load it's subjected to; hence, the loss of density. A fundamental principle of solid mechanics is when two materials are joined; the stiffer material or structure bears the majority of the load.

- What type of uncemented stems are associated with this?

Fully porous coated stems as most of the load goes through the stem. Distal bone loading as more of the mechanical load bypasses the proximal femur

- What factors affect stem stiffness?

Stem stiffness approximates to radius^4, Co–Cr alloy is stiffer than titanium, solid and round stems are stiffer. Hollow, slots, flutes and a taper design reduce stiffness

- What is Hoek's law?

When two adjacent springs are loaded, load passes through the stiffer spring (the stem) bypassing the spring that is less stiff (femur).

Basic science oral 2

- Comparison of the biomechanics of the Charnley and Exeter THA
- Loaded taper vs composite beam biomechanics

Laminated clinical photograph shown of an Exeter and Charnley femoral stem

EXAMINER: What are these components?

CANDIDATE: This is an Exeter and a Charnley femoral stem. Both are cemented stems but have a different design philosophy. *(Model answer is given in the applied basic science chapter, Chapter 31)*

EXAMINER: What are the controversies surrounding the choice of cemented and uncemented implants with NJR and NICE guidelines?

CANDIDATE: The NICE-issued guidance suggesting minimum follow up of 10 years, just 7% of uncemented acetabular components in the NJR had the top ODEP rating of 10A, meaning strong 10-year data to support its use. This compared with 44% of the cemented acetabular components that had the top rating. The difference was less marked for cemented and uncemented femoral components with a 10A rating being used in 83% of cemented and 76% of uncemented femoral components.

Basic science oral 3

Discussion of metal-backed cups:

- Wear
- Creep
- Osteolysis
- Fatigue failure

Adult and pathology oral 1

- History of THA
- Judet hip – Manufacture and mode of failure
- Bone cement

Basic science oral 4

- Cementless femoral stems: Bone ingrowth, surface patterning, coatings, etc
- There are two basic methods of biological fixation either a porous coated metallic surface or a grit-blasted surface
- Ingrowth occurs when bone grows inside a porous surface. On-growth occurs when bone grows onto a roughened surface. The surface characteristics of an implant determine which occurs

EXAMINER: What type of THA would you use?

CANDIDATE: I would use a _____ cemented femoral stem because:

- Good long-term, peer-reviewed follow-up results have been published (probably the most important reason for using it and should be stated first)
- I am familiar with the instruments and find them easy to use
- Most of my training has been with the _____ hip

Evidence from the National Joint Registry (NJR) of England and Wales supports the use of an all cemented THA with a metal on polyethylene (MoP) bearing surface. An overall revision rate of 3.51 (3.31–3.72) at 11 years was reported with the 12th annual report.

Then go on and talk about the design features of you first-choice hip.

Complications of THA

Infection

- Overall in UK ~1%

Dislocation

- Incidence ~2–3%

Limb length discrepancy

Incidence 1–27%(~15%). Mean LLD ranges reported in literature from 2.8–11.6mm. LLD discrepancy perceived in >1/3rd patients with a 1cm difference following THA. Possible effects:

- Patient dissatisfaction with potential for litigation
- Short leg limp
- Vaulting type gait pattern
- Low back pain

- Groin pain
- Need for shoe raise

Nerve injuries

Incidence of sciatic nerve injury:

- 0.7–3% in primary THA
- 2.9–7.5% in revision THA

Risk factors include:

- Revision procedures (3–8%)
- Female gender (less soft-tissue mass)
- THA for DDH (5.8%)
- Post-traumatic osteoarthritis
- Posterior approach to the hip
- Cementless femoral fixation (mechanism unclear)
- >4 cm lengthening of the extremity

Femoral nerve injury:

- 0.04%–0.4% in primary THA

Consider MRI to look for gluteal haematoma. Haematoma can damage the nerve either directly (increased pressure on the nerve) or indirectly (ischaemia as a result of vasa vasorum compression). Get urgent post op radiographs to look for excessive leg lengthening or component mal-alignment. With an uncemented cup if significant acetabular screw penetration assess with an urgent CT scan or Judet view and consider urgent re-exploration of the hip. Check coagulation.

Re-explore a hip if there is sciatic nerve palsy, especially if it is progressive, painful (ongoing compression) and there is evidence of haematoma or a strong suspicion of direct injury (transected or ligated with a poorly placed suture). If there is documented leg lengthening then re-explore and carry out limb shortening by modular prosthetic head replacement. More than 90% of clinically evident nerve injuries involve the sciatic nerve, with approximately 50% of these involving the peroneal division only. Nearly 80% of all injuries will have an incomplete recovery. EMG studies may be used to assess the level of the lesion and monitor recovery but do not show any immediate abnormality.

Aseptic loosening

This is the most serious long-term problem with THA.

Haemorrhage and haematomas

Common urgent sources of venous and arterial bleeding include branches of obturator and femoral vessels, medial circumflex vessels, and inferior and superior gluteal vessels.

Vascular injuries

These are extremely rare (0.2–0.3%). Most vascular injuries occur during revision surgery because of distorted anatomy and scarring. The femoral vessels are primarily at risk from retraction and dissection over the front of the acetabulum. Penetration of the medial wall of the of acetabulum may injure the common iliac artery or the superficial iliac vein.

Vascular injury may occur with extraction of intrapelvic cement and medially migrated sockets. CT arteriography is helpful in identifying proximity of vascular structures to implant and intrapelvic control of vessels prior to component removal is occasionally required. With difficult cases get the vascular surgeons to scrub in on the case.

Urinary tract complications

- Bladder infection is the most common complication, with ~7–14% after THA
- Urinary obstruction should be treated before THA

Trochanteric non-union and migration

- This is a concern with the Charnley approach

Heterotopic ossification

- Incidence is variable (3–50%)
- Only 2–7% have significant symptoms
- Candidates may be asked risk factors, classification, prevention and management of HO in the oral or clinical exam (discussed later in this chapter)

Gastrointestinal

- Bleeding gastric ulcer, acute cholecystitis and postoperative ileus (usually neurogenic)

Myocardial infarction and/or congestive heart failure

- Preoperative cardiac opinion is advisable if the patient has a significant history of ischaemic heart disease

Fat embolism syndrome

- In fat embolism syndrome fat particles and bone marrow are forced into the circulation at the time of femoral preparation and stem insertion

Death[39]

The 11th NJR report in 2014 performed an in-depth analysis of factors associated with 90-day mortality after THA. Severe liver disease was associated with a 10-fold and metastatic cancer a 7-fold increase, congestive cardiac failure and myocardial infarction with a 3-fold increase and renal disease a 2-fold increase in relative risk of death within 90 days of surgery. With adjustment for age and gender the mortality risk had halved over an 8 year period. Four treatment variables were associated with decreased mortality: (1) Spinal anaesthetic or a combination of spinal and another anaesthetic, (2) posterior approach, (3) the use of mechanical thromboprophylaxis and (4) the use of chemical thromboprophylaxis, but multivariable analysis showed adjusting for these factors does not fully account for the decreased mortality over time. Type of prosthesis was unrelated to mortality. Being overweight was associated with lower mortality, but a lot of data entries were missing (59.5%).

Examination corner

Adult and pathology oral 1

Describe your preferred approach to the hip for THA

Can you quote a dislocation rate for your approach?

What prostheses would you choose for the femur and acetabulum and why?

Can you quote survival rates from the Swedish Hip Registrar for your implant?

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EXAMINER: What are the complications following THA?

The candidate went through the various complications that can occur and their incidence.

EXAMINER: What is the overall complication rate?

CANDIDATE: Overall 10% of patients are not happy with their THA

EXAMINER: Are you going to tell patient that?

CANDIDATE: I would warn the patient that 1 in 10 of patients who get a THA either have a significant complication or are not entirely happy with the outcome of surgery.

Adult elective orthopaedics oral 3: Radiograph of an arthritic right hip

- Describe the radiographic features
- What are the radiographic differences between an osteoarthritic and a rheumatoid hip?
- What are the indications for THA?
- Give a detailed preoperative assessment of the patient

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- Go through obtaining informed consent for a THA
- General discussion about the Swedish arthroplasty hip register. Voluntary basis, collect and analysis of outcome data about primary THA and re-operations, revisions, etc. Allows identification of predictors for both good and poor results, sets revision THA as the endpoint for survival analysis and concerns with completeness of reporting

Registries should comply with local, federal and state legislation regarding privacy
Registries in general require skilled statistical analysis, interpretation by skilled orthopaedic surgeons and outcomes to be peer-reviewed
They enhance the professional development of surgeons and improve patient safety and outcome. Funding comes from government funds, surgeons' fees, implant company fees, donations, etc

Adult elective orthopaedics oral 5

- How would you plan if you were to start using a different type of THA?
- Survival analysis – Details, methods, Kaplan–Meier curve, etc
- Draw survival analysis curve and describe it
- Confidence intervals

Dislocation of THA[s]

Introduction

Early dislocation occurs in the early postoperative period (within 6 months) after THA and is usually caused by non-compliance with postoperative instructions before full muscular strength is attained or a technical error during surgery.

Dislocation occurring between 6 months and 5 years is categorized as intermediate and is usually the result of older age, female gender (decreased muscle mass) and predisposing factors (AVN, inflammatory arthritis, etc).

Late dislocation occurs after 5 years and generally requires surgical treatment. It has a multifactorial etiology polyethylene (bearing surface) wear, deterioration in muscle mass, neurological impairment, and fractures such as trochanteric avulsion as a result of wear and osteolysis. Additional predisposing factors for late dislocation include younger age (greater wear), female gender (muscle laxity), unrecognised component malpositioning, and prosthesis–bone impingement as a result of a change in body habitus (weight loss).

Incidence

The reported incidence varies widely, from 0.3% to >10% in different series, with 2–3% a generally accepted figure for primary THA. This figure increases dramatically after each revision operation and can be as high as 25% after multiple operations.

Patient-related factors[t]

- History previous hip surgery (osteotomy, conversion of prior arthodesis). Factors such as poor abductor function, bone loss and deformity increase the dislocation risk
- Revision hip arthroplasty. The main reasons are compromised abductor function and bone loss that leads to compromises in implant orientation and location
- Pre-existing neurological disease. Weakness in muscles around the hip or hip contractures
- Muscular weakness
- Diagnosis, i.e. fractured neck of femur (advanced age, poorer or damaged muscles, greater propensity for falls and altered proximal femoral anatomy), AVN, DDH (poorer muscle strength, bone deformity and alterations from normal implant position)
- Age >80 years (relative risk 1.3) Reasons may include a greater risk of falls, poorer soft tissues and greater

[s] See website additional information www.postgraduateorthopaedics.com.

[t] 'Several patient risk factors for dislocation after THA have been identified and these include . . .' Practice the talk. The FRCS (Tr & Orth) is not just about reading facts in a book.

incidence of confusion and non-compliance with dislocation precautions
- Female gender (relative risk 2.1). The literature is unclear. Hypothesis is an increased tissue laxity. Duration of follow up may influence absolute rates as women live longer
- Inflammatory arthritis. Poor soft-tissue quality
- Higher ASA score
- Cognitive dysfunction (confusion, dementia, substance abuse). Poor compliance with hip dislocation precautions
- Stroke, Parkinson's disease and grand mal epilepsy: despite a general belief that there is a higher rate of dislocation in such patients, there is very little evidence to support this

Implant-related factors

- Head size. The distance a head must travel to dislocation is defined as excursion. The greater the excision distance the more stable the hip. Excursion distance is usually one-half the diameter of the femoral head. Large femoral head sizes increase volumetric PE wear that can lead to increased periprosthetic osteolysis
- Femoral offset
- Head–neck ratio
- Acetabular component design (hooded or constrained cups)

Surgical factors

- **Surgical approach**: Multiple previous studies have shown a higher dislocation rate with the posterior approach[u]. However, recent studies suggest that with careful posterior soft tissue and capsular repair along with reattachment of the external rotator muscles, rates are now equivalent to the anterolateral approach[40]. Posterior approach also predisposes to retroversion of the cup
- **Acetabular position and orientation**: Anteversion should be 10 ± 10° (>25° increased risk of dislocation). The theta angle or angle of inclination (i.e. coronal tilt) should be 40 ± 10°
- **Femoral position and orientation**: Anteversion should be 5–10° (if >15° there is an increased risk of dislocation)
- **Impingement**: Femur against the pelvis or residue osteophytes, femoral prosthetic neck on the acetabular cup
- **Soft-tissue tension**: Preoperative templating should assess head offset and neck length such that when the prosthetic stem is inserted the appropriate neck length and offset are restored

[u] Be careful with this topic. Most hip surgeons consider the higher dislocation rate historical and that today it is much less of a problem, especially if using larger femoral head sizes.

Surgeon factors

- **Surgeon volume**: Surgeons who perform <30 THA per year have about 3 times the dislocation risk. A review of Medicare patients in the USA described varying rates of dislocation according to surgeon volume (1–5/year, 4.2%; 6–10, 3.4%; 11–25, 2.6%; 26–50, 2.4%; >50, 1.5%)[41]. A systemic review of the literature published in 2006 by Battaglia et al.[42] (from Charlottesville, Virginia, USA[v]) demonstrated a substantial positive association between surgical volumes and improvement in most THA outcomes, including dislocation. Lower dislocation rates were associated with increasing surgical volume and this correlation appears to be stronger for surgeon volumes than for hospital volumes.

Hip stability

Dislocation is frequently a multifactorial issue. Assessment should focus on the following four variables:

1. **Component design:** range of motion of a hip prosthesis consists of two parts; the primary arc and the lever range. The primary arc is the arc of motion allowed between the two ends of impingement and is controlled mainly by the head/neck ratio. By maximizing this ratio, best stability is achieved. The head/neck ratio is defined as the diameter of the head divided by the diameter of the neck. Using a large head with a narrow neck will maximize this and increase stability. Addition of a skirt (femoral neck collar) or an acetabular lip will decrease the ratio and thus decrease the primary arc before impingement.

The second part of the range of motion is called the lever range. This is primarily controlled by the head radius. The lever range is essentially the range of motion allowed between head/neck impingement and dislocation of the head. This is closely related to the excursion or jump distance, which is the distance the femoral head has to travel after primary impingement to dislocate. This distance is equal to the radius of the femoral head. Maximizing the femoral head size diameter will increase both the lever range and the excursion distance, which in theory increases hip stability.

2. **Component alignment:** The optimal acetabular component position is in 15–20° anteversion and 40–45° inclination. The socket must be placed as medial and as inferior as possible to place the hip centre of rotation in an anatomical position. This will reduce the reactive forces the THA is subjected to. The femoral stem is best placed in 0–15° anteversion. Placement of the acetabular of femoral components in increased anteversion increases the risk of anterior dislocation,

[v] In the viva or clinical discussion most fair minded examiners would be suitably impressed if you quickly throw in the origin of a paper and this moves up the examination process onto a different platform. The occasional examiner may however think you are being too smart or smug. Still other examiners will say you don't really need to know papers to pass the exam!

whereas increased retroversion increases the risk of posterior dislocation. An open cup (>50° inclination) increases the risk of posterior-superior dislocation, whereas a closed cup (<40° inclination) increases the risk of inferior dislocation.

3. **Soft-tissue tension:** this refers to the abductor complex tension primarily. The abductor complex is made of the gluteus medius and gluteus minimus muscles. Factors affecting tension of the complex include the position of the hip centre of rotation (COR), the hip offset and the neck length. Decreasing the hip offset will result in a reduced abductor moment arm (d). This results in a reduced moment of force from the abductors (M_{ab}) as $M_{ab} = F_{ab}xd$. This will increase the joint reaction force generated by the hip. Clinically, this will present as a Trendelenburg gait with a gluteus medius lurch when walking. The risk of dislocation is also increased. A low neck cut will result in a short THA neck. This will also have a negative effect on the abductor complex tension and reduce the offset. Impingement of the greater trochanter with the ilium can also occur further reducing stability. Compensating for a low neck cut by using a neck collar will reduce the primary arc by reducing the head/neck ratio, which can cause further instability.

4. **Soft-tissue function:** This is affected by a wide range of neuromuscular disorders and local hip soft-tissue problems. The neuromuscular disorders can be classified into central (cerebral palsy, stroke, seizures, etc) and peripheral (spinal stenosis, neuropathy . . . etc). Local soft-tissue factors include trauma, ageing, infection, malignancy . . . etc).

Management

As a general rule, if the hip dislocates more than twice, recurrent dislocation is likely, and the hip should be revised to enhance stability. Remember to rule out infection and look for an obvious cause such as component malposition, retained osteophytes or cement. Although in many circumstances the cause of recurrent dislocation is multifactorial, there is usually one main area that stands out.

Beware though of finding 'the cause' as even the 'obvious causes' such as component mal-alignment may have a disappointing outcome despite surgical correction.

- **Conservative:** May very occasionally be indicated in elderly, non-mobile, medically unfit patients not in pain
- **Closed reduction under GA/spinal and EUA:** It is important to screen the hip under image intensification to assess for stability and determine the positions that the hip dislocates. A period of bed rest (Charnley wedge) followed by mobilization with a hip brace worn for usually 6 weeks. Good success rate in first-time dislocations without a clearcut mechanical problem. Less successful in recurrent dislocations
- **Revision of the arthroplasty components to improve position:** Applicable if there is significant component malpositioning
- **Modular component exchange (dry exchange):** Only indicated if the components are reasonably well positioned. Options include increasing the head size and/or neck length and changing the acetabular liner. Large femoral head sizes increase the head-to-neck ratio and jump distance required for dislocation. Increasing the neck length increases femoral offset and lessens the risk of impingement
- **Removal of sources of impingement:** Cement, osteophytes, etc. Usually in the postoperative period
- **Posterior lip augmentation device (PLAD):** Possible option for a recurrent dislocating Charnley in an elderly woman with a small head implanted using a posterior approach. Unlikely to be successful in other situations. Concerns with impingement, wear and limited hip movements. Advantages include reduced operating time, lower intraoperative blood loss and shorter hospital stay.
- **Advancement of the greater trochanter:** Increases abductor tension and stability. This operation is somewhat historic as the advent of modular implants allows an increased femoral neck length to accomplish the same goals without the possibility of greater trochanteric non-union. Therefore, it is best suited for proximal migration of an ununited trochanter after a trochanteric osteotomy
- **Soft-tissue augmentation:** Reinforcement of the hip abductor muscles or the posterior aspect of the hip joint using Achilles tendon allograft, fascia lata or synthetic ligament in patients who are poor candidates for other options such as constrained liners. These procedures can be technically demanding and are likely to fail in patients with any component malpositioning
- **Bipolar hip arthroplasty:** May have a role in the salvage management of complex recurrent instability in which other stabilisation procedures have failed. This procedure has a high failure rate in this situation, and offers only modest improvement in function. It increases the overall range of motion (ROM) with articulation at two bearing surfaces. This provides a greater safe arc of motion, increased stability, improved head: neck ratio and a larger jump distance. The main disadvantage is that the mobile head articulates directly with acetabular bone and can cause pain and medial bone erosion
- **Constrained acetabular socket design:** Not a good choice for a young patient as there is a high failure rate after 5 years owing to significant shear forces transmitted to the bone–prosthesis interface leading to accelerated wear and loosening. There is a restricted ROM and residual hip pain can be very problematic. Consider as a last chance bail-out option when other procedures have failed. This can be technically difficult surgery, and is usually successful in preventing dislocation but patients may not tolerate the implant very well. Complications include liner displacement from the acetabular shell or an acetabular cup dislodging from the acetabulum. When dislocations occur with a constrained device they are difficult to manage. There may be the possibility of converting an uncemented

cup to a constrained option. In general try to use an uncemented constrained socket in preference to a cemented socket as this increases implant longevity

- **Tripolar arthroplasty (dual mobility):** dual-mobility hip components provide for an additional articular surface, with the goal of improving hip range of motion until impingement occurs. A large polyethylene liner articulates with a polished metal acetabular component, and an additional smaller metal head is snap-fit within the polyethylene liner. In the first articulation the head is "engaged" but mobile within the polyethylene (PE) liner. If the femoral neck and the rim of the PE liner come into contact, a second articulation begins to function and consists of the back of the PE liner and the metallic acetabular shell. These components have been used for primary total hip arthroplasty in patients at high risk for dislocation, total hip arthroplasty in the setting of femoral neck fracture, revision for hip instability, and revision for large MoM hip
- **Resection arthroplasty:** Usually used in the multiply revised patient with significant soft-tissue and bone deficiency. Not a good option as it leaves the patient with a shortened leg and significant limp

Examination corner

Adult elective orthopaedics oral 1: Radiograph of a dislocated right Thompson's prosthesis (Figure 16.15)

EXAMINER: This is a radiograph of a 78-year-old woman who had a cemented Thompson's hemiarthroplasty performed for a fractured neck of femur 5 days previously. She was mobilizing well until she developed pain and had this radiograph taken.

CANDIDATE: This shows a dislocated prosthesis.

EXAMINER: What are the causes of dislocation?

CANDIDATE: Causes of dislocation could include the Thompson's prosthesis inserted incorrectly usually too much anteversion, residue osteophytes causing impingement, retained cement, weak abductor function, faulty repair of the surrounding soft tissues, non-compliance with postoperative instructions.

EXAMINER: How are you going to manage this patient? She mobilized well round the house prior to the fracture and occasionally went out with her daughter to the shops.

CANDIDATE: She needs to be taken to theatre and a closed manipulation and screening performed of this hip to access for stability.

EXAMINER: What are the chances of obtaining a closed reduction?

CANDIDATE: In most cases I have been involved with the hip has relocated without any difficulties encountered.

EXAMINER: Closed reduction failed. What are you going to do?

CANDIDATE: I would perform an opened reduction of the hip.

EXAMINER: Would you do anything else at the time of opened reduction?

CANDIDATE: In the form of any type of major revision – No. She probably wouldn't have been worked up for this. I would attempt to get the hip back into the joint and look for any obvious cause for the dislocation. I would manage the patient with bed rest for 3 weeks and then cautiously with a hip brace.

EXAMINER: She dislocates again whilst in bed and has developed a sacral pressure sore.

CANDIDATE: This is a difficult situation. Further attempts at closed or open reduction are unlikely to be successful. I would refer on to an experienced hip surgeon.

EXAMINER: You are the experienced hip surgeon.

CANDIDATE: The choice lies with either a Girdlestone procedure or revision surgery. A Girdlestone procedure isn't a particularly good option, as she will find it very difficult to walk again. How fit is she for revision surgery?

EXAMINER: She wants surgery so she can walk normally again. She has a number of co morbidity factors and it would be very risky to perform major revision surgery.

CANDIDATE: After a full discussion with the patient and her relatives, and if the risks of surgery were acceptable I would revise the stem to a 44 Exeter DDH stem and perform a cement in cement revision. For the acetabulum I would use a cemented constrained liner.

EXAMINER: Why would you want to use a cemented constrained acetabular cup – Why not just cement a straightforward acetabular cup into the socket and use a large head?

CANDIDATE: I think that there would be a risk of a further dislocation if the cup wasn't constrained. Even with a large femoral head there would still be a significant risk of dislocation. A constrained cup is a safer option in someone who is elderly and has significant co-morbidity factors.

EXAMINER: Good. What are the indications for a constrained implant?

CANDIDATE: I would consider a constrained implant in the following circumstances: Multiple dislocations with previous failed attempts of management, patients with cognitive issues, patients with very poor abductor function and where there is no obvious cause for instability. The failure rate using other management options in these situations is high.

Ideally they are best suited for elderly low-activity patients and I would try to avoid using them in younger patients because of the worry of impingement and PE-induced osteolysis with early loosening and failure.

EXAMINER: We used a cemented constrained cup with a cement in cement femoral stem revision. This is her postoperative radiograph (Figure 16.16). At surgery the prosthesis had been inserted in about 40° anteversion, the neck cut too low and the femoral head size was too large.

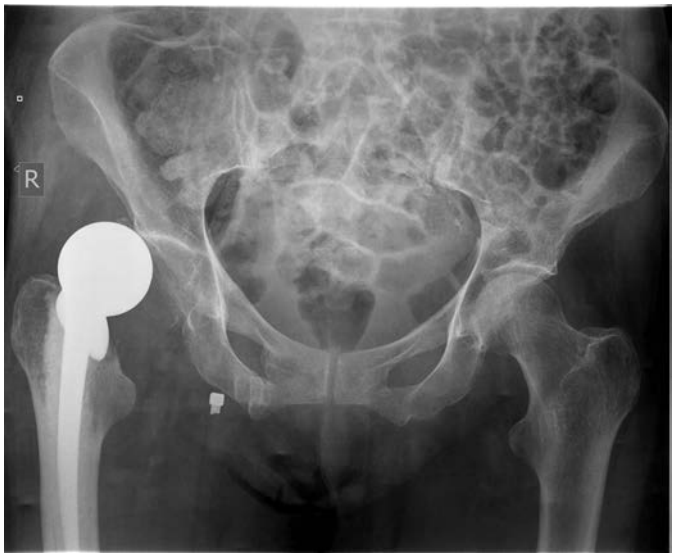

Figure 16.15 Dislocated right Thompson prosthesis

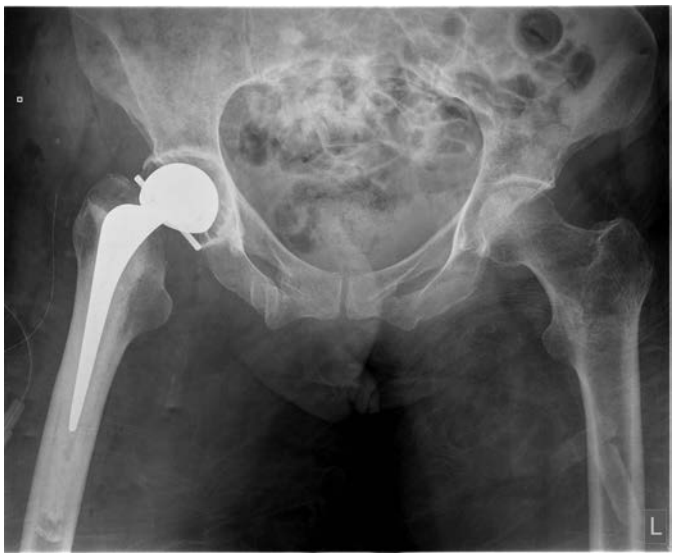

Figure 16.16 Radiograph following revision to constrained THA

Adult elective orthopaedics oral 2

Radiographs of a posteriorly dislocated hip – Uncemented components. Examiner said this THA was performed 2 weeks ago. First dislocation.

- Describe the findings

Dislocated THA. There were no other abnormal findings. Satisfactory component orientation.

- What might have caused this?

The candidate explained that, as it was the first episode of dislocation, it could be due to patient non-compliance (taking into account the radiographic parameters). Infection should be ruled out through a careful history and blood tests.

- Treatment?

Closed reduction in A&E under sedation if safe or closed reduction in theatres under GA with assessment of the range of motion and stability. Hip brace for 6 weeks.

- If closed reduction fails?

Open reduction and assessment of soft-tissue interposition in the acetabulum, soft-tissue tension or an impingement problem. If a simple cause is identified, one must be prepared to address this (liner/head exchange, excision of acetabular osteophyte . . . etc).

- The hip dislocates again 2 weeks later?

Closed/open reduction. CT scan to assess component orientation (anteversion femoral stem, acetabular component version. Rule out infection (history-has the patient shown any signs of infection since the operation, blood tests-inflammatory markers) Discuss findings with patient and counsel regarding non-operative vs revision if a cause is identified.

With a viva question on hip dislocation there are three possible answer options:

Option 1

Candidates can immediately start discussing patient related factors continuing on with implant, surgical and surgeon factors as they settle into the question

- Patient-related factors
- Implant-related factors
- Surgical factors
- Surgeon factors

Option 2

The second option is to discuss THA factors relating to hip stability. This is more difficult as you may get side tracked by the examiners probing you in further detail about femoral offset or head/neck ratio (component design). In addition, there is some overlap of headings for example component design (e.g. offset) will also affects soft-tissue tensioning.

- Component alignment
- Component design
- Soft-tissue tensioning
- Soft-tissue functioning

This answer option is good to use if a candidate is shown a radiograph of a dislocated THA

Option 3

The third option is to randomly mention any factor associated with hip dislocation that comes into your head. This is an unstructured method and doesn't impress the examiners. This approach is usually seen from candidates who haven't thought through the topic beforehand. Option 3 may lead you up a cornered blind alley discussing obscure factors related to hip dislocation. The thread of the question can get lost. Avoid.

There are several clinical scenarios that the examiners can present:

- Dislocation in the immediate postoperative period. Either a discussion of conservative management and factors to prevent re-dislocation or a grossly mal-aligned implant requiring revision

- A dislocated MoM hip. This is a curved ball that can go anywhere
- A dislocated hip with avulsed greater trochanter. The Woo and Murray paper[43] reported a 6-fold increase in dislocation rate to 17.6% with trochanteric avulsion. Re-attachment generally advised. Methods (Dall-Miles cable grip system), success rate etc
- A dislocated revised hip. Discussion may focus on a constrained acetabular socket design or conversion to dual mobility
- A dislocated revised hip following a previous MoM complicated with ALVAL (again constrained cup or dual mobility option)
- A dislocated Thompson ultimately leading to a constrained cup option
- Loose total hip that has dislocated. The hip will need to be revised. The viva would discuss how to plan and perform revision of that particular hip
- Late dislocated hip first time no obvious cause. How to manage

Although there are a large number of potential management options for a dislocated hip the actual choice is likely to be limited to three or four obvious preferences. This would be in keeping with realistic clinical situations that examiners are now encouraged to ask candidates in the exam.

You still, however, would need to know and be able to discuss all the options available in the viva exam.

Basic science 1

You may be presented a clinical condition such as dementia or Parkinson's disease that increases the risk of THA dislocation and asked to comment on factors you can alter to minimize this risk

Anterolateral approach, large head size and maximizing head/neck ratio. Consider using dual motion hip prosthesis. Take care with analgesia avoiding strong opiates to minimise potential for confusion post op, consider nerve blocks peri-operatively etc. Consider ITU bed initially for close medical management of patient, careful fluid balance etc.

Infection complicating THA

Incidence

- Approximately 1% after primary and 3–4% after revision hip surgery

Classification

Fitzgerald

- Acute postoperative period (up to 3 months)
- Delayed deep infection (3–24 months)
- Late haematogenous >24 months

Approximately one-third of the infections fall into each group.

Coventry

I. First 30 days
   Immediate postoperative period – The infected haematoma or superficial infection that progresses to a deep infection

II. 6–24 months
   Smaller inoculum or lower virulence at the time of surgery, chronic indolent infection

III. >2 years
   Least common, usually haematogenous spread although bacteria implanted at the time of the original surgery may have remained dormant until a change in the host's immunity occurs (onset of diabetes, malignancy, etc)

Tsukayama et al.[44]

1. Early postoperative infection
   Presents <1 month after surgery with a febrile patient and a red swollen discharging wound
2. Late chronic postoperative infection
   The patient is well, the wound has healed well, there is a worsening of hip pain and a never pain-free interval
3. Acute haematogenous infection
   This can occur several years after surgery with a history of bacteraemia (UTI or other source of infection) and severe hip pain in a previously well-functioning hip
4. Positive intraoperative culture
   At least three samples from different locations taken with clean instruments. This occurs when a preoperative presumptive diagnosis of aseptic loosening was made

AAOS

AAOS make a clear distinction between early and late PJIs: an early infection occurs within three weeks of the procedure, whereas PJI that develops thereafter is considered late

Organisms

Numerous studies have shown that Gram-positive organisms are the most common bacteria causing joint infections, with *Staphylococcus aureus* and *Staphylococcus epidermidis* accounting for the majority of infections. Enterococci, streptococci and Gram-negative organisms such as *Escherichia coli, Pseudomonas* species and *Klebsiella* species are less common but not infrequently reported.

The glycocalyx

This is the polysaccharide biofilm that permits increased adherence and survival of bacteria on biosynthetic surfaces. Numerous factors, including restricted penetration of antimicrobials into the biofilm, decreased bacterial growth rates and expression of biofilm-specific resistance genes, all contribute to bacterial and biofilm resistance.

Oral questions

What factors are involved in reducing the infection rate in THA surgery?

This is a classic FRCS (Tr & Orth) oral question. Dividing your answer into preoperative, perioperative and postoperative

factors greatly simplifies your answer and, more importantly, demonstrates to the examiners a more structured approach in your viva technique. That said, most candidates, even if they mention factors in a random haphazard manner, should still be able to obtain a pass mark for this question.

Prevention of infection in THA

Prophylactic measures to reduce hip arthroplasty infection are given in Table 16.9.

Preoperative factors

- Same-day admission
- Separation of elective from trauma cases
- All septic lesions should be examined and treated (feet, urinary, dental)
- Shave in the anaesthetic room (not night before)

Perioperative factors

- Antibiotic prophylaxis: Systemic antibiotics, antibiotic-loaded cement
- Surgical technique: Gentle handling of tissues, careful haemostasis, limitation of haematoma formation, avoid cremation of tissues/necrosis, length of surgery, wound lavage, etc
- Movement: Avoid unnecessary theatre personnel movement during surgery
- Ace masks: BOA guidelines
- Gowns: Modern, weaved patterns
- Gloves and hands: Two pairs of gloves, changing the outer ones frequently
- Head gear: No hair exposed
- Body exhaust systems
- Sterile drapes: Disposable non-woven drapes
- Drainage wound: Arguments for and against
- Ventilation system: Laminar flow, ultra-clean-air system
- Ultraviolet light: Bactericidal

Postoperative factors

- Antibiotic cover for urethral catheterisation
- The risk of infection is increased in rheumatoid arthritis, diabetes, those with immunosuppression and those with a history of previous joint infection

Table 16.9 Prophylactic measures for hip arthroplasty infection

MRC trial[45]	Factor
Antibiotic-loaded cement	11
Systemic antibiotics	4.8
Ultraclean air	2.6
Plastic isolators	2.2
Body exhaust suit	2.2

Diagnosis

This can be difficult, especially if a low-grade infection is present. The surgeon's clinical diagnostic skills and judgement are more important than any specific test.

Investigations

History

Type 1

Continuous pain, usually fever, erythema, swollen and tender fluctuant wound; either an infected haematoma or deep spread from a superficial wound.

Type 2

Gradual reduction in function of the hip with increasing pain. Hip never 'feeling right' from the time of original operation. Prolonged period of wound discharge postoperatively.

Type 3

History of sepsis. Dental extraction, chest or urine infection.

Blood tests

White blood cell count

Usually normal and not helpful unless the infection is acute.

Erythrocyte sedimentation rate (ESR)

An ESR >35 mm/h 1 year after THA in the absence of any other systemic illness suggests hip infection until proven otherwise. However, the ESR may not always rise in the presence of deep sepsis. It has low specificity and sensitivity as a marker of prosthetic joint infection

C-reactive protein (CRP)

This acute-phase reactant peaks 48 hours postoperatively and returns to normal in 2–3 weeks. Based on multiple studies >10 mg/L is significant. Sensitivity is 96%; specificity, 92%.

Interleukin-6 (IL-6)

IL-6 is produced by stimulated monocytes and macrophages, and it induces the production of several acute phase proteins including CRP. IL-6 peaks at 2 days following uncomplicated arthroplasty and rapidly returns to a normal value. Recent studies suggest that IL-6 is a more accurate marker for periprosthetic infection than ESR or CRP.

Radiographs

These are of limited value with respect to the infected hip. Both infected and aseptic hips can have similar appearances. However, some radiographic signs suggestive of infection include:

- Localized or irregular, scalloped pattern of endosteal bone erosion
- Rapidly progressive radiolucent lines
- Periosteal new bone formation (considered by some to be pathognomonic of deep infection)

- Evidence of early loosening
- Lacy pattern of new bone formation
- Area of bone erosion >2 mm about the entire cement mass of stem or cup

Radiographic signs of loosening are seen in two-thirds of late infections, but <50% of early infections.

Radionuclide imaging

Technetium-99m scan

This is very sensitive but is non-specific. Increased uptake can be found in stress fractures, tumours, loosening, heterotopic bone formation and other inflammatory and metabolic disorders. A technetium scan can remain positive for up to 2 years following an uncomplicated THA. The technetium scan is most useful if negative, as infection is unlikely, and it allows elimination of many of the component-related causes of pain at the site of a THA.

^{111}Indium-labelled white cell scan (leukocyte scan)

In theory in a labelled leukocyte scan, indium-111 should not accumulate at sites of increased bone turnover in the absence of infection. The usefulness of this scan remains controversial. It has a limited role because of its sensitivity of 44%, its specificity of 100% and accuracy of 82%[46].

Hip aspiration, arthrogram and needle biopsy

Aspiration should not be performed routinely for all patients with pain at the site of a THA because the false-positive rate is unacceptably high. It is most useful where there is clinical or radiographic evidence of infection or elevation of either the CRP or ESR. If aspiration is to be performed, all antibiotics should be stopped for at least 4 weeks to improve sensitivity. Transport should be rapid to allow immediate incubation and to minimize the risk of a false-negative aspiration. Samples require extended culture. A spectrum of sensitivity (67–92%) and specificity (94–97%) is reported in the literature[47]. Follow the standard protocol. The accumulation of dye in pockets with an arthrogram may suggest abscess formation (pseudobursa).

Biopsy at operation, frozen section and Gram stain

Intraoperative cultures are not always positive and frozen section is not available in all centres but can be a valuable diagnostic adjunct in equivocal cases.

Infection is defined if >10 neutrophil polymorphs per high-powered field are seen[48].

Management

Suppression treatment

Long-term oral antibiotic suppressant treatment alone will not eradicate deep infection but may control the sepsis and may have its place in an elderly patient who is medically unfit for major surgery.

Debridement, antibiotics and retention of prosthesis

This is carried out for either early postoperative infection or acute haematogenous infection if the duration of clinical signs and symptoms is <3 weeks (before formation of the glycolax), the components are stable at the time of debridement, the organism sensitivity is known and the overlying soft tissues and skin are of good quality.

Debridement involves removal of fibrous membranes, sinus tracts and devitalized bone and soft tissue, and exchange of the polyethylene liner and femoral head. If one or both components are loose, both components, all cement and all infected and necrotic tissue should be removed. The success rate is very variable, from 16% to 89% depending on how strictly treatment criteria are followed[49]. Following debridement antibiotics are continued for a minimum of 6 weeks.

Single-stage procedure

Advantages

- Only one major operation
- Improved postoperative mobility and pain
- Avoids disuse atrophy, limb shortening and soft-tissue scarring associated with a second procedure
- Reduced cost

Avoids the complications of cement spacer use (spacer fracture, abraded particles from the spacer, bone resorption)

Disadvantages

- Demanding prolonged procedure
- Antibiotic sensitivities must be known preoperatively
- Use of antibiotic-loaded cement for femoral fixation is considered essential, which limits the choice of prosthesis

Contraindicated

- If antibiotic sensitivities are not known preoperatively
- Doubt about the adequacy of debridement
- Massive bone loss requiring grafting (owing to increased risk of sepsis)
- Presence of multiresistant bacteria

Results

One-stage exchange arthroplasty has been popularized by Buchholz et al.[50], whose group reported a 77% success rate in 583 patients. Wroblewski[51] reported a 91% success rate, and a very recent study by Zeller et al. reported a 94% success rate[52].

Two-stage revision

The first stage consists of excision of the sinuses, the drainage of all abscesses and the meticulous removal of all foreign material: Membranes, cement, plugs and any potentially infected soft tissue. The timing of the second stage depends on the response to antibiotics, the patient's general well-being, wound healing and bone stock.

Advantages

- Adequacy of debridement – Can be repeated at the time of reimplantation
- Infected organism known and appropriate antibiotic given
- Persisting foci of infection can be identified
- Allows clinical assessment of treatment prior to reimplantation
- Allows uncemented reconstruction
- Augmentation with allograft may be carried out with greater confidence

Disadvantages

- Two operations and their associated morbidities
- Prolonged period of bed rest and hospital stay between the two stages resulting in a limited functional outcome
- Increased cost
- The second stage of a two-stage revision is further complicated by altered anatomy and loss of planes of dissection

Most reported protocols advise a 6-week gap between the two stages but this can be increased if necessary. Recently it has been suggested that shortening the interval period to 3 weeks does not increase the rate of infection.

A delayed exchange (two-stage procedure) is indicated for:

- Resistant organisms
- Gram-negative organisms (*Pseudomonas, E. coli*)
- Draining sinus
- Unhealthy or oedematous soft tissue
- Well-established osteomyelitis with loss of bone stock

Results

A recent review of the Norwegian Arthroplasty Register[53] comprising of 784 infected THAs reported a success rate of 94%. Two-stage revision is probably safer and more successful than a single stage revision and the majority of surgeons would perform a two-stage revision. Both methods in experienced hands give similar results and both methods have advantages and disadvantages. As yet, no randomised clinical trial exists to compare the two methods of treatment. The complexity of the operative procedure and the many factors involved has discouraged investigators from evaluating the timing of surgery.

Antibiotic spacer

A cement spacer, the most common being PROSTALAC (prosthesis of antibiotic-loaded acrylic cement), can be inserted between first and second stage revisions to maintain soft-tissue balance and leg lengths. This allows local delivery of a high concentration of antibiotics. A custom-made spacer in the operating room can be used, which consists of coating a small, inexpensive sterile femoral component with antibiotic-laden cement.

The choice between an articulating and non-articulating spacer remains controversial; however, most surgeons prefer articulating spacers when feasible in order to maintain leg length and soft-tissue tension. These permit partial to full weight-bearing, but there is an associated risk of dislocation. Non-articulating spacers are preferable in patients with extensive bone loss, or with deficient abductors secondary to a high risk of dislocation if an articulating spacer is selected.

Salvage

If definitive treatment fails the following salvage operations may be required.

Resection arthroplasty

This is an occasionally necessary salvage procedure. It often provides marked relief of pain but results in the use of ambulatory aids, patients fatigue easily and have a Trendelenburg gait. Patients may experience hip joint pain and have a large leg length discrepancy.

Arthrodesis

This is a technically demanding procedure and is rarely performed.

Amputation

This is occasionally performed on patients with a life-threatening or limb-threatening infection or who have massive soft-tissue and bone loss or vascular injury. The presence of systemic co-morbidities is strongly associated with the rate of amputation.

Antibiotics in cement

This is a controversial issue. The use of antibiotic-impregnated cement in primary THA may lead to the emergence of resistant organisms.

The prophylactic use of antibiotics with dental treatment

In 1997 a panel of experts adopted by the American Academy of Orthopaedic Surgeons (AAOS) and the American Dental Association decided that antibiotic prophylaxis was not routinely indicated for dental patients with total joint arthroplasties, but should be considered in a small number of patients undergoing procedures with a high incidence of bacteraemia.

Examination corner

Adult elective orthopaedics oral 1: Infected THA

- Risk factors
- Investigations
- Management

Risk factors include presence of co-morbidities (morbid obesity, rheumatoid arthritis, myocardial infarction, atrial fibrillation), higher ASA scores (>2), bilateral procedures, allogenic blood transfusion, postoperative surgical site infection and longer hospital stay[54].

Adult elective orthopaedics oral 2: infected THA

Most of this oral seemed to be spent on discussing the investigations and various management options of the infected THA.

Table 16.10 Vancouver classification

Type	Location	Subtype	Management options
A	Trochanteric	A_G: Greater trochanter	Treat conservatively with protected weight-bearing. Indications for ORIF: Displaced >2.5 cm, abductor weakness due to non-union and chronic pain
		A_L: Lesser trochanter	Treat conservatively with protected weight-bearing unless a large portion of the medial cortex is involved
B	Around or just distal to stem	B_1: Stable prosthesis	ORIF with cerclage wires alone if long oblique or plate osteosynthesis. Cortical strut grafts and cerclage fixation can be used separately or utilized in combination with a plate
		B_2: Unstable prosthesis	The fracture need to be bypassed with a longer (revision) stem with plate reinforcement, with or without cortical strut
		B_3: B_2 + inadequate bone stock	A combination of a revision stem and bone grafting (impaction or strut grafting). In severe osteolysis revision stems with distal screw fixation are preferred
C	Well below the stem		Ignore implant and manage fracture with locking plate

Adult elective orthopaedics oral 3
How would you manage the infected THA?

A major part of this oral answer is to be able to discuss the advantages and disadvantages of one-stage vs two-stage revision hip surgery for infection:

- Conservatively on long-term antibiotic suppression: Low virulence organisms, patient unfit for surgery
- Incision and drainage and washout: Only applicable in the early postoperative stage or within 3 weeks of an acute haematogenous infection
- One-stage or two-stage procedure
- Resection arthroplasty
- Arthrodesis (controversial)
- Amputation: For uncontrollable life-threatening sepsis

Adult elective orthopaedics oral 4
Comment on a THA radiograph.

- Painful.

Why?

- Infection
- Investigations and management.

Adult elective orthopaedics oral 5
Infection control in theatres, including MRC trial on the effects of laminar flow, antibiotics and exhaust suits.

Adult elective orthopaedics oral 6

- Prevention of sepsis following THA: Preoperative and intraoperative measures
- Management of wound haematoma following THA

Basic science oral 1

- General discussion about the prevention of infection in THA
- Discussion of laminar flow. Score 8 candidates should be aware of the recent controversies suggesting no benefit from laminar flow use in reducing early deep infection

Periprosthetic femoral fractures and failure modes

Cemented implants tend to fracture late (5 years or so). They occur most commonly at the stem tip or distal to the prosthesis. This is due to the modulus mismatch in the area resulting in rising stresses.

In revision cases, fractures tend to occur at the site of cortical defects from previous operations. Fractures also occur if the new stem does not bypass a cortical defect by >2 cortical diameters.

Uncemented implants tend to fracture within the first 6 months after implantation.

Incidence

- 1% primary THA
- 4.2% revision THA

History

Important points in the history include:

- Loosening of the prosthesis may proceed many periprosthetic fracture and, therefore, symptoms suggestive of loosening such as constant thigh pain or start-up pain after getting up from sitting should be documented
- Onset of pain: differentiate between a traumatic event (with emphasis on the mechanism of injury) or whether the pain had a non-traumatic spontaneous onset

- Any stigmata of perioperative infection should be very carefully investigated in the history. Any history of prolonged wound healing, draining sinuses, or repetitive antibiotic use should alert the surgeon to previous infection

Classification

Many classifications are descriptive and give information about the site of the fracture but are of little value in formulating a strategy for management.

Duncan and Masri (Vancouver; Table 16.10)

This takes into account three key factors: The fracture site, the stability of the femoral component and the quality of the proximal femoral bone. Although the classification system has proved to be reliable, reproducible and valid one concern is that plain radiographs may fail to distinguish between B_1 and B_2 fractures adequately. This can become problematic intraoperatively as a more extensive procedure may be required without the necessary equipment or implants being on hand or scheduled operating time available[w].

Type A_G and A_L (around trochanteric region)

Usually stable and minimally displaced. Displaced fractures are commonly related to osteopenia, and can usually be fixed adequately by circlage wires supplemented by screws or hook plates if required.

Type B (around femoral stem)

- **B_1 – Prosthesis well fixed:** This occurs in the region of the tip of a well-fixed stem. Spiral and long oblique fractures can be fixed by circlage wires or cables and crimpsleeves. Supplementary fixation can be obtained by using either an onlay cortical strut graft or plate. Short, oblique or transverse fractures can be slow to heal, and are treated with biplanar fixation on the anterior and lateral aspects with any combination of plates and cortical onlay grafts. Bone graft may also be used to enhance fracture healing
- **B_2 – Prosthesis loose and good bone stock:** The best method of management is to use a revision stem, which bypasses the site of the fracture by at least 5 cm or twice the outer diameter of the diaphysis. In most cases a long uncemented stem, which achieves good diaphyseal fixation with or without diaphyseal locking screws, provides the most effective contemporary method for managing these fractures. Occasionally, a cemented long stem prosthesis is used in elderly patients with osteoporotic bone to allow immediate weight bearing
- **B_3 – Prosthesis loose and poor bone stock:** A challenging fracture to manage with a high rate of complications. Best managed surgically, if medically fit, with proximal femoral replacement or so called mega-prosthesis for low demand and elderly. In a young patient, an allograft–prosthesis composite is an attractive option

[w] A nasty surprise to keep you on your toes.

Type C (well distal to stem)

- Fractures well distal to a solidly fixed stem. Type C fractures are best managed with ORIF using a non contact bridging (NCB) periprosthetic locking plate

Complications

- Mal-union (5–30%)
- Non-union (10–30%)
- Periprosthetic refracture
- Infection (10%)
- Reduced function (one-third)
- Plate failure (15%)
- Instability/dislocation (10%)
- Death

Examination corner

Trauma oral 1: Radiograph of a supracondylar fracture in an 80-year-old woman distal to a THA

For management of this case, ORIF was suggested to avoid problems with a stress riser above the supracondylar nail and difficulty with proximal locking so close to the fracture.

I was then shown a radiograph of a retrograde nail with substandard fixation. I was asked how I would manage this if the patient was still on the operating table – Would I remove the fixation? I answered that I would not but that I would consider supplementary fixation and/or a cast brace.

Adult elective orthopaedics oral 1: AP radiograph demonstrating a periprosthetic fracture at the tip of a cemented THA with well-fixed cemented cup

CANDIDATE: This is an AP radiograph of the pelvis, which demonstrates a cemented THA with a periprosthetic fracture just proximal the tip of the prosthesis. The cup appears well fixed, there are no lucencies seen in any of the three Dee Lee and Charnley zones. I would like to see an immediate postoperative film for comparison, to see whether these changes are progressive or were present immediately postoperatively. A lateral radiograph would also be useful.

EXAMINER: How would you manage this patient?

CANDIDATE: I would take a good history, clinical examination and radiological work up of this patient. The type of fracture, and systemic and local host factors should all be considered.

I would request routine blood tests including FBC, U&Es, blood glucose, LFTs and clotting screen. I would cross-match the patient 4 units and order an ECG and chest x-ray. I would attempt to involve my anaesthetic colleagues for early review of the patient in case any further investigations needed to be performed prior to surgery such as ECHO cardiogram or pulmonary function tests. I would apply temporary Thomas Splint traction while I plan definite surgery[x].

[x] Or you could cut to the chase and say 'assuming the patient has been fully worked up for surgery including relevant blood tests, investigations and anaesthetic review, I would treat the fracture according to the Vancouver classification'.

EXAMINER: What classification of periprosthetic hip fractures do you know?

CANDIDATE: I am familiar with the Vancouver classification system. This periprosthetic fracture is a B_2.

EXAMINER: What do you mean by a B_2 fracture[y]?

CANDIDATE: There are three key features to the Vancouver classification (fracture location, stability of the implant, and surrounding bone stock) and three anatomical fracture locations described (trochanteric, around the stem and distal to stem).

EXAMINER: What are the surgical options?

How would you plan for surgery?

CANDIDATE: I would manage the periprosthetic fracture according to the Vancouver classification and perform a revision hip replacement using a long stem uncemented prosthesis. Prior to surgery I would attempt to obtain the original operative notes with implant labels[z]. I would want to know the bearing surfaces and types of implant. I would have all the revision equipment and prostheses available to enable me to deal with any intraoperative eventuality. My preference would be to schedule the surgery on an urgent extra half-day elective list rather than a busy trauma list with theatre staff unfamiliar with complex revision hip systems. I would make sure the company rep. was also available. I would make sure the cell saver was available, order one femoral head frozen allograft and have freezed dried allograft available if necessary. I would make sure OSCAR cement removal system was available I would liaise with my anaesthetic colleagues in case a HDU bed is needed postoperatively[aa].

The acetabular component appears to be well fixed and in a good position. There is no obvious wear of the cup. Taking these points into account I would attempt to retain the acetabular component, providing it was not loose or obviously worn, damaged or mal-positioned upon intraoperative inspection. I would have equipment and prostheses ready 'on the shelf' to perform a revision of the acetabulum if required.

I would approach the hip through a posterior approach incorporating the old incision into this if possible. I would open up the fracture site and expose the cement mantle and this may allow for adequate cement removal but I would have a contingency plan of performing an extended trochanteric osteotomy (ETO) if needed. I would initially use osteotomes to remove as much cement as possible proximally and then OSCAR to remove the distal cement and bone plug. I would then use cerclage cables to reconstruct the femur and sequentially ream up the femoral canal until the reamer bites and chatters with the femoral cortex. I would plan to use a long stem uncemented modular tapered fluted revision prosthesis aiming for a good scratch fit distally. If this could not be achieved I would use a long stem uncemented implant with a distal interlocking screw option. I would hesitate to use cement as long-term results can be poor. The femoral bone surface in revisions is often sclerotic, hard and resistant to cement interdigitation. Using cement after an osteotomy may compromise osteotomy healing.

[y] The examiner wasn't happy with the candidate just saying 'B_2' he wanted a more comprehensive account.

[z] Not always possible to obtain old medical records.

[aa] There are a lot of facts to cover in a short space of time but the score 8 candidate will manage it (usually without coming across too rushed for time) – they just keep talking.

Aseptic loosening of THA

Wear debris

The generation of particulate debris after THA occurs as a result of two processes:

- Wear
- Corrosion

The fundamental mechanisms of wear include adhesion, abrasion and fatigue. Wear debris sources include PE, cement and metal particles. PE-bearing surfaces are thought to be the major factor responsible for periprosthetic osteolysis and aseptic loosening in THA.

Studies have shown there is a critical size of particle. Small phagocytosible particles 0.5–10.0 μm in size are more active than large (>10 μm) or very small particles (<0.5 μm). Particles >10 μm stimulate a giant cell response with the formation of multinucleated giant cell but no osteolysis. Below 0.5 μm the particle size is too small to significantly activate a response. Irregularly shaped particles are more active than spherical particles.

Modes of wear

The mechanical conditions under which the prosthesis was functioning when the wear occurred has been termed the wear modes.

Mode 1 The generation of wear debris that occurs with motion between the two bearing surfaces as intended by the designers

Mode 2 Refers to a primary bearing surface rubbing against a secondary surface in a manner not intended by the designers. Usually this mode of wear occurs after excessive wear in mode 1. An example would be a femoral head articulating with a metal acetabular backing following wearing through of the polyethylene

Mode 3 Refers to two primary surfaces with interposed third body particles. This is known as third body abrasion or third body wear

Mode 4 Refers to two non-bearing surfaces (non-primary) rubbing together. Includes the back-sided wear of an acetabulum liner, fretting and corrosion of modular taper connections, and fretting between a metallic substance and a fixation screw. Particles produced by mode 4 wear can migrate to the primary bearing surfaces and induce third body wear (mode 3)

Table 16.11 Modes of femoral stem failure

Mode	Mechanism	Cause	Findings
Ia	Pistoning	Subsidence of stem within cement mantle	Radiolucent line (RLL) between stem and cement in zones I and II. Distal cement fracture
Ib	Pistoning	Subsidence of stem and cement within bone	RLL all seven zones
II	Medial stem pivot	Centre rotation of middle stem	Medial migration of proximal stem
III	Calcar pivot	Centre of rotation at calcar; distal toggle	Sclerosis and thickening of bone at stem tip
IV	Bending cantilever fatigue	Proximal resorption leaving distal stem fixed	Stem crack or fracture RLLs zones I and II, VI and VII

Osteolysis[bb]

PE particles (0.10–10.0 μm) generated from MoP articulations are the main culprit in the phenomenon of osteolysis. These particles activate phagocytes releasing numerous cytokines such as IL-1 and IL-6. These in turn activate the osteoblasts, which produce the receptor activator of nuclear factor kB ligand (RANKL). This attaches to the RANK receptor on osteoclasts promoting bone resorption and osteolysis. RANKL is blocked by osteoprotegerin (OPG). The RANKL : OPG ratio in the bone microenvironment determines overall bone homeostasis.

Modes of cemented femoral stem loosening[cc]

With cemented femoral implants Gruen described four modes of failure[55].

Mode 1: Pistoning behaviour

1a. A radiolucent line is seen between the stem and cement at the superolateral part of the stem. The stem is displaced distally, producing the radiolucent zone and a punched out fracture of the cement near the tip of the cement mass

1b. A radiolucent zone can be seen about the entire cement mass, often with a halo or thin line of reactive sclerotic bone about the radiolucent zone

Mode 2: Medial stem pivot

This is caused by medial migration of the proximal portion of the stem. Lateral migration of the distal tip results from inadequate superomedial and inferolateral cement support. This may produce a fracture of the cement at the midstem and a fracture of the sclerotic bone lateral to the tip of the stem.

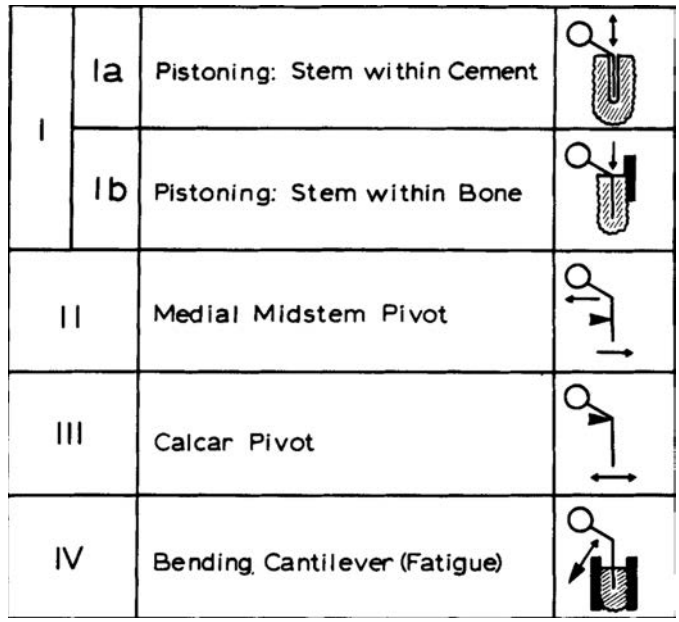

Figure 16.17 Four modes of cemented femoral stem failure according to Gruen et al.[54]

Mode 3: Calcar pivot

This is caused by medial and lateral toggle of the distal end of the stem. The distal stem lacks support and a bone reaction develops. Adequate proximal support produces a windscreen wiper type of reaction at the distal stem, with sclerosis and thickening of the cortex medially and laterally at the level of the tip of the stem.

Mode 4: Cantilever bending

This is caused by proximal loss of support of the stem while distally the stem is securely fixed. Radiolucent zones may

[bb] This is a large basic science topic which can easily be asked as a viva question. Please see www.postgraduateorthopaedics.com for additional information including classic references to know.

[cc] You may be shown a radiograph of a loose THA as a lead in into modes of cemented stem failure. The question can be chalenging for even the best prepared candidate.Two out of every 10 candidates in practice vivas are absolutely spot on anwering this question, whilst the remaining candidates tend to get the modes of failure mixed up in places. The second part of the viva question will deal with the priniples of how you would revise the THA (rehearse your answer).

Table 16.12 Definitions of radiographic loosening of cemented femoral stems according to the criteria of Harris et al.[55]

Definite loosening: • Subsidence of the component • Fracture of the stem • Cement mantle fracture • Radiolucent line between the stem and cement mantle not present on the immediate postoperative radiograph
Probable loosening: • Radiolucent line at the bone/cement interface that is either continuous or >2 mm wide at some point
Possible loosening: • Radiolucent line at the cement/bone interface between 50% and 100% of the total bone/cement interface not present on the immediate postoperative radiograph

develop proximally, medially and laterally to the stem, and may progress to stem failure.

Radiographic features of cemented femoral stem loosening

There are various radiological indicators suggestive of femoral loosening including:

- Stem migration or subsidence
- Cement or component fracture
- Osteolysis (non-linear radiolucency of >5 mm)
- Increased cement–bone interface >2 mm
- Radiolucent line between stem and cement at superolateral part of stem of >2 mm (subsidence)
- Radiolucent line between stem and cement in all Gruen zones >1 mm
- Inadequate cementation(Grade C or D according to Barrack et al.) or cement thickness (<2 mm in any Gruen zone)

In 1982 Harris et al.[56] defined criteria for the radiographic identification of loosening of cemented femoral stems into definite, probable and possible (Table 16.12). The presence of 'probably loose' and 'possible loose' were both based on the presence and extent of bone–cement radiolucencies By 1993 Harris felt that these radiographic findings were no longer valid or useful. A previous autopsy study by Harris had shown most of the radiolucent lines were secondary to adaptive remodelling rather than to disruption of bone continuity[57].

Technical problems that contribute to stem loosening

- Failure to remove adequate cancellous bone medially so that the column of cement does not rest on dense cancellous or cortical bone
- Inadequate quantity of cement
- Cement laminations and voids

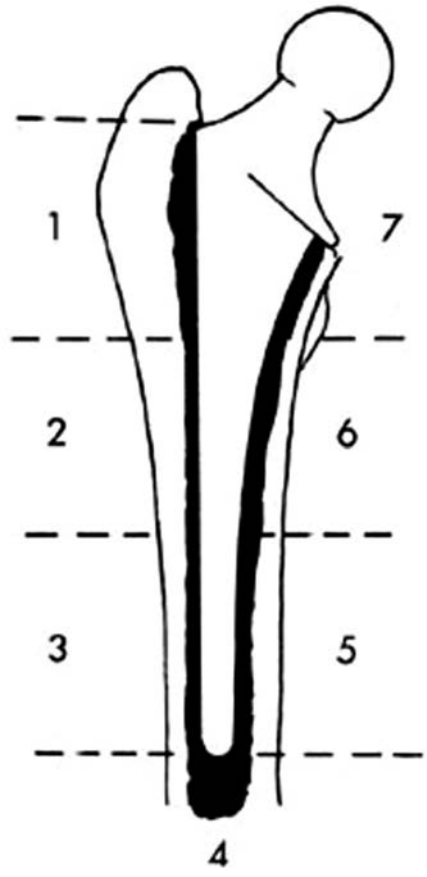

Figure 16.18 Gruen zones

- Failure to prevent cement motion while the cement is hardening
- Failure to position the component in a neutral or mildly valgus position

Radiographic features of cemented acetabulum loosening

- Bone–cement lucency >2 mm and/or progressive
- Medial migration (and protrusion) of cement and cup (into the pelvis)
- Change in inclination of the cup (indicating component migration) >5°
- Eccentric polyethylene wear of the cup
- Fracture of the cup and/or cement (rare)

Classic reference

Harris WH, McCarthy JC Jr, O'Neill DA. Femoral component loosening using contemporary techniques of femoral cement fixation. *J Bone Joint Surg Am*. 1982;64:1063–7.

Institution: Massachusetts General Hospital Boston

Harris et al. reported on the results of 171 THAs following the introduction of second generation cementing techniques.

A 1.1% incidence of definite loosening of the femoral component at a mean duration of 3.3 years follow up was noted. None of the femoral components were probably loose and 7 (4%) were possibly loose. In addition, Harris et al. proposed a radiological classification system for femoral stem loosening. Three categories were defined: Definite loosening, probably loosening and possible loosening. Second generation cementing techniques were developed in the mid 1970s to provide more reproducible interdigitation of cement into bone. This was one of the first published series of the early results following its introduction. Lower rates of loosening and increased rates of survival of the femoral component were reported compared to first generation cementing techniques

Zones of loosening

Gruen zones (femur; Figure 16.18)

Evaluation of radiographic stability is graded using the zonal analysis described by Gruen et al.[55]. The femur is divided into seven zones on the anteroposterior radiograph. One is the greater trochanter (first site of osteolysis), while seven is the lesser trochanter[dd].

Age-related expansion of the femoral canal and cortical thinning may give the appearance of a progressively widening radiolucency at the bone–cement interface. These radiolucencies do not typically have an associated sclerotic line seen in loose femoral stems.

Osteolysis typically has more irregularity with variable areas of cortical thinning and ectasia.

Classic reference

Gruen TA, McNeice GM, Amstutz HC. Modes of failure of cemented stem-type femoral components: A radiographic analysis of loosening *Clin Orthop*. 1979;141:17–27.

Institution: University of California Los Angles

Gruen et al. developed a widely used system in which the femoral component interface is considered in seven zones. These allow the location of cement fractures or lucent lines either at the cement–bone or the cement–prosthesis interface.

They also comprehensively review the four Gruen mechanical modes of cemented femoral stem failure.

The study involved a retrospective sequential radiographic evaluation of 301 patients with 389 THA with a follow up of 6 months to 6 years (mean follow up 3 years). There was radiographic evidence of loosening in 76 of the 389 hips (19.5%).

Serial radiographic examination of these 76 hips demonstrated that 56 (14.4%) had progressive femoral loosening and were classified into the 4 modes of failure. Mode Ib was the most common cause of failure in 5.1% of cases. Modes Ia, II and IV were each seen in 3% of cases. Mode III (calcar pivot) was rarely seen only in three hips (0.7%).

There were three hips with initially a mode Ia failure with a subsequent distal cement fracture with poor proximal–medial support. This progressed into mode II.

DeLee and Charnley zones (acetabulum; Figure 16.19)

The acetabulum is divided into three zones: Superior (1), middle (2) and inferior (3). Zone 3 is the most common area for osteolysis.

Incidence of loosening

With current cementing techniques there is a rate of femoral loosening of ~3% at 11 years.

Acetabular component loosening tends to be a late finding. It increases dramatically after 10 years to between 11% and 41% at 10–15-year follow-up. Cement is strongest in compression, has poor tensile strength and only moderate shear stress. Acetabular cup inclination means that mainly shear and tension forces are placed on the acetabular component

Grading of cement technique

Barrack et al.[58] have classified the quality of cement mantle radiographically into four grades (Table 16.13).

Grade C and D mantles have been shown to have greater rates of aseptic loosening but there is high interobserver variability in cement mantle grading. This grading system has been criticized since it is somewhat influenced by the amount of cancellous bone removed during reaming and broaching. When the entire cancellous bed is removed, there will often be 'whiteout' (indicating good cementing technique), and yet there will be no cancellous foothold for the cement.

It is suggested that a minimum of 2 mm of cement thickness be allowed between prosthesis and bone. The two-thirds rule states that two-thirds of the canal is displaced by the femoral stem and the other third by cement.

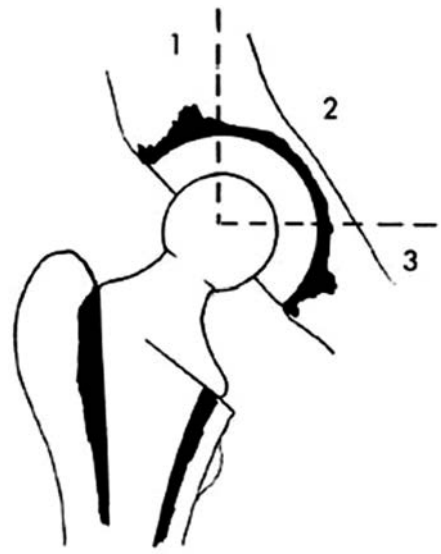

Figure 16.19 DeLee and Charnley zones

[dd] Try not to get them the wrong way round! The examiners will be happy to point this mistake out to you.

Table 16.13 Barrack et al.[55] cement grading

Grade A	Medullary canal completely filled with cement (white out) with no distinguishable border between the cement and the bone
Grade B	Near complete filling. Slight radiolucency exists bone–cement interface
Grade C1	Radiolucency >50% at the bone cement interface
Grade C2	Cement mantles have areas in which the cement thickness is <1 mm, or the prosthesis is up against bone
Grade D	Radiolucency >100% bone cement interface including absence cement distal to the cement tip. Cement mantles have gross deficiencies or multiple large voids

Mulroy et al. report a thin (<1 mm) femoral cement mantle and defects in the cement mantle are associated with early loosening[59]. Jasty et al. found that cement voids and stem abutment against the femur (indicating an inadequate cement mantle) were associated with loosening[60]. A mantle defect where the prosthesis touches bone creates an area of concentrated stress and is associated with higher wear rates.

In the early 1970s, a contradictory cementing technique was introduced that involved implantation of a canal-filling femoral component in a line-to-line manner associated with a thin cement mantle This principle has given excellent long-term clinical and radiological results and has been named the 'French paradox'. An explanation of this phenomenon has been recently provided by in vitro studies which showed that a thin cement mantle in conjunction with a canal-filling stem was supported mainly by cortical bone and was subjected to low stresses.

Classic reference

Barrack RL, Mulroy RD Jr, Harris WH. Improved cementing techniques and femoral component loosening in young patients with hip arthroplasty: A 12-year radiographic review *J Bone Joint Surg Am*. 1992;74:385–9.

Institution: Massachusetts General Hospital Boston

Barrack and colleagues report a series of 50 THAs in 44 patients <50 years at mean follow up of 12 years. They attributed a diminished incidence of femoral aseptic loosening (2%) to improved second-generation cementing techniques. Adequate femoral cement grades (Barrack grade A or B) were achieved in 100% of cases.

Barrack et al. proposed a four-scale femoral cement mantle grading system to assess femoral cementation on immediate postoperative films. This classification was subsequently modified by Mulroy with C being divided into C1 and C2. Harris et al have shown the strongest predictive factor for mechanical failure is excessively thin mantles or mantles with defects (C2 grade) as opposed to mantles with single or multiple voids (C1 grade).

Examination corner

Adult elective orthopaedics oral 1

The candidate was shown a radiograph demonstrating gross aseptic loosening of a THA.

CANDIDATE: This is a difficult and challenging situation. Postoperative film would be useful for comparison, to see whether these changes are progressive or were present immediately postoperatively. A lateral radiograph would also be useful. I would be very concerned about catastrophic failure occurring in the near future and regard the case as urgent. Looking at the acetabular side there are AAOS grade 3 bone loss changes present *(brief pause)*. The cup has rotated and is obviously loose.

The candidate briefly mentioned the "AAOS classification of bony acetabular defects" to the examiners. He was hoping they would ask him about this classification system. The candidate thought this wasn't too obvious but some examiners are much cleverer than they appear. They simply ignored it and let him continue talking.Classification systems aren't essential to know for the exam and examiners generally don't ask you about them but by the same token they are helpful to know.

Adult elective orthopaedics oral 2

Small 5 × 7 inch postoperative photograph shown of an AP radiograph pelvis.

EXAMINER: This patient had revision surgery to his right hip performed with impaction bone grafting of the femur 3 months previously. What do you think of the radiograph?

CANDIDATE: My mind went blank. There was nothing very obvious to say about the radiograph. I mumbled something nonsensical.

EXAMINER: The femoral stem has subsided and sunk into the femur. This is one of the worries of impaction grafting along with the increased risk of infection.

EXAMINER: What type and size of bone graft would you use for impaction grafting?

CANDIDATE: Small particles.

EXAMINER: The term you are looking for is 'crouton size' particles. Let us move on and talk about types of bone graft. Can you name the various types of bone graft that exist?

CANDIDATE: Autograft is from the same person, allograft from another person, xenograft from a different species and isograft from identical twins. Or they can be described in terms of tissue composition, either cortical, cancellous, corticocancellous and osteochondral, etc.

EXAMINER: Which graft is best in terms of incorporation?

CANDIDATE: Cancellous autograft

EXAMINER: Why?

CANDIDATE: It is the best in terms of osteoconductive, osteoinduction and osteogenesis potential.

EXAMINER: What do you mean by these terms that you have just used?

CANDIDATE: I went on to describe fairly well osteoconductive, osteoinduction and osteogenesis.

Impaction grafting using fresh–frozen morsellized allograft is effective for both acetabular and femoral reconstruction. There are problems with cost, supply and potential infection. There are worries about the biomechanical variability of donated bone owing to its mode of preparation and its biological variability. Concerns with transmission of infection from donor to recipient have led to irradiation of bone allograft as a means of sterilisation. The typical gamma radiation dose for bone used in impaction grafting is 25 kGy. There are worries that irradiation may affect the mechanical properties of the graft and its long-term incorporation. A dose of 25 kGy does not greatly affect the mechanical properties of bone, but increasing the dose has a detrimental non-linear effect on bone strength and bone incorporation. Irradiation has been shown to impair osteoconductive capacity of bone graft. It is postulated that oxidation of lipids present in the marrow renders them cytotoxic to osteoblasts. Washing of irradiated graft removes fat, which may diminish the properties related to oxidized lipids.

Tight packing of allograft chips into the proximal part of the femur to obtain initial implant stability is crucial for long-term survival of the reconstruction. Risks include subsidence (50%), high postoperative fracture risk, perforation (14%, manage with either cable, mesh or strut graft) and it is technically difficult, with cost issues.

Adult elective orthopaedics oral 2: X-ray of massive subsidence after impaction grafting

- What is impaction grafting?
- Complications?

Adult elective orthopaedics 3: Aseptic loosening of THA

CANDIDATE 1: This is an AP radiograph of the pelvis. It shows a THA in situ but I am not familiar with the type of prosthesis used. Turning towards the femoral component what we can see is a straight stem prosthesis with a modular head. The head size would appear to be large, possibly 28 mm. The tip is blunt and there is no cement plug, suggesting first-generation cementing techniques. There are trochanteric wires present, which would be in keeping with a trochanteric approach.

We can see lucencies in Gruen zones I, II, IV and VII. There is bony sclerosis around the tip of the prosthesis and the tip is in contact with the lateral cortex of the bone. This is suggestive of Gruen mode II failure of the medial stem pivot.

A lateral radiograph of the hip would be helpful at this stage.

Turning towards the acetabular component there are lucencies present in all three DeLee and Charnley zones. A postoperative radiograph would be helpful to decide whether these changes were present postoperatively or are progressive. There is obvious wear of the acetabular cup as shown by superior migration of the femoral head. There are also significant acetabular bony defects present, probably AAOS type III, a combination of segmental and cavitatory defects.

CANDIDATE 2: This radiograph demonstrates gross aseptic loosening of a THA of both the acetabular and femoral components.

COMMENT: The diagnosis is obvious but candidate 1 has given a more complete and comprehensive answer. Answering this type of oral question is rather like passing your driving test, demonstrating to the examiner that you are looking in the mirror before you pull out. Rather than the examiners 'assuming' that candidate 2 'knows their stuff', candidate 1 has put them at ease by demonstrating that 'they do indeed know what they are talking about'.

Adult elective orthopaedics oral 4: Radiograph of broken femoral prosthesis

CANDIDATE: This is an AP radiograph of the pelvis. It shows a broken femoral prosthesis. This is Gruen mode IV failure, a bending cantilever failure. It is the most common form of failure[ee]. The other modes of failure are pistoning, either the stem within cement or the stem within bone, medial stem pivot and calcar pivot.

EXAMINER: What do you think is happening at the neck of the prosthesis? *(It was obvious osteolysis)*.

CANDIDATE: Bone resorption is taking place here and this has led to cantilever failure. Bone resorption is also present superolaterally. The acetabular component is loose also. There are lucencies in DeLee and Charnley zones 1, 2 and 3.

EXAMINER: What do you think of this area here? *(Large lucency in acetabular bone superiorly.)*

CANDIDATE: There is probably a segmental and possibly also rim defect in the acetabulun caused by osteolysis. Bone graft will be needed when revising the cup.

Adult elective orthopaedics oral 5

CANDIDATE: This is an AP radiograph of the pelvis, which demonstrates Gruen mode IV failure, bending cantilever fatigue with complete stem fracture of the femoral prosthesis. This mode of failure is caused by proximal loss of support of the stem while distally the stem is securely fixed.

Distally the stem appears to be well fixed, proximally it is loose within the cement mantle with lucent lines particularly in Gruen zones I, II, VI and VII. progressing to stem failure.
Other modes of failure include mode IA subsidence of the stem in the cement mantle, and IB subsidence of cement mantle and stem[ff].

Mode II failure is medial stem pivot and mode III failure is calcar pivot.

Looking at the acetabulum there appears to be lucencies in DeLee and Charnley zones 1–3, suggesting that the acetabular component is also loose.

[ee] This was actually incorrect but we don't think the examiners realized this. Mode Ib is the most common cause of failure in 5.1% of cases.

[ff] Not asked for but the examiners seemed pleased I had continued on.

EXAMINER: You need to look a little bit more closely on the acetabular side, especially superiorly.

CANDIDATE: There is a large lytic defect superiorly.

EXAMINER: There is obviously a large amount of bone loss superiorly and one would also have to revise the acetabular component at the time of surgery using bone graft.

Tell me what you know about types of bone graft?

The other fork in the road with this viva involves the examiner asking how you would manage this condition. Your answer would involve reciting a standard (ideally rehearsed) answer for how you would a perform revision hip arthroplasty along with a tailored response to the specific clinical situation presented[gg]

CANDIDATE: I would take a full history and clinical examination.

I would counsel the patient regarding the natural history of the condition and recommend revision is undertaken on an urgent basis as the situation is likely to deteriorate and may lead to catastrophic periprostatic fracture.

I would order blood tests to check for inflammatory markers – FBC, CRP and ESR to exclude prosthetic infection.

I would obtain a lateral radiograph of the hip/prosthesis. The femoral bone stock appears satisfactory, but if there was any doubt I would order a CT scan.

I would obtain the original operative notes to check what surgical approach was used and which implants were inserted. The acetabular cup is loose, and there is evidence of excessive polyethylene wear on the preop radiographs and so needs to be revised

I would position the patient laterally; give prophylactic antibiotics and approach the hip via a posterior approach.

After dislocating the THA I would attempt to remove the proximal stem and cement knocking it out with a mallet after using a flexible osteotome to disturb the cement implant interface.

I would plan an extended trochanteric osteotomy as access to the distal stem and cement mantle may be difficult. I would remove the remainder of the cement, the distal stem and the cement restrictor with a combination of osteotomes, reamers and the OSCAR system.

I would repair the ETO using a cerclage cable system.

I would then plan to implant a long-stem modular, uncemented, tapered, fluted revision prosthesis, aiming for a good distal press-fit after reaming the femoral canal.

If this cannot be achieved further options would be either a distally locking THA stem or a long cemented femoral stem although I would be concerned about introducing cement into the osteotomy site which would compromise osteotomy healing. In addition, the results of cemented femoral revision stems have traditionally been disappointing.

I would remove the acetabular component and cement using osteotomes. I would assess the remaining bone stock and decide upon either a further cemented or uncemented acetabular component. My preference would be for an uncemented tantulum multihole revision component using multiple screws for additional fixation and femoral head allograft to restore bone stock. I would attempt to upsize the femoral head size if possible. If I was significantly concerned about the risk of dislocation but the cup was solidly fixed I would perform a cement in cement acetabular revision to allow for upsizing the femoral head.

Adult elective orthopaedics oral 6

How would you investigate and manage a patient with early loosening of a THA?

The question is essentially about excluding infection as a cause of early loosening.

Basic science oral 1: Retrieval of THA – Discussion on reason for failure

- Osteolysis
- Why would you not have a complete cement mantle? Canal too small, prosthesis too big, mal-alignment and poor surgical technique

Basic science oral 2

Candidate was given a worn plastic acetabular cup and asked to comment.

- Methods to reduce oxidation of highly cross-linked PE. Discussed post-irradiation remelting to quench residue free radicals and addition of antioxidants such as vitamin E to PE powder prior to consolidation into a solid state to improve the resistance to oxidation
- Highly cross-linked PE has a susceptibility to crack propagation and failure. Inferior mechanical properties: Reduced Young's modulus, yield strength, fracture toughness, fatigue crack resistance
- Discussion on aseptic loosening of THA, wear particles, sources, etc. then followed

EXAMINER: How do you assess wear of a THA at follow-up clinic?

CANDIDATE: My mind went blank and I waffled on about nothing in particular. I thought the examiners were looking for a complicated answer. Keep things simple, all they were looking for was comparison of the degree of migration of the femoral head into the acetabular component on serial radiographs. We eventually got there but I didn't do too well with it. This is basic stuff that can catch you out if you are not careful.

Adult elective orthopaedics oral 7

I was shown an x-ray of a THA with severe loosening of the femoral compartment in all Gruen zones. The examiners asked me what the mechanism of loosening was. I told them it was pistoning of the prosthesis and the cement mantle affecting all seven zones. They asked me to enumerate all the Gruen zones and describe the different modes of loosening one can get.'

[gg] Don't forget to look at the acetabulum – Is it loose and requires revision or well fixed and can be left alone.

'The examiners then asked me what I would do with the THA. I said I would plan for revision surgery. They asked me how I would go about this. I started off with history, examination and investigations. I mentioned performing a full blood count, CRP and ESR. The examiner asked me whether I would do a bone scan. I said I would prefer a white cell scan as a bone scan itself was of doubtful value. I then mentioned that I would like to perform an aspiration of the hip to rule out infection, which the examiners were happy with. They asked what the management would be if the aspirate is negative. I said I would offer revision THA. They asked what information I would give the theatre staff. I said I would get the old notes to find out what type of prosthesis was used and also inform the theatre staff about bone grafting and also the different types of cabling devices and osteotomy plates available in case we have to do an extended trochanteric osteotomy. I told them I would then proceed with an uncemented long femoral stem. The examiners seemed satisfied with the answer.'

Design features of THA components

Femoral component design

Femoral stems can be cemented or uncemented. Some design features are common to both

Medial offset or femoral offset: Perpendicular distance between the centre of the femoral head and the long axis of the distal part of the stem. Primarily a function of stem design. Inadequate restoration leads to increased joint reaction force, bony impingement and dislocation while excessive offset can lead to stem fracture or loosening.

Neck length: Measured from the centre of head to base of collar.

Neck shaft angle: Typically about 135°.

Longitudinal slots/grooves: Improves rotational stability of the stem within the cement mantle. Decreases stress shielding, and increases the interlock between the stem and the cement.

Cement centralizer: Provides a more uniform cement mantle.

Ratio of femoral head diameter to the femoral neck diameter: If increased there is a greater primary arc of motion.

Stem cross-section (oval or square): The cross-sectional shape influences the distribution of cement within the femoral canal, the rotational stability of the implant and the stress distribution within the cement mantle.

Stems with an oval cross-section have a better fit within the medullary canal and can occupy more of the cavity, leaving less room for cement and cancellous bone. More rectangular cross-sections such as the Exeter (Stryker) are limited in size by their contact against the inner cortex of the oval cross-section of the medullary canal.

Overall shape: Straight (curved only in the frontal and not sagittal plane) or **anatomica**l (designed to fit the sagittal intramedullary anatomy).

Anatomically shaped components are designed to fit the overall shape of the femur in a better way allowing for better centralisation of the stem and a more even thickness of the cement mantle. Compared with symmetrical stems, anatomical stems generate different strains within the cement mantle because of their specific shape. They are of the composite-beam type since their shape limits the subsidence required to achieve a stable position.

All stems are now straight distally but excessive proximal curve with cemented stems should be avoided to prevent cement overhanging proximally and laterally which may be problematic if revision is required

Surface finish (matt or polished): Polished stems are preferred with loaded-taper design since they allow stepwise subsidence to a stable position, with the associated micromovement producing less metal and cement debris at the cement–stem interface. In the composite–beam prostheses, roughening the surface to increase the cement–stem bonding enhances stability. Changing the Exeter stem from a polished to a matt finish resulted in a much higher failure rate.

Collar or collarless: May promote direct transfer of load from the implant to the medial cement mantle and/or the bone of the medial femoral neck. Direct collar-bone contact can unload the vulnerable proximal cement mantle. A collar may reduce tensile stresses in the stem and reduce overall migration. A collar may control insertion, especially when the stem is undersized compared with the broach. May be useful as an aiming device for determining version and as a stop point while inserting the stem. It may prevent the stem from 'settling' during cyclic loading, and does not avoid micromovement of the stem. Additionally in the long term it does not prevent absorption of the calcar.

Shape of the tip: Tapered or blunt.

Modularity (non-modular, modular): Modular heads allow for adjustment in neck lengths.

Head size

This influences the range of motion, wear and dislocation.

22.25 mm head

- Low frictional torque
- Higher rate of dislocation
- Greater linear wear and creep

32 mm head

- Greater stability and range of movement but increased volumetric wear
- Less space is left for the acetabular component, resulting in a thinner layer of polyethylene

28 mm head

The 28-mm head is a reasonable compromise as it produces the least linear wear and volumetric wear rates similar to the 22-mm head.

Table 16.14 Classification system of cementless femoral stem designs

General Category	Type	Geometry	Description	Location of fixation
Straight stems				
Tapered proximal fixation	1	Single wedge	Narrows medially-laterally. Proximally coated. Flat stem, thin in anterior–posterior plane	Metaphyseal
Tapered proximal fixation	2	Double wedge, metaphyseal filling	Narrows distally in both medial–lateral and anterior–posterior planes. Wider than type 1. Fills metaphyseal region	Metaphyseal
Tapered proximal fixation	3A	Tapered, round	Rounded tapered conical stem with porous coating at proximal two-thirds	Metaphyseal–diaphyseal junction
Tapered distal fixation	3B	Tapered, splined	Conical taper with longitudinal raised splines	Metaphyseal–diaphyseal junction and proximal diaphyseal
Tapered distal fixation	3C	Tapered, rectangular	Rectangular cross-section with four-point rotational support in metaphyseal–diaphyseal region	Metaphyseal–diaphyseal junction and proximal diaphyseal
Distally fixed	4	Cylindrical, fully coated	Extensive porous coating. Proximal collar to enhance proximal bone loading and axial stability	Primarily diaphyseal
Modular	5		Metaphyseal and diaphyseal components prepared independently	Metaphyseal and diaphyseal
Curved, anatomic stem	6		Proximal portion is wide in both lateral and posterior planes. Posterior bow in metaphysis, anterior bow in diaphysis	Metaphyseal

36 mm head

- Increases impingement free ROM
- Reduced dislocation risk
- Increased torsional forces at the head–neck junction
- Increased trunion wear and corrosion

Cementless femoral component

Uncemented stems rely on biological fixation and are mainly two types; bone ingrowth and bone ongrowth designs. Ingrowth is the formation of bone inside a porous surface whereas ongrowth refers to bone growth over a roughened surface. Ingrowth requires a pore size between 50 μm and 400 μm and the percentage of voids within the coating should be between 30% and 40% to maintain mechanical strength.

Ingrowth surfaces include sintered beads, fiber mesh, and porous metals. Sintered beads are microspheres of either cobalt chromium or titanium alloy attached by the use of high temperatures. Fiber mesh coatings are metal pads attached by diffusion bonding. Porous metals have a uniform three-dimensional network, with high interconnectivity of the voids and high porosity (75–85%) compared with that of sintered beads and fibre metal coatings (30–50%).

Ongrowth surfaces are created by grit blasting or plasma spraying. An abrasive grit blasted surface creates microdivots on the surface, which are of similar size to pores in porous-coated designs. The depth of the divot (distance from peak to valley) is referred to as the surface roughness of the stem. Bone grows into the divots achieving biological fixation. Plasma spraying involves mixing metal powders with an inert gas that is pressurized and ionized, forming a high-energy flame. The molten material is sprayed onto the implant, creating a textured surface.

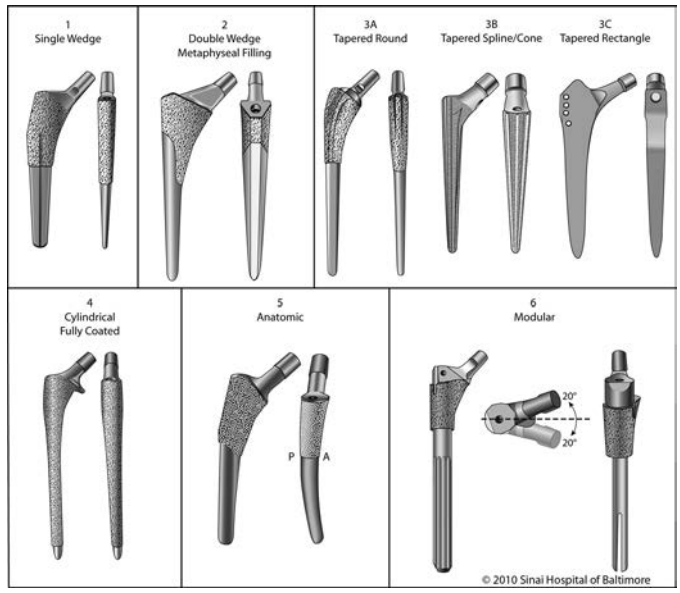

Figure 16.20 Classification of the cementless femoral stem designs

It is important in both designs to achieve early stable press fit or frictional fit fixation to allow for bony osseointegration. Press fit fixation is achieved by under-reaming of the bone. When the prosthesis is wedged in, compression hoop stresses stabilise the implant to achieve a rigid fixation. On the other hand, frictional fit is achieved by line-to-line reaming of the bone. The rough surface of the prosthesis provides enough resistance to motion, which achieves implant stability when it is impacted into its final position. This is also called scratch fit or interference fit. Stems can be either proximally porous coated or fully porous coated. Advocates of the use of proximal coated stems argue that this decreases the incidence of stress shielding and loss of proximal femoral bone density as a result of proximal femoral loading (Hoek's law).

Berry et al.[61] classified cementless stems into four categories based on geometry and fixation: (1) wedged-shaped metaphyseal filling implant; (2) single wedge shaped implants that are relatively thin in an anterior to posterior direction; (3) tapered stems that are designed to get most fixation at the metaphyseal-diaphyseal junction; (4) distally cylindrical or fluted stems designed to obtain initial fixation in the diaphysis.

Khanuja et al.[62] further modified these categories into six types based on shape, amount of osseous contact and progression of stem fixation from proximal to distal (Table 16.14 and Figure 16.20).

Type 1 stems also called single wedge are designed to engage metaphyseal cortical bone in one plane: Medial to lateral . They are flat and thin in the anterior-posterior plane.

The component narrows proximally, primarily in the mediallateral plane, and tapers distally. Initial stability is by wedge fixation in the medial-lateral plane or three-point fixation along the stem length. The broad flat shape achieves rotational stability.

Type 2 (double wedge) stems obtain proximal cortical contact in two planes: Anterior–posterior and medial-lateral. Diaphyseal engagement is necessary to enhance rotational stability.

Type 3 stems achieve fixation in the metaphyseal-diaphyseal junction and proximal diaphysis. They have a long consistent taper in both the medial-lateral and anterior posterior plane. They are divided into three subgroups on the basis of their shape and means of fixation.

Type 3A is tapered and round. Most have porous coating on the proximal two-thirds and obtain three-point fixation. Proximal fins or ribs may be added for rotational stability.

Preparation requires reamers distally and broaches proximally.

Type 3B stems have a conical taper with longitudinal raised splines to provide rotational stability and fixation. The profile of these stems is very narrow providing the ability to change version in difficult cases. The preparation for these stems is done by conical reamers.

Type 3C stems are tapered, rectangular and conical, grit-blasted across its entire length.

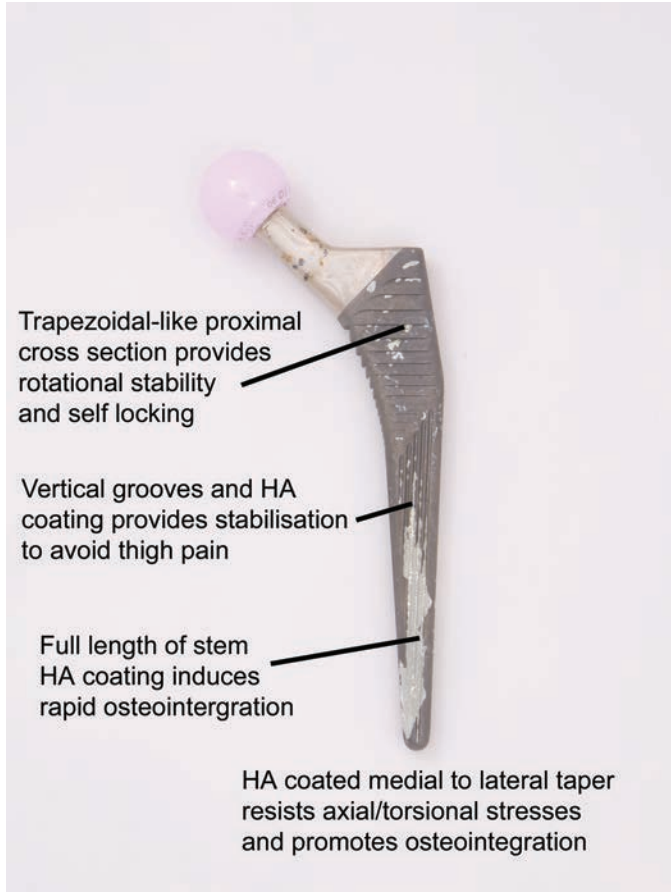

Figure 16.21 Corail Standard Offset 135° neck angle, collarless uncemented femoral stem. The 155 µm hydroxyapatite coating on the grit-blasted surface of the corail stem induces rapid osteointegration.

Type 4 is cylindrical and fully coated along the entire prosthesis. A proximal collar enhances axial stability and transmits forces to the calcar.

Type 5 is modular which allows independent preparation and separate components for the metaphysis and diaphysis.

Type 6 prostheses are curved anatomic stems that match the proximal femoral endosteal geometry. Stability is achieved through metaphyseal fill and the distal curve.

Some implants combine ingrowth and ongrowth technology. An example of an ongrowth coating material is hydroxyapatite (HA); $Ca_{10}(Po_4)_6(OH)_2$. It is plasma sprayed either directly on the implant or onto a porous coating.

NJR data from England and Wales show that the Corail® stem (Figure 16.21) is the most common uncemented stem used in primary THA. It is made of forged titanium alloy (TiAl6V4). It is a straight implant, with a quadrangular cross-section. The trapezoidal-like proximal cross-section provides rotational stability and self locking in the metaphyseal area. The distal portion has a tapered design, to produce a stiffness gradient and to avoid medullary canal blocking. Macro-textural features (horizontal and vertical grooves) enhance primary mechanical

stability, which may be further augmented by the use of an optional collar. The HA coating is applied to the entire stem in order to prevent the release of metal ions, to provide for maximum osseointegration at the interface and to prevent the interposition of a fibrous membrane around the distal portion of the stem. HA coating is applied using an atmospheric plasma spray process. The thickness of the ceramic layer is 150 μm.

HA is osteoconductive and enhances growth of mineralised bone onto the implant essentially by providing bone with its mineral phase substrate. Theoretically, its osteoconductive properties lead to bi-directional gap closure (from bone to prosthesis and from prosthesis to bone), giving more rapid closure. Studies have demonstrated no difference in the clinical and radiographic outcomes when stems with hydroxyapatite were compared with the same stems without hydroxyapatite. There have been cases of HA coatings delaminating from the prosthesis – Usually if it is applied too thickly.

Other concerns include excessive third body abrasion wear if coating fragments are present within the joint space. However, this theoretical risk has never been proven. Fragments released after coating degradation remain in the immediate environment of the intramedullary part of the stem and do not migrate into the surrounding soft tissues or in the joint cavity. There have been concerns that HA may induce osteolysis but this is attributable to migration of other particles. Complete coating resorption is a recognised occurrence with HA. Extraction of a well-osseointegrated implant can be a challenging procedure. However, a specific strategy and technique using dedicated instrumentation will considerably lessen the risks and complications associated with prosthesis removal.

Classic reference

Engh CA, Bobyn JD, Glassman AH. Porous-coated hip replacement. The factors governing bone ingrowth, stress shielding, and clinical results *J Bone Joint Surg Br*. 1987;69-B:45–55.

Engh et al. presented a comprehensive review of factors controlling bone ingrowth and biological fixation of uncemented femoral stems.

The authors defined the criteria for reporting definite femoral stem loosening when reporting survivorship analysis of uncemented THA.

The paper also presented the first classification system for reporting stress shielding which has stood the test of time and is still widely used

1. First degree – A slight rounding off of the proximal medial edge of the cut femoral neck
2. Second degree – A rounding off of the proximal medial femoral neck combined with loss of medial cortical density at level 1 on the AP radiographs
3. Third degree – More extensive resorption of the cortical bone extending from level 1 into level 2
4. Fourth degree – Severe resorption of cortical bone extending below levels 1 and 2 into the diaphysis

Radiographs were also evaluated for cortical hypertrophy, pedestal formation, and spot welds. A bony pedestral is bone accumulation within the medullary canal below the tip of a mechanically loose stem. The bony pedestral attempts to stop the stem from sinking further down the medullary canal.

The authors also proposed radiographic features of uncemented femoral component stability.

- A **stable implant** demonstrated no migration and spot welds. A spot weld indicates stable osteointegration
- **Stable fibrous ingrowth** occurred when an implant showed no progressive migration, sclerotic lines around the porous surface and less atrophy of the medial femoral neck than observed when bone growth occurs
- An **unstable implant** was defined as one with definite evidence of progressive subsidence or migration of the implant. Increased cortical density and thickening typically occur beneath the collar and at the end of the stem

Cemented femoral component

Cement fixation relies on microinterlock with endosteal bone. It is important to point out that cement fatigues with cyclic loading and this starts in areas of stress points in the mantle (such as defects where the prosthesis touches bone). To achieve optimal cement fixation, the following strategies are employed:

- Decreasing porosity of cement by vacuum mixing
- Pulsatile lavage of bone prior to cementing to produce clean dry trabeculae, which improves interdigitation
- Pressurisation to improve microinterlock
- Use of a stem centralizer to achieve a uniform mantle thickness and eliminate defects

Two main femoral stem designs philosophies are used in cemented THA:

- Taper slip, 'force-closed' fixation
- Composite beam or 'shaped-closed' fixation

A loaded taper design stem (double or triple taper) is polished and collarless. This allows some subsidence within the mantle when axial load is applied. The viscoelastic properties of polymethylmethacrylate (PMMA) will result in radial forces being generated as a result of axial loading and wedging of the stem within the mantle. These are transferred to bone as hoop stresses, which enhance fixation and stability of the stem. A distal centralizer is used to facilitate subsidence of the stem to a stable position without creating excessive stresses in the distal cement mantle. Examples of taper loaded stems include the Exeter stem (Stryker, Kalamazoo, Michigan, USA), CPT stem (Zimmer, Warsaw, Indiana, USA) and C stem (DePuy, Warsaw, Indiana, USA).

The second design is referred to as composite beam. This has a matt finish with a roughened surface. This allows for rigid bonding between the stem and cement. Most of these stem designs have a collar to enhance stability and to eliminate subsidence within the mantle. An example is the Stanmore stem (Biomet, Bridgend, UK).

Radiostereometric analysis (RSA) has shown that loaded-taper and composite-beam stems migrate differently over time. In the first year of implantation, loaded-taper stems show an initial subsidence between 0.9 mm and 1.4 mm and retroversion between 0.4 mm and 0.5 mm. After the initial year, stems

tend to stabilise. Initial migration seems to be independent of the type of cement, its viscosity and the thickness of the cement mantle. The cement mantle surrounding these stems migrates only slightly within the femur, which does not appear to compromise long-term results.

Stems relying on the composite-beam principle have more initial stability (especially longitudinal) with migration between 0.1 mm and 0.5 mm during the first year. Migration into retroversion during the first year is usually between 0.28 mm and 0.8 mm, but is sometimes $\geq$1.0 mm and can even be as much as 2.0 mm. In some instances migration at the cement–bone interface has also been seen. Both factors are worrying since excessive and continuous migration is predictive of failure.

Acetabular component

Metal-backed cemented acetabular sockets have higher failure rates compared to all PE cups. Elevated posterior lip designs are thought to reduce the risk of dislocation. Flanges on the acetabular components are designed to improve pressurisation of cement.

Cementless design

The initial stability of an implant is achieved by mechanical interlock with the host bone. This is then converted to a long-term secondary stability by the ingrowth/on growth of a stable biological interface.

Attempts to improve bone ingrowth into metal implants have centred on either porous coating and/or coating with hydroxyapatite. The optimum thickness of hydroxyapatite for coating is approximately 50 μm. A pore size of 50 μm is accepted as the minimum for bony ingrowth with an ideal pore size of between 50 μm and 400 μm to enhance bone ingrowth. Hydroxyapatite stimulates bone growth onto a prosthesis, achieving osseointegration and facilitating a biological bone between implant and bone. This biological bone can provide mineralized continuity around the prosthesis, with a sealing effect and a reduction in large early migration.

Threaded designs

Threaded cups can be either non-porous or porous coated.

Non-porous coated threaded cups rely on a mechanical interlock between the acetabular bone and the implant threads for both initial stability and long-term fixation. They have fallen out of favour in recent years as a number of studies have shown unacceptable high early revision rates.

Hemispheric designs

The majority of acetabular components are hemispherical and are available in incremental sizes.

Initial fixation of the acetabular component is usually accomplished by either a press-fit technique or a line-to-line fit. The press-fit technique involves the bone prepared being sized slightly smaller in diameter than the actual component. A line-to-line fit involves preparing bone to the same size as the implant and securing with screws.

Surgical tip: If an intraoperative fracture occurs during placing an uncemented shell in the acetabulum, assess stability of the shell. If this is stable, add screws. If unstable, remove the shell, stabilise/fix the fracture then reinsert the shell with screws.

Hybrid THA

Hybrid THA combining a cemented stem and a cementless socket was introduced because of the high rates of failure of cemented THA in young patients, particularly on the acetabular side.

Data from the Swedish hip registry have reported 4–11% aseptic loosening rates for hybrid THA. This is a less popular option today as initial enthusiasm from a few years ago has died down. There are fewer complications when using an cemented femoral stem in osteoporotic bone.

Examination corner

Basic science oral 1: Design features of THA

The cemented femoral stem can be either polished, collarless and tapered (loaded taper) or non-polished (matt finish), collared and rectangular (composite-beam). Stem subsidence occurs with loaded taper stem designs, the initial subsidence occurring because of cement creep allowing ongoing radial compression of the cement, sealing the stem–cement interface.

Basic science oral 2: Materials properties of ceramic on ceramic bearing surfaces

- Bioinert and minimal inflammatory response. Excellent abrasive resistance, low coefficient friction, high impact strength, chemical resistant, high Young's modulus elasticity, excellent wetability properties and absence of metal ion release
- Superior wear resistance provides an excellent choice for young and active patients
- The 6 Vs
  1. Very strong
  2. Very stiff
  3. Very hard
  4. Very biocompatible
  5. Very reduced volumetric wear debris (compared with other bearings)
  6. Very brittle

Ceramic-bearing surfaces are associated with audible squeaking. The incidence of squeaking has been reported to vary widely, from 1% to 20%, with 3% being a generally accepted figure.

A definite aetiology for squeaking in ceramic on ceramic bearing hips remains elusive and controversial.

The cause is both complicated and multifactorial, with studies reporting mixed findings with factors such as cup

position, patient age, height and weight. Recent work has implicated stem alloys, stem geometries and neck geometries as causative factors. The V-40 neck and titanium-molybdenum-zirconium-iron stem are particularly prone to cause squeaking.

The current explanation for squeaking is based on the visible wear stripe concept. Retrieval analysis suggests a heavy wear pattern during activities at the extremes of motion such as rising from a seated position or other high flexion activities. On these occasions, edge loading between the ceramic head and the posterior rim of the ceramic cup occurs. Squeaking can also occur with metal on metal articulations.

Chip fractures and cracks can occur at the rim of the acetabular liners, particularly when components are malpositioned.

Adult elective orthopaedics oral 1

I was shown an AP radiograph of an uncemented THA and asked to comment. The hip was uncemented which caught me off guard initially.

I used the zones described by Gruen and DeLee and Charnley to assess the location and extent of radiolucent lines and osteolysis. I mentioned that I would like to assess serial radiographs of the hip. Periprosthetic cystic or scalloped lesions with a diameter exceeding 2 mm not present on immediate postoperative films would be highly suggestive of osteolysis

I mentioned that there were radiolucent lines in Gruen zones III, IV and V only. There was no evidence of endosteal bone formation (spot welds) at the bone interface. The examiner pointed out pedestal formation at the stem tip suggestive of implant loosening which i had missed but in retrospect was quite obvious.

I pointed out that serial radiographs should be reviewed for femoral neck fretting, which would be suggestive of impingement of the metal neck on the acetabular shell. The radiograph showed a rounding off of the medial edge of the resected femoral neck

There was no evidence of calcar resorption of the femoral neck which, if present, suggests significant stress shielding. I mentioned checking for femoral stem migration on serial radiographs (measured as the difference between the shoulder of the implant and the greater trochanter).

Effective joint space was then discussed.

'The effective joint space has been defined as all periprosthetic regions that are accessible to joint fluid and, thus, particulate debris. An inflammatory response generated by osteolysis produces an increased hydrostatic pressure that allows for dissemination of particulate debris within the effective joint space. Once wear particles are generated they will follow the path of least resistance and will, thus, find their way between the cement–bone interface or implant–bone interface in the case of uncemented designs.

It is important to achieve an adequate seal to prevent this path for particulate debris. This can be achieved by either a complete cement mantle or a circumferentially proximally coated cementless implant.'

With uncemented designs, an intact mechanical barrier at the prosthesis–bone interface may reduce the ingress of particular debris along relevant interfaces and retard periprosthetic bone loss.

Metal on metal (MoM) hip articulations

Introduction

This is still a controversial topic ideal for the FRCS (Tr & Orth) exam.

There is recent ongoing debate concerning unexplained hip pain, early failure and formation of pseudotumours. NJR data have shown an unexplained high early rate of failure for all designs of MoM hip resurfacings and total hip replacements.

The 11th annual report in 2013 detailed that only 1.1% of hip resurfacing and 0.9% of hip arthroplasty involved a MoM-bearing surface. General recommendations are that MoM THA give poor implant survival compared with other options and should not be implanted. Whilst not contraindicated at best there is a limited role for MoM hip resurfacing procedure.

Resurfacing arthroplasty

The ideal candidate for an MoM hip resurfacing arthroplasty is a relatively young man with normal anatomy and primary osteoarthritis. For the exam be very careful about suggesting a MoM hip resurfacing procedure. This surgery should ideally only be performed in a small number of highly selective patients by a hip surgeon with extensive resurfacing experience performing a minium number of procedures per year and having peer reviewed resurfacing results published in the literature.

Contraindications to resurfacing

- Femoral head too deformed, e.g. SUFE
- Acetabular morphology unsuitable, e.g. severe DDH
- Narrow femoral neck (risk of notching and fracture)
- Chronic renal failure (absolute)
- History of metal hypersensitivity (e.g. jewellery)(absolute)
- Large femoral head cysts
- Large areas of AVN
- Inflammatory arthropathy
- Being female and wanting to have children
- Small femoral head size (<46 mm)
- Large BMI (BMI>35 kg/m^2)
- Large leg length discrepancy
- Severe osteoporosis (inadequate femoral head bone stock) (absolute)

Complications

AVN/collapse of the femoral head

This can present as postnecrotic fractures as late as 2–3 years after implantation.

Femoral neck fracture (1–3%)

This is the most common mode of failure. Most occur in the first 9 months.

The Australian joint registry found a significant gender difference between men (0.96%) and women (1.98%). This was attributed to postmenopausal women having a reduced bone density or the increased risk of overpenetration of cement into osteoporotic bone. Carrothers et al. (Oswestry) found no significant gender difference between men (1.0%, 33 of 3346) and women (1.3%, 21 of 1654)[63].

Predisposing factors for femoral neck fracture are (1) technical issues (notching of the superior neck, varus component positioning, undersizing of the femoral component, inadequate pin centering technique, incomplete seating of the femoral component, cement overpenetration with thermal necrosis, uncovered reamed bone), (2) head perfusion issues (posterior approach, cylindrical reaming), (3) host issues (age, female, bone quality, anatomy) and (4) surgeon issues (experience, learning curve). A smaller head size is particularly prone to femoral neck fracture.

Femoral head loosening (0.4%)

Cement mantle, depth of cement penetration within the resurfaced head, bone density and clearance between the reamed head and femoral component are all associated with implant survival.

Acetabular component loosening (0.6%)

The relative risk is much higher for women vs men (4.9).

Metallosis

Metallosis is the macroscopic staining of the soft tissues and is associated with abnormal wear usually of the bearing surface or taper junction.

ALVAL (aseptic lymphocyte-dominated vasculitis-associated lesions)

This is a delayed hypersensitivity-like reaction. Histological analysis of soft tissues retrieved at revision surgery or biopsy demonstrates an immunological response, which leads to periprosthetic osteolysis.

Pseudotumour (massive granuloma, neocapsule tissue reaction)

A pseudotumour can cause extensive collateral damage. Often there is formation of a synovial-like biomembrane which can produce collagenase, IL-1 and TNF which may lead to bone resorption and osteolysis. These are best diagnosed using a metal artifact reduction sequence (MARS) MRI scan. Ultrasound still has a role as it is cheaper, more available has no radiation risk and allows for hip aspiration in certain situations. The detection of small or deep lesions is, however, difficult.

Adverse reactions to metal debris (ARMD)

This is an umbrella term to describe joint failures associated with pain, a large sterile effusion of the hip and/or macroscopic necrosis/metallosis. There is no clear consensus defining the boundaries between metallosis, ALVAL and pseudotumours

Risk of neoplasm

At present the primary concern of long-term induction of neoplasm is unfounded. There is no documented increased risk of neoplasm. Chromosomal abnormalities in peripheral blood are more common with MoM bearing articulations.

Revision

Patients are more likely statistically to require revision surgery for pain if they have a high abduction angle, are female, have a small femoral component or a high BMI. All of these variables are associated with higher rates of wear.

There is a large disparity in the revision rates of different resurfacing implants. The Birmingham Hip Resurfacing (BHR) (Smith & Nephew, Warwick, UK) had the lowest revision rate (8.85% at 10 years) in comparison to an overall revision rate of 12.63 at 10 years for resurfacing prostheses. A higher revision rate (28.28% at 10 years) in the ASR® (DePuy, Warsaw, Indiana) due to excess metal debris and component loosening led to its withdrawal[64]. Female gender has been found to be associated with a higher incidence of pseudotumours; however, this may be due to the smaller head sizes used in females.[62] This study found a revision rate at 6 years of 6% in those over 40 years and 13% in females below 40 years of age.

Large diameter MoM THA

These were introduced in 2003 to treat failure of the femoral component of MoM resurfacing when the acetabular component was well fixed. They began to be used in large numbers instead of MoM hip resurfacing in patients with a poor quality femoral head. Perceived advantages included a low dislocation rate, a greater range of movement, lower wear rates and a potential for greater longevity compared to MoP THA.

The most common presenting symptom of a failed MoM THA is pain, located predominantly in the groin and occasionally radiating to the greater trochanter and down the thigh, and frequently associated with clicking and clunking sensations. Over the past few years concerns have been raised in relation to reports of catastrophic soft-tissue reactions resulting in implant failure and associated complications. Periprosthetic tissue sampling during revision surgery of MoM articulations has shown the presence of ARMD and ALVAL, which includes type IV hypersensitivity reactions and immunological response to metal wear debris. It remains to be shown whether these adverse reactions are dose-dependent and whether they are mediated primarily by an immune response to, or a direct toxic effect of the metal debris.

A pseudotumour has been defined as 'a soft-tissue mass associated with the implant which is neither malignant nor infective in nature'[65]. ALVAL is a histological diagnosis, which has also been used to describe the clinical appearance of tissue

necrosis and abnormal joint fluid at revision surgery. Metallosis is defined as aseptic fibrosis, local necrosis, or loosening of a device secondary to metallic corrosion and release of wear debris.

MoM failures seem to relate to poor technique including component malposition, use in unsuitable patients whose anatomy dictated malposition/edge bearing, and the wide introduction of some unsatisfactory implants with insufficient in vivo evaluation. The evidence from the literature is clear that all medical devices depend on appropriate surgical technique and patient selection. It appears that these simple rules affect the success of MoM hips more than other hip types.

It has been shown that steeply inclined acetabular components with inclination angles of >55° combined with smaller size of component were likely to give rise to higher serum levels of cobalt and chromium[66]. Malpositioning of the components can lead to earlier failure and revision. A study published in the Lancet in 2012,[67] based on data from the NJR of England and Wales, analysed 402 051 hip arthroplasty and showed that 6.2% of MoM implants had failed within 5 years, compared to 1.7% of MoP and 2.3% of CoC hip implants. The same study concluded that each 1 mm increase in head size of MoM implants was associated with a 2% increase of failure.

Another important source of wear in MoM (THA) bearings is the head-neck taper. The use of large femoral heads on narrow-stem tapers increases the risk of corrosive wear at the trunnion as a result of increased frictional torque, which is associated with large diameter heads(trunnionosis). This may contribute to any elevated metal ions and adverse tissue reactions. It has been shown that the failure rate of the ASR® (DePuy, Warsaw, Indiana) MoM THA is higher than that of the resurfacing version, which supports the trunnion wear theory[68,69].

Examination corner

Adult elective orthopaedic oral 1

The examiner handed me a MoM prosthesis.

EXAMINER: What is this prosthesis?

EXAMINER: What are its design characteristics?

EXAMINER: What is the advantage of MoM articulation?

CANDIDATE: Less wear debris, a lower risk of osteolysis and less aseptic loosening compared with metal on PE articulations.

EXAMINER: Do you know any literature evidence?

EXAMINER: What are the problems of metal debris?

CANDIDATE: There are local and systemic effects of metal ions. ALVAL, pseudotumours and ARMD are causes for concern.

EXAMINER: What does the BHS guideline say about this?

CANDIDATE: A recent document giving information and advice to surgeons on metal on metal bearings was published jointly by the BHS and BOA. They recommend that worsening or severe pain, rising metal ions or increasing size of cystic or solid mass are concerning and may require revision surgery. There is increasing evidence that solid masses are more concerning than cystic ones. There is also evidence that cystic masses are found adjacent to well functioning hips.

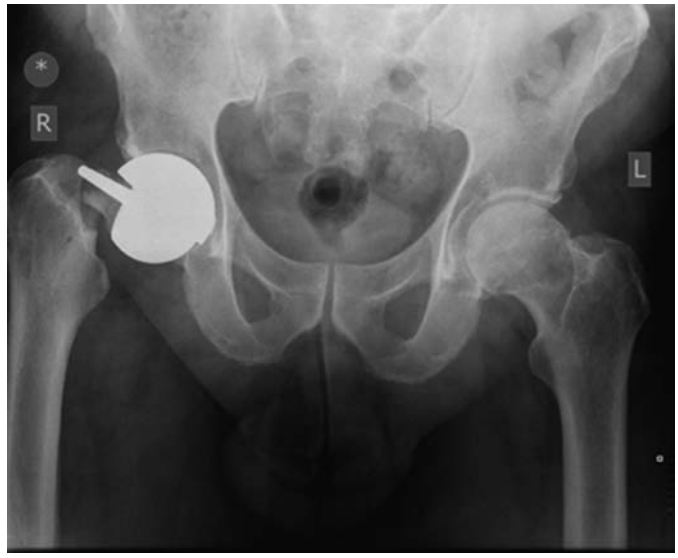

Figure 16.22 Radiograph of femoral neck fracture hip resurfacing

The viva could now lead onto the ASR® implant, design characteristics and mode of failure, although it would be taking on a basic science slant[hh]. This could lead on to discussion of joint registries, how to set one up, etc.

The ASR® was less forgiving of component malorientation, both because the cup was subhemispherical-designed to reduce impingement, and because of the internal groove designed to accommodate the cup inserter. The effective arc of cover was essentially reduced, rim loading occurred earlier and there was a higher than expected failure of this device. In addition there was suboptimal manufacturing of the cobalt chromium material. It has been recalled from the market.

Adult elective orthopaedic oral 2: Radiograph of a femoral neck fracture following MoM resurfacing

EXAMINER: This is a radiograph of a 51-year-old man who presented to casualty with right hip pain (Figure 16.22). He had an MoM hip resurfacing procedure 1 week previously.

CANDIDATE: The radiograph demonstrates a fairly obvious femoral neck component fracture.

EXAMINER: What are the potential causes for this?

CANDIDATE: The fracture occurred in the early postoperative period so the most obvious causes would be notching of the femoral neck at surgery. Notching increases the risk of fracture.

EXAMINER: How can you prevent this?

CANDIDATE:

- Accurate templating prior to surgery
- Accurate central pin placement using a reliable jig system. Some jigs are more user-friendly than others and are designed to minimize this risk

[hh] The adult and pathology can go towards basic science and vice versa. The boundaries aren't always clear cut.

- Upsizing the head when performing the initial femoral head reaming and only downsizing if there is no chance of notching
- Experienced surgeon past the learning curve

EXAMINER: Any other risk factors?

CANDIDATE: A smaller femoral component size is prone to this mode of failure. Accurate component placement without notching is more difficult when the femoral head size decreases. Also, following head preparation, the ratio of femoral bone to metal component decreases disproportionately with decreasing head size. Other risk factors include varus alignment of the femoral component and a thick cement mantle at the dome of the femoral head.

EXAMINER: So how can we minimize these risks at surgery?

CANDIDATE: I can't think of anything else.

EXAMINER: Computer navigation.

EXAMINER: How well do patients do if they require revision of an MoM hip?

CANDIDATE: Patients with femoral loosening have significantly lower hip scores after revision surgery than did those with femoral neck fracture and those with femoral head collapse or AVN. If the revision is performed for pseudotumours then results tend to be much poorer because of the associated soft-tissue damage.

EXAMINER: You mentioned a learning curve for MoM resurfacing. Is there any literature that has looked into this?

CANDIDATE: De Smet et al. published an annotation in the *Journal of Bone and Joint Surgery* summarizing the Ghent advanced hip resurfacing course[70]. They stated that an orthopaedic surgeon should have a minimum experience of 200 conventional THAs before starting hip resurfacing. Opinion varied on the number of resurfacings needed to overcome the learning curve, ranging from 20 (36% of voters) to 50 (28% of voters) and >50 (30% of voters).

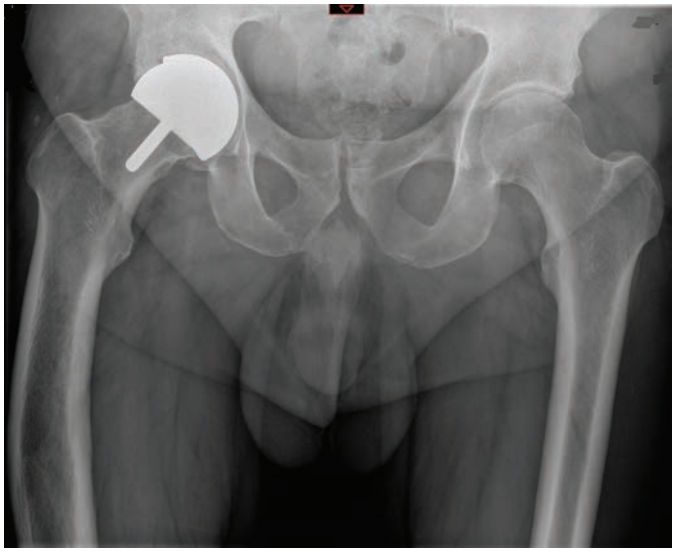

Figure 16.23 Radiograph of MoM hip resurfacing with opened cup

Adult elective orthopaedic oral 3: radiograph of an opened MoM acetabular cup

EXAMINER: This is a postoperative check radiograph of a hip resurfacing arthroplasty (Figure 16.23). Would you like to comment on the x-ray?

CANDIDATE: The cup is opened and the cup angle is steep and more than the 40° currently recommended.

EXAMINER: This is the postoperative radiograph. Would you go back in and reposition the cup into a more closed vertical position?

CANDIDATE: This is a difficult dilemma in the postoperative period. If the cup is in a reasonable position albeit opened more than ideal I would leave it. If it was markedly opened I would revise it. I would probably want to discuss it with my colleagues and take their advice.

EXAMINER: What angle of cup inclination is recommended?

CANDIDATE: With conventional hips cups were inserted at 45° of abduction and 20° of anteversion. It is now recommended to insert the cup in 40° of abduction.

EXAMINER: OK, you don't do anything but he presents to your clinic 2 years after surgery with progressively increasing hip pain. What are you going to do?

CANDIDATE: I would investigate him for a painful MoM hip resurfacing as per BHS guidelines. I would first want to exclude infection – I would go through the history and examination findings looking for pointers towards infection such as a postoperative wound haematoma or washout. I would measure ESR/CRP, IL-6, perform a bone scan and aspirate the hip.

EXAMINER: You have excluded infection.

CANDIDATE: There are many causes for a painful resurfacing hip implant. I would want to consider soft-tissue issues such as psoas/adductor tendonitis – I would request a lateral radiograph looking for excessive retroversion of the acetabular component that leads to the cup uncovering anteriorly which may be the reason. I would exclude referred pain from elsewhere such as the spine, sacrum or femoral hernia. Other causes of failure could include impingement, aseptic loosening of components, femoral neck fracture or resorption/AVN. The radiographs perhaps suggest an element of HO.

EXAMINER: HO has been excluded as a significant factor. All these other factors have also been excluded.

CANDIDATE: I would examine for any groin swellings, as well as the hip range of movement, pain and limping. I would want to measure serum cobalt/chromium levels.

EXAMINER: There are marginally elevated.

CANDIDATE: I would request an MARS MRI scan specifically looking for any evidence of soft-tissue masses such as pseudotumours. I would obtain the old operating notes to ascertain the particular model of the prosthesis and head size

EXAMINER: The MRI is normal. There is no evidence of fluid collections or pseudotumours.

CANDIDATE: Then I wouldn't do anything at the moment. I would leave alone and follow up in clinic regularly.

EXAMINER: You wouldn't revise the hip.

CANDIDATE: No.

EXAMINER: The patient returns 6 months later, still in pain, in fact getting worse.

CANDIDATE: I would re-measure his serum cobalt/chromium levels.

EXAMINER: Why?

CANDIDATE: If levels are raised and climbing compared to previous results and the hip is becoming progressively more painful and with the high cup component inclination angle I would need to consider revision.

EXAMINER: The MRI was normal.

CANDIDATE: I might want to repeat the hip MRI as things may have changed. If any fluid is present around the hip MRI I would perform a hip aspiration looking for brown-stained fluid typical of pseudotumours.

EXAMINER: You can also get a milky fluid with pseudotumours.

CANDIDATE: I would discuss his case with an experienced revision hip surgeon as per BHS/BOA guidelines.

EXAMINER: Blood metal ion levels were static, repeat MRI again showed no evidence of pseudotumours and we are observing the patient at present. If there is any change we will need to revise. A fluid collection by itself around the joint in an asymptomatic patient, unless very large can be safely observed with interval scanning. The MARS MRI scan is more important in the decision-making process to revise the MoM hip replacement than elevated cobalt/chromium levels. A significant worry would be patients with muscle or bone damage on MARS MRI.

COMMENT: This was a good candidate answer; they were familiar with the workup of the painful MoM hip resurfacing. Everything was covered, including BHS/BOA national guidelines. Sensible practical answers were given not just reading facts from the book and ticking boxes.

Adult elective orthopaedic oral 4: photograph of aspirated fluid from a painful MoM hip

EXAMINER: This is a photograph of joint aspirate fluid from a patient being investigated for a painful MoM hip resurfacing (Figure 16.24). What does it show?

CANDIDATE: Brown coffee-like fluid.

EXAMINER: Why aspirate the hip?

CANDIDATE: If you suspect infection.

EXAMINER: What do you think of the aspirate?

CANDIDATE: The aspirate is more suggestive of a pseudotumour fluid collection.

EXAMINER: What would you find at surgery?

CANDIDATE: Either a cystic or solid mass with extensive soft-tissue destruction and necrosis.

EXAMINER: What would the serum cobalt and chromium levels show?

CANDIDATE: They are generally raised in cases of pseudotumours and ALVAL, although a very small number of patients who develop pseudotumours have normal metal ion levels.

EXAMINER: That's the point – You don't need raised serum cobalt/chromium levels to develop pseudotumours, although if they are raised it is more likely.

Figure 16.24 Aspirated fluid from a painful MoM hip

EXAMINER: What are the advantages of an MoM hip resurfacing?

CANDIDATE: The advantages of hip resurfacing arthroplasty include improved range of movement, improved gait parameters, ease and decreased morbidity of revision arthroplasty, reduced dislocation rates, normal femoral loading and reduced stress-shielding, simpler management of a degenerated hip with a deformity in the proximal femoral metaphysis (after trauma or osteotomy), less risk of infection, and a reduced risk of DVT/PE secondary to not using instruments in the femur.

The procedure preserves the femoral neck and part of the head and does not invade the femoral canal, thus, preserving bone stock.

Recent studies have shown preservation of bone mineral density (BMD) in the femoral neck after resurfacing arthroplasty, and a significant increase in BMD in Gruen zone VII educ.

Revision total hip arthroplasty (THA)

Surgical goals in revision hip surgery

1. Removal of loose components without significant destruction of host bone and tissue
2. Reconstruction of bone defects with bone graft with or without metal augmentation
3. Stable revision implants
4. Restoration of normal hip centre of rotation

Indications

Indications for revision hip surgery include:

- Painful aseptic loosening of one or both components
- Catastrophic implant failure
- Recurrent dislocation or instability
- Infection
- Periprosthetic fracture
- Progressive osteolysis
- Excessive wear of components
- Adverse Soft Tissue Reaction to Particulate Debris

Contraindications

Contraindications for surgery would include:

- Referred pain from elsewhere
- Medically unfit patient
- Caution with painless LLD
- Rarely indicated for painless loss of motion
- Be very careful about operating for hip pain when no obvious cause is found. Preferable to obtain a second opinion and leave to experienced revision hip surgeon
- When the pain present prior to the index procedure persists postoperatively the pain was probably not from the hip originally

Acetabular reconstruction

Acetabular defect classification systems are used to predict the extent of intraoperative bone loss and guide reconstructive options. Several classification exist;the two most commonly cited are those by D'Antonio (AAOS classification) (Table 16.15) and Paprosky (Table 16.16).

The AAOS classification is descriptive and does not provide the surgeon with a guide for reconstructive options. Poor reliability has been reported. The Paprosky classification system is based on the status of the acetabular rim, dome, columns and contact area available for ingrowth at the time of revision. It assesses the severity of bone stock loss and the ability of the acetabulum to contribute to implant stability. It is preferred by some surgeons because it is simple, useful for operative planning and allows critical evaluation of various management options and their outcomes. The intra- and interobserver reliability have been found to be moderate to poor by some authors.

AAOS classification system (Table 16.15)

This has special categories for pelvic dissociation and arthrodesis.

Table 16.15 AAOS classification system for acetabular deficiencies in THA

Type	Lesion	
Type I	Segmental deficiency • Peripheral (rim) • Central (medial wall absent)	Loss of part of the acetabular rim or medial wall
Type II	Cavitary deficiency • Peripheral (superior, anterior, posterior) • Medial (medial wall intact)	Volumetric loss in the bony substance of the acetabular cavity
Type III	Combined deficiencies	Combination of segmental bone loss and cavitary deficiency
Type IV	Pelvic discontinuity	Complete separation between the superior and inferior acetabulum
Type V	Arthrodesis	

Paprosky classification system (Table 16.16)

Four radiographic criteria are assessed:

- **Acetabular component migration.** Superior migration involves acetabular dome loss, superior/medial greater involvement of the anterior column, superior/lateral greater involvement of the posterior column
- **Osteolysis of ischium.** Indicates bone loss from the inferior aspect of the posterior column
- **Osteolysis of the teardrop.** Severe involvement means complete obliteration of the teardrop
- **Kohler's line**. Position of the implant relative to Kohler's line

Defects are classified by type, indicating whether the remaining acetabular structures are completely supportive (type 1), partially supportive (type 2), or non-supportive (type 3).

Type 1: Supportive rim with no bone lysis or component migration

Type 2: Anterior and posterior columns are intact. Some destruction of the dome and medial wall of the acetabulum. In type 2A there is superior bone loss from component migration or osteolysis but an intact acetabular rim (contained cavitary). In type 2B, the superolateral rim is absent so the defect is considered segmental (uncontained). Type 2C demonstrate more localised medial wall destruction

Type 3: Defects demonstrate extensive superior migration of the acetabular component with >2 cm of superior bone loss and loss of the superolateral rim. Type 3A defects demonstrate moderate, but not complete, destruction of the teardrop (medial wall of the teardrop is still present) and moderate lysis of the ischium. Because the medial wall is present, the component usually migrates superolaterally. Type 3B defects show complete obliteration of the teardrop and severe lysis of the ischium, usually resulting in superomedial component migration.

Talk the radiograph talk

'The AP pelvic radiograph shows a type 1 AAOS segmental defect to the superior peripheral acetabular rim following THA.'
'The radiograph demonstrates a Paprosky type 2C acetabular defect. The teardrop is obliterated with generalized rim enlargement and severe medial wall destruction.'

Table 16.16 Paprosky classification of acetabular defects

Type	Radiographic finding	Intraoperative finding	Trial stability
I	No cup migration No substantial bone loss	Intact rim and no distortion. No major osteolysis. Bone loss minimal Small focal areas contained bone loss Columns intact	Full
IIA	Superior(or superomedial) migration of <3 cm No substantial ischial lysis No substantial teardrop lysis	Superomedial bone loss Columns supportive and rim intact Migration into defect under thin superior rim Host-bone contact of >50%	Full
IIB	Superior (or superolateral) migration of <3 cm	Uncontained superior rim defect <1/3 Columns supportive Host-bone contact of >50%	Full
IIC	Medial wall defect Cup medial to Kohler line	Uncontained medial wall defect Rim intact and rim columns supportive	Full
IIIA	Superolateral cup migration Moderate ischial lysis Partial teardrop destruction Kohler line intact	Unsupportive dome Columns intact Host–bone contact of 40–60%	Partial
IIIB	Superomedial migration Severe ischial destruction Teardrop loss Migration medial to Kohler line	Risk of pelvic discontinuity Bone contact of <40% Rim defect of >50%	None

Acetabular reconstruction options

The ideal revision socket should be simply to insert, have good long-term survival results, be reliable, be able to accommodate unusual defects, should give initial stability and facilitate bone stock preservation It should allow the option of using a large head and the choice of various bearing surfaces

Acetabular reconstruction options include:

Isolated acetabular liner exchange

Indicated for a well-fixed and well-oriented acetabular component with progressive acetabular osteolysis. Patients are often asymptomatic. Goals of surgery are to prevent full-thickness liner wear with associated catastrophic failure. Bone-grafting of the osteolytic lesion is often required. Postoperative dislocation is a concern. Use of a modified Hardinge approach and largest possible femoral head component(optimizes head : Neck ratio) reduces this risk.

Uncemented hemispherical cup with or without bone graft

In most patients, bone loss encountered at acetabular revision can be managed with a standard cementless hemispherical component if initial stability and sufficient component-bone contact is achieved. Fixation is usually supplemented with transacetabular screws, and contained bony defects are filled with cancellous bone allograft. A major advance is the use of second generation porous acetabular components such as tantalum or titanium metal. These make a major difference in the setting of difficult acetabular revisions involving massive bone loss.

Structured bulk allograft

In hips with substantial segmental acetabular bone loss, the structural support necessary for hemispherical component stability is lost. Structural allograft can be used in this situation. Potential for restoration of normal hip center and bone stock for future revisions but technical difficulty so needs careful planning. Complications include unsuccessful osseointegration of graft into host leading to implant failure, increased risk of infection, increased operating time and increased blood loss.

Metal augments(tantalum)

An alternative for structural support involves use of special modular porous metal augments. There are several choices of augments that can be sized, oriented, and positioned to closely match the dimensions of segmental acetabular defects.

Cemented cup

Less favoured over last 2 decades, disappointing results in the revision setting.

Impaction grafting ± mesh

Initially described with cemented acetabular cup, this technique can be used with cementless components. Potential for restoration of lost bone stock and versatility in managing both contained and segmental patterns of bone loss. Technically difficult and time consuming.

Cage

Usually reserved with the most difficult acetabular revisions with massive bone loss and pelvic discontinuity. Historically, these constructs lack the potential for biological fixation and were doomed to fatigue failure. To address this limitation, cages and custom triflange components have recently been designed and manufactured with ingrowth surfaces.

Acetabular cages provide short-term stabilisation. In this setting, the device provides fixation and stability during the period of time that another device (such as a hemispherical cup) achieves long-term biological osseous ingrowth.

Failure of graft incorporation with resorption leads to eventual cage fatigue and construct failure.

Contemporary reference

Deirmengian GK, Zmistowski B, O'Neil JT, Hozack WJ. Management of acetabular bone loss in revision total hip arthroplasty. *J Bone Joint Surg Am*. 2011;93:1842–1852.

Current concepts review

Current concepts overview of acetabular reconstructive options.

Contemporary reference

Reid C, Grobler GP, Dower BJ, Nortje MB, Walters J. Revision total hip arthroplasty: Addressing acetabular bone loss. *SA Orthop J*. 2012;11:34–46.

Provides a useful management framework(history, clinical examination and investigations) that can be utilized in the isolated loose acetabular cup needing revision viva scenario.

Femoral reconstruction

Several classification systems exist for describing femoral bone loss in revision hip surgery.

AAOS Classification system for femoral defects (Table 16.17)

Whilst the AAOS classification system is well established and highly descriptive in detailing osseous abnormalities, it does not provide a guide for reconstruction.

Type 1: These defects are segmental in nature, typically involving the proximal part of the femur

Type 2: These defects typically involve ballooning of the cortex to create an ecstatic canal. There is an intact proximal femoral tube with irregular endosteal loss and cavitation

Table 16.17 AAOS classification of femoral abnormalities in THA

Type	Lesion
Type I	Segmental (loss of bone supporting shell of femur) a. Proximal i. Partial ii. Complete b. Intercalary c. Greater trochanter
Type II	Cavitory (loss of endosteal and cancellous bone but intact cortical shell)
Type III	Combined segmental and cavitary
Type IV	Mal-alignment (loss of normal femoral geometry) owing to previous surgery (osteotomy), trauma a. Rotational b. Angular
Type V	Stenosis (occlusion of canal from trauma, fixation devices or bony hypertrophy)
Type VI	Femoral discontinuity (loss of femoral integrity from fracture/non-union)

Type 3: There is a proximal femur, which is both ballooned and deficient in its cortical integrity

Type 4: These defects are characterized by mal-alignment involving either rotatory or angular deformity

Type 5: These features are often the sequel to previous periprosthetic fractures

Type 6: Characterized by periprosthetic discontinuity between the upper and lower halves of the femoral shaft

Weeden and Paprosky classification system for femoral defects (Table 16.18)

The Weeden and Paprosky classification depends on the quantity of metaphyseal and diaphyseal bone stock. It is based on the principle that as proximal bone becomes weak and unsupportive, the relatively spared diaphyseal bone can be successfully used to provide reliable, long-term fixation. The system assigns the femur to one of four categories on the basis of the extent and location of bone loss.

Implant options[ii]

- Proximally loading modular porous coated stems
- Cylindrical extensively porous coated ingrowth stems
- Modular revision stems
- Impaction bone grafting
- Distally locked prosthesis
- Megaprosthesis
- Allograft–prosthesis composites(proximal femoral replacement)

[ii] See website www.postgraduateorthopaedics.com for additional information.

Table 16.18 Weeden and Paprosky classification system for femoral defects

Type I: Minimal metaphyseal cancellous bone loss/normal intact diaphysis Type I defects are seen after removal of uncemented component without biological ingrowth on its surface. The diaphysis and metaphysis are intact and there is partial loss of the calcar and AP bone stock. Uncommon in the revision setting
Type II: Extensive metaphyseal cancellous bone loss/normal intact diaphysis Often seen after removal of cemented prosthesis. Calcar deficiency and major AP bone loss. Common finding in the early stages of aseptic loosening
Type IIIA: Metaphysis severely damaged >4 cm diaphyseal bone for distal fixation • Grossly loose femoral component • First-generation cementing techniques • Extensive metaphyseal bone loss, leaving it unsupported. Most frequent encountered defect in femoral revision surgery
Type IIIB: Metaphysis severely damaged/ <4 cm diaphyseal bone for distal fixation These defects extend slightly further than type IIIA, although reliable fixation can be achieved just past the isthmus of the femur Seen with the use of longer cementless stems
Type IV: Extensive metaphyseal and diaphyseal bone loss/ isthmus non-supportive Extensive defect with severe metaphyseal and diaphyseal bone loss and a widened canal that cannot provide adequate fixation for a long stem

Postoperative complications

Failure rates of revision THA are three times that of primary surgery.

- Infection (12–17%)
- Dislocation (5–10%)
- Vascular injury
- Nerve palsy
- Cortical perforation
- Fracture
- Heterotopic ossification
- LLD
- DVT/PE rate similar to those for a primary operation

Conversion of hip arthrodesis to THA

This is indicated if a fused hip causes severe persistent low back pain, pain in the ipsilateral knee, or the pseudarthrosis is painful (rule out infection).

Contralateral hip pain is rarely an isolated problem. The contralateral joints are especially vulnerable if the hip has been fused in a poor position (flexed >30°, adducted >10° or abducted to any extent). In this situation osteotomy should be considered first to correct the position.

One needs to try assess the function of the abductors preoperatively. Inadequate strength of the abductor muscles results in a Trendelenburg's gait, a feeling of instability of the hip, the probable need for a walking aid and the inability to stand on one leg. The strength of the abductor muscles postoperatively is related to preoperative muscle mass as well as to the placement of hip center. Exposure of the hip is difficult because of the distorted anatomy and soft tissue contractures.

Results

The complication rate for conversion can be high. One study reported a 33% failure at 10 years with a previous history of surgical fusion because of loosening, infection or recurrent dislocation. Nerve palsy has been reported to be as high as 7% in some series.

Hip arthrodesis

A hip arthrodesis, when performed correctly, preserves bone stock, provides indefinite pain relief, allows a reasonably active lifestyle albeit with some restriction of physical activity and allows conversion to a THA at a later date. The patient must understand and accept before surgery the disabilities associated with the procedure.

Indications

This is indicated in a young patient with unilateral OA hip. It is especially suited in the young man with OA secondary to trauma and involved in heavy manual work. The long-term results of THA in this patient population are disappointing.

Prerequisites

The patient must have a normal contralateral hip, ipsilateral knee and spine, as a fused hip increases the stresses on these joints and the clinical results of hip fusion can be compromised.

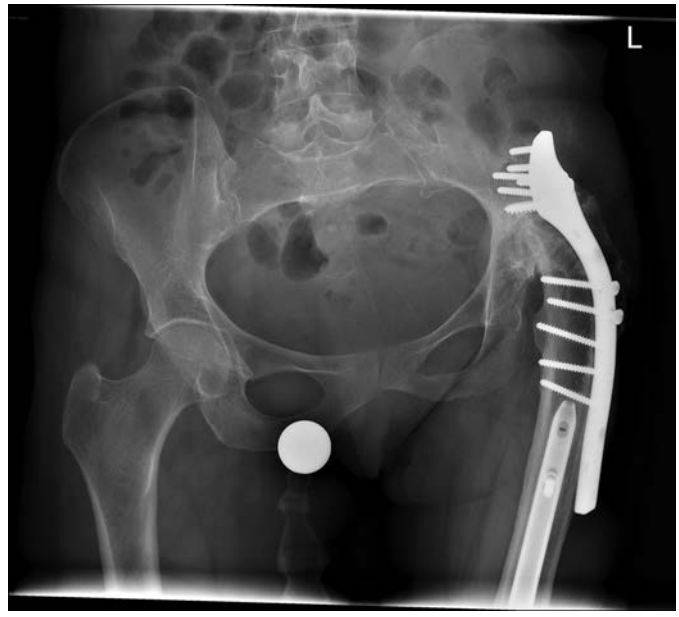

Figure 16.25 Fused left hip

Contraindications
Active infection, obesity, poor bone stock.

Advantages
Painless and stable joint for many years.

Disadvantages
- Immobile joint
- Leg length discrepancy
- Pain in adjacent joints with long-term follow up

Techniques
Fusion of the hip may be obtained by extra-articular, intra-articular or combined intra-articular and extra-articular methods.

AO Cobra head plate technique
Fixation spanning the pelvis and proximal femur. Stable but disrupts hip abductors and requires bone graft (Figure16.25).

Trans-articular sliding hip screw
The lag screw is inserted just superior to the dome of the acetabulum. Poor fixation achieved owing to a large lever arm and increased torque and, therefore, hip spica casting may be required postoperatively.

Anterior plating technique
An extended Smith–Petersen approach is used and, although the femoral head and acetabulum can be prepared for hip arthrodesis, the abductor mechanism is not violated. The fusion plate is taken across the anterior column of the pelvis superiorly into the sacroiliac joint.

Combined intra-articular and extra-articular fusion
Combination of plating and lag screw fixation.

Position
- Avoid abduction and internal rotation
- 20–30° flexion
- Neutral – 5° external rotation
- Neutral – 5° adduction

Arthrodesis in an abducted position produces pelvic obliquity and a limp. More flexion produces a greater leg length discrepancy and lumbar lordosis, whilst less flexion creates sitting difficulties.

Complications
Most patients will have complications from this surgery, which may be either major or minor.
- Malposition (most common)
- Neurovascular injury
- Femoral fracture in the first year following surgery
- Failure of internal fixation
- Non-union (pseudoarthrosis rate of 15–25%)
- OA hip, knee, spine
- Instability ipsilateral knee
- LLD (common)

Hip fusion and contralateral THA
THA has 40% more mechanical failure and loosening. Consider an anterior approach to reduce the risk of dislocation (particularly when sitting) if there is a contralateral arthrodesis.

Informed consent
Warn about variable amounts of leg length discrepancy and a possible need for a shoe lift postoperatively.

Examination corner

Adult elective orthopaedics oral 1: Radiograph of pelvis showing ankylosed right hip possibly caused by old tuberculosis

EXAMINER: What is the position of the hip for fusion?

CANDIDATE: 30° flexion, neutral to 5° external rotation and neutral or slight adduction.

EXAMINER: What are the various techniques that can be used for hip fusion?

EXAMINER: What effect does arthrodesis have on a contralateral total hip replacement?

CANDIDATE: Mechanical loosening occurs at a higher rate when the opposite hip has been arthrodesed.

Heterotopic ossification following THA

Definition
Heterotopic ossification is the abnormal formation of mature lamellar bone outside the skeleton usually in soft tissue.

Incidence
The radiographic incidence of HO following primary THA has been reported to vary between 5% and 90% (21% in Brooker et al.'s original paper[71]), but only 3–7% have significant symptoms.

Predisposing risk factors
- Male (2× >F)
- Hypertrophic OA
- Ankylosing spondylitis
- Diffuse idiopathic skeletal hyperostosis (DISH)
- Post-traumatic OA
- Prior hip fusion
- Paget's disease
- Rheumatoid arthritis
- History of previous HO in ipsilateral or contralateral hip
- Advanced age

Surgical risk factors

- Intraoperative muscle ischaemia
- Direct lateral approach[72]
- Extent of soft-tissue dissection
- Bone trauma
- Persistence of bone debris (reamings, marrow within the surgical field)

Pathology

Inappropriate differentiation of pluripotent mesenchymal stem cells into osteoblastic cells. A definite causal factor has not been identified. Research suggests overexpression of BMP-4 and PGE_2.

Clinical features

HO following THA is usually painless and noted as an incidental finding on radiographs. When symptomatic it presents with limited hip motion and leads to a poor functional outcome. Pain is rare but can occur. Occasionally, localized warmth, mild oedema and erythema may occur similar to infection. Surgical excision is rarely indicated owing to the high incidence of recurrence.

Surgery may be indicated in the rare cases of:

- A severe restriction of hip range of motion
- Severe pain from impingement

Allow the process to mature (sharp cortical and trabecular markings) before operative resection. Some authors recommend waiting 12 months before operative resection.

Radiology

Calcification of soft tissues can occur as early as 2 weeks postoperatively, maturing fully by 1 year. Bone scans are positive after 3 weeks, with increased uptake in the soft tissues.

Classification: Brooker 1–4

Brooker et al.[68] described four stages based on an AP radiograph of the pelvis:

- Islands of bone within the soft tissues about the hip
- Bone spurs from the proximal femur or pelvis with at least 1 cm between opposing bone surfaces
- Bone spurs with <1 cm gap
- Apparent bony ankylosis of the hip

Grade 1 or 2 does not influence the outcome of THA, but grades 3 or 4 have a less favourable result.

Classic reference

Brooker AF, Bowerman JW, Robinson RA, Riley LH Jr. Ectopic ossification following total hip replacement: Incidence and a method of classification *J Bone Joint Surg Am*. 1973;55:1629–32.

Determination of ectopic bone formation was made on AP radiographs at a minimum of 6 months following THA in 100 consecutive patients. Significant HO was found in 21 hips following THA rated as Class I (7), Class II (5), Class III (7) and Class IV (2).

HO rated Class I, II or III did not affect the Harris Hip Score (HHS) whilst the two patients with Brooker class IV had lower than anticipated HHS.

Typically HO is asymptomatic, but higher Brooker grades can result in impairment of hip arthroplasty function due to pain, impingement, instability, decreased range of movement or ankyloses, trochanteric bursitis and nerve irritation.

In the early days of THA, HO was considered a minor problem that had little effect on the clinical outcome. In 1973, Brooker et al[68] introduced a method of classifying HO and reported an incidence of 21%. In this article, HO influenced the functional outcome of the surgery only in cases with a complete bony bridge.

One main concern is that bone that appears to be bridging in an AP radiograph, may actually be located either anterior or posterior to the hip and will, therefore, not cause a significant loss of hip range of motion. The overall incidence of HO originally reported in Brooker's paper (21%) is substantially lower than that accepted these days.[69] A recent systematic review has reported an incidence of 43% and that of severe HO (Brooker grades III/IV) of 9%[73]. The reproducibility of the Brooker classification and its use for clinical and research purposes is still controversial. A number of authors have questioned the low interobserver and intraobserver reliability achieved when using this classification system. Several studies have reported poorer clinical outcomes with Brooker grade III HO than what was reported in the original paper by Brooker[74,75].

Prevention

- External beam radiation therapy 700–800 rads single dose (<4 h preop or 72 h postop)
- Indomethacin 75 mg for 6 weeks. Acts by inhibiting the production of prostaglandins. Potential side effects include GI bleeding, renal impairment, severe allergic reactions, headaches, etc. A worry if uncemented components are used, as bony in growth may be affected
- Combination therapy. Radiotherapy and NSAIDs suggested for patients at highest risk for HO

Examination corner

Adult and pathology oral 1

The candidate was shown an AP radiograph of a primary THA performed several months previously. There were severe Brooker grade 4 changes on the radiograph.

The candidate was asked about predisposing causes, Brooker classification and about which symptoms the patient was likely to complain.

Causes are different to risk factors and include trauma, spinal cord injury, severe burns and genetic conditions such as fibrodysplasia ossificans progressiva (AD, stone man syndrome).

CANDIDATE: Pain is an uncommon feature of the condition. Stiffness may be present but it would need to lead to a significant disability before one would consider surgery. With Brooker grade 4 changes the patient may complain of difficulty with sitting, ascending stairs, or putting on shoes and socks.

Adult and pathology oral 2

'I was shown an AP pelvic radiograph with a THA in situ that had evidence of severe heterotopic ossification. I was asked about predisposing causes and the Brooker classification.'

Basic science oral 1

Discussion of the management and prophylaxis of heterotopic ossification after a pelvic fracture.

Possible role for postoperative irradiation after fixation of complicated acetabular fractures. Risk factors include iliofemoral surgical approach; T type fractures; and presence of associated abdomen and chest injuries. If symptoms severe consider surgical excision.

Osteotomy

Introduction

Osteotomy aims to improve congruency and reduce point loading by restoring proper biomechanics. This is achieved by increasing the surface area available to transfer loads, decreasing muscle forces across the joint and reorientating the weight-bearing surfaces of the joint to allow normal areas to articulate, moving away the diseased areas from the weight-bearing axis. Proximal femoral osteotomy, pelvic osteotomy or both can achieve these goals. Proximal femoral osteotomy should be considered when the predominant deformity is in the proximal femur. Patients with inflammatory arthritis are not suitable candidates for osteotomy. Timely intervention is required as the prognosis is adversely affected by the presence of advanced arthrosis.

Indications for proximal femoral osteotomy

- Young patient with advanced OA hip to avoid THA
- Post-Perthes' hinged abduction disease (valgus extension osteotomy)
- SUFE (flexion osteotomy)
- DDH (varus derotational osteotomy to address the anteverted valgus neck)
- Avascular necrosis
- Idiopathic protrusio (valgus extension osteotomy)
- Mal-union of trochanteric fractures
- Congenital coxa vara

Indications for pelvic osteotomy

DDH rarely involves a primary femoral deformity; hence, it is usually treated with a pelvic osteotomy rather than isolated femoral osteotomy. Acetabular osteotomies in the adult patient have been classified into two groups: Reconstructive and salvage osteotomies. Periacetabular osteotomy has recently emerged as the method of choice for young adults with significant hip dysplasia and minimal arthritic changes.

Clinical

With osteotomy motion is neither lost nor gained but its range is altered. The patient must have sufficient preoperative motion so that correction leaves a functional range of movement. Mechanical hip pain commonly occurs with weight-bearing and may be associated with a subjective feeling of instability or weakness and clicking or locking. Exclude painful hip conditions other than mechanically induced pain. Chondral defects and loose bodies may also mimic symptoms of mechanical hip pain. In the assessment of the patient's active and passive range of motion, the presence of flexion, abduction and external rotation contractures should be noted along with any leg length discrepancy.

Radiographs

AP and lateral radiographs of the pelvis and the proximal femur. On the femoral side assess for:

- Poor bone quality
- An abnormal femoral neck shaft angle
- Incongruity of the femoral head
- Unusual trochanteric anatomy

Whilst with the acetabulum evaluate for:

- Poor bone stock
- Presence of cysts and osteophytes
- Degree of dysplasia

Functional radiographs (maximum abduction and adduction) are helpful in establishing which position of the proximal femur will improve the congruency of the hip joint and coverage of the femoral head. Other studies include a three-dimensional CT scan, CT arthrogram or MRI scan.

Contraindications

- Stiffness
- Obesity
- Inflammatory joint disease
- Presence of significant arthrosis
- Stiff hip (minimum 90° flexion, 15° abduction/adduction)

Technical considerations

The aims of surgery are:

- Elimination of impingement

- Correction of deformity
- Restoration of a pain-free range of movement
- Maintenance of the mechanical axis of the femur in both coronal and sagittal planes
- Maintain or restore equal leg lengths
- Restoration of proper rotational alignment

Types of osteotomies

The major types of femoral osteotomy are:

- Flexion
- Extension
- Varus
- Valgus
- Rotational
- Combinations of the above

Varus osteotomy

A prerequiste for surgery is congruency of the joint in the realigned position. This is confirmed with improved femoral head coverage seen on the functional abduction view radiographs. Some surgeons perform hip arthrograms to assess this prior to the osteotomy. The patient should have a minimum of 15° abduction preoperatively. The osteotomy works by shifting the greater and lesser trochanters upwards, reducing the tension of the abductors and iliopsoas and, therefore, the vertical compression forces. This improves a Trendelenburg gait pattern. A disadvantage is that this osteotomy usually shortens the leg by at least 1 cm. The most common technique is to excise a medially based wedge of predetermined size and fixation of the osteotomy with a blade plate device. Varus osteotomy displaces the centre of hip rotation medially and should be combined with medial displacement of the femoral shaft to maintain the lower extremity mechanical axis passing through the centre of the knee. This avoids overloading the medial compartment of the ipsilateral knee but results in a laterally prominent proximal femur, which may cause cosmetic concerns.

Valgus osteotomy

This is usually indicated as a salvage procedure in a young patient for an OA hip or post-Perthes' disease deformity with coxa magna, hinged abduction and a large medial osteophyte. An acceptable passive range of motion is required, with a minimum flexion of 90° and adduction of 15° preoperatively. An adduction functional film should show improved congruency of the joint. Valgus osteotomy generally lengthens the limb. If lengthening is undesirable, a closing wedge can be used but this may shorten the leg by as much as 2 cm.

A valgus osteotomy displaces the centre of hip rotation laterally and should be combined with lateral displacement of the femoral shaft to align the mechanical axis of the limb through the centre of the knee and avoid overloading the lateral compartment.

Flexion osteotomy

Indications include hip extension contracture, AVN with anterior involvement in the sagittal plane and posterior sparing of the femoral head. The apex of the osteotomy is located posteriorly and so a wedge of bone is removed anteriorly. The shaft of the femur is flexed and the proximal femur is extended. A posterior closing wedge may be better, less likely to compromise future stem insertions.

Extension osteotomy

Indications for extension osteotomy include hip flexion contracture and deficient anterior acetabular coverage (seen frequently with DDH). The apex of the osteotomy is located anteriorly so that the shaft of the femur is extended and the proximal femur is flexed.

THA after previous osteotomy

A previous femoral osteotomy may render subsequent conversion to THA technically difficult because of distortion of the proximal femoral anatomy. Varus and valgus osteotomies may alter neck shaft angle and be rotationally mal-aligned. Rotational mal-alignment can affect the estimation of anteversion of the femoral component. A custom-made femoral prosthesis or intraoperative femoral osteotomy may be necessary for success.

Femoral acetabular impingement

Femoral acetabular impingement encompasses a spectrum of disease patterns and severity. It is a cause of hip pain, restricted hip motion, labral disease, articular cartilage degeneration and secondary osteoarthritis.

It is recognised as a sequela of common paediatric hip conditions such as Perthes' disease and SUFE.

Cam impingement

The cam type of impingement is caused by an overgrowth of the anterior and anterosuperior femoral head-neck junction, leading to an increased peripheral radius of the head entering the acetabulum throughout the range of movement of the hip. The chondral rim of the acetabulum is vulnerable to damage. Predisposing factors include SUFE, mal-union of a femoral neck or head fracture and femoral retroversion.

Pincer impingement

Pincer type impingement occurs because of acetabular overcoverage of the femoral head caused by a deep or retroverted acetabulum. A centre-edge angle of $>40°$ is considered diagnostic of pincer impingement. This results in degeneration, ossification and tears of the anterosuperior portion of the labrum as well as a posteroinferior contrecoup pattern of cartilage loss from the femoral head and corresponding acetabulum. Predisposing factors include acetabular protrusio, acetabular retroversion and mal-union of an acetabular fracture.

Clinical findings

Patients present with hip pain that is located in the anterior part of the groin and sometimes the lateral aspect of the hip but without greater trochanter tenderness. The pain can be sharp and catching, worse with sitting and deep flexion. If the pain involves substantial catching or popping, suspect labral pathology. On clinical examination patients demonstrate a painful range of hip movement, particularly internal rotation and positive impingement test.

The impingement test is performed by placing the patient in the supine position with the hip in 90° flexion and then adducting and internally rotating the hip.

Investigations

- AP and cross-table lateral (groin lateral) radiographs of the hip
- Three-dimensional CT scan to confirm the diagnosis and define the bony lesion
- MRI to evaluate abnormal (bump) head-neck junction and possible labral and chondral pathology

Joint preservation surgery

The objectives are to eliminate abnormal contact between the proximal part of the femur and the acetabulum and to address intra-articular labral and articular cartilage abnormalities. Surgical options include open dislocation, arthroscopy and limited open approaches and arthroscopic techniques alone.

Combined arthroscopy and limited femoral head-neck osteochondroplasty allows evaluation and treatment of intra-articular labral and cartilage injuries whilst offering direct visualisation for osteochondroplasty.

Many surgeons prefer an all-arthroscopic technique of femoral osteochondroplasty, citing better function and patient outcomes. It should be performed fairly cautiously to reduce the risk of femoral neck fracture. Insufficient bony resection is a common cause for revision arthroscopy.

Pigmented villonodular synovitis (PVNS) of the hip

Introduction

PVNS is a proliferative disease of synovial tissue, which affects the knee, hip, ankle and elbow. A slow-growing benign locally invasive tumour of the synovium, the disease usually presents as a monoarticular haemarthrosis, and may exist in a nodular or a diffuse form. The hip is involved in 15% of cases.

Clinical features

- Acute episodic attacks of hip pain and swelling
- Groin pain and restriction of movement
- Always consider PVNS in a younger patient with unexplained hip pain

There are two subtypes:

Diffuse form

- The disease may be active or inactive
- Look for periarticular erosions on radiographs
- A diffuse mass may be present on examination

Nodular form

- Less common than the diffuse form of PVNS
- Does not show the same destructive changes as the diffuse form
- May cause recurrent haemarthrosis and aspirate may be of normal colour (instead of the classic brown colour)

Radiology

Radiographs show cysts on both sides of the joint and are not confined to the weight-bearing areas. MRI will demonstrate hyperplastic synovium.

Management

Ultrasound-guided biopsy is recommended for histological diagnosis. Conservative treatment of symptomatic PVNS of the hip in the young patient has included external beam radiation and open synovectomy, with THA reserved for aggressive end-stage disease. Arthroscopic synovectomy or open synovectomy is viewed as the treatment of choice for the active form of diffuse disease. Radiation may control PVNS in extensive recurrent disease.

Examination corner

Long case 1: PVNS right hip

Radiographs of pelvis

Adult elective orthopaedics oral 1

- The candidate was shown an AP radiograph of the hip of a young woman with rapid deterioration in hip function
- Radiographic features included joint space narrowing and lytic defects in the bone on both sides of the joint
- What findings at surgery would you expect?

Tuberculosis of the hip

Introduction

The hip is the most commonly affected joint and accounts for 15% of all cases of osteoarticular tuberculosis. The initial lesion usually starts as an osteomyelitis in one of the bones adjacent to the joint (osseous tuberculosis). In some cases the disease may begin in the synovium (synovial tuberculosis) but spreads quickly to involve the articular cartilage and bone (articular tuberculosis). A progressive pattern of destruction of the hip occurs in patients who are not treated. Treatment must be instituted early with the aim of salvaging the hip.

Clinical features

There is an insidious onset with aching in the groin and thigh and limp. Later the pain becomes more severe and causes sleep disturbance. A child may complain of night cries', the so-called 'starting pain'.

All movements of the hip are grossly limited by pain and spasm. The leg is scarred and thin, and shortening is often severe because many factors can contribute (adduction deformity, bone destruction, damage to the upper femoral epiphysis).

Radiology

Radiology is often non-specific. The earliest change is *diffuse osteoporosis* but with a normal joint space. There maybe a *lytic lesion* involving either the head of the femur or the acetabulum. The outline of the *articular ends* of the bone becomes *irregular* because of destruction by the disease process.

Management

Chemotherapy is the main basis of management.

Skin traction in a Thomas splint:

- Provides rest of the affected part
- Relieves muscle spasm
- Prevents and corrects deformity
- Maintains joint space
- Minimizes chances of developing a wandering acetabulum

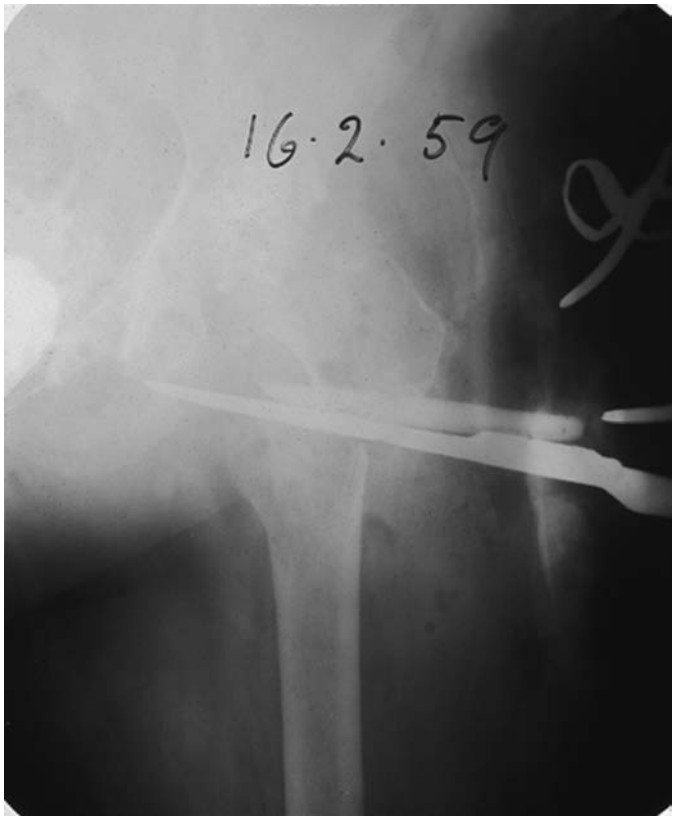

Figure 16.26 Brittain ischiofemoral arthrodesis

Joint arthroplasty:

The timing of total hip replacement (THA) in patients with active tuberculosis (TB) of the hip is controversial, because of the potential risk of reactivation of infection. Hardinge and Sir John Charnley[76] recommended in 1977 postponing THA until any sinuses had not discharged for 20 years, or until the affected hip had been ankylosed for more than 10 years. Another study from the 80s[77] recommended that joint arthroplasty is not performed in the active stage and should only be considered after a safe period of absolute disease quiescence. Ankylosis of the hip/knee may occur spontaneously, and it may be unnecessary to perform arthrodesis. Conversion of ankylosis or arthrodesis should be covered by antituberculosis treatment for 3 months before surgery and 9 months postoperatively. The authors of both studies concluded that there was a low probability of reactivation if:

- >10 years since infection
- Solid arthrodesis
- Previous medical treatment

However, a recent systematic review[78] has suggested that THA in patients with active TB of the hip is a safe procedure, providing symptomatic relief and functional improvement if undertaken in association with extensive debridement and appropriate antituberculosis treatment. They reached this conclusion after reviewing multiple databases referenced articles published between 1950 and 2012. A total of 6 articles were identified, comprising 65 patients. TB was confirmed histologically in all patients. The mean follow-up was 53.2 months (24–108 months). Antituberculosis treatment continued postoperatively for between 6 and 15 months, after debridement and THA. One non-compliant patient had reactivation of infection. At the final follow-up the mean HHS was 91.7 (56–98).

The surgical challenges encountered include scarring and adhesions in the hip area, shortening, anatomical distortion of the acetabulum and femur, LLD and bony defects.

Examination corner

Adult elective orthopaedic oral: Radiograph demonstrating a Brittain ischiofemoral arthrodesis (Figure 16.26)

This is a classic and distinct spot diagnosis of the adult and pathology oral. It is an extra-articular hip arthrodesis used to treat tuberculosis infection. This concept was first popularized by Brittain of Norwich in 1941. Subtrochanteric osteotomy and medial displacement of the femoral shaft with a tibial graft bridging the femur and ischium are carried out. It is a clever concept based on the principle that compression provided by the adduction forces will induce hypertrophy of the tibial graft (as opposed to iliofemoral grafts, which are under distraction). The graft was also extracapsular, i.e. could be performed away from the tuberculous infection. The structure that is particularly at risk when performing an ischiofemoral arthrodesis is the sciatic nerve. This is put at even more risk if there is a severe fixed flexion deformity of the hip as this effectively drags the nerve forward into the plane of the strut graft between the femur and the ischium.

General orthopaedic and adult oral

This was actually a spot MRI diagnosis of spinal tuberculosis with a discussion of the differential diagnosis.

EXAMINER: What are the current recommendations for antituberculosis treatment?

CANDIDATE: Either a triple or four-phase drug treatment. This is the initial intensive phase, which is for a period of 2 months. This is followed by a continuation phase with rifampicin and isoniazid, which is usually continued for a period of 6–9 months.

EXAMINER: What about long-term therapy of 12–18 months.

CANDIDATE: Orthopaedic surgeons initially favoured long-term therapy but the short course therapy of 6–9 months used successfully for pulmonary tuberculosis has been shown to be equally successful with osteoarticular tuberculosis. It is now thought that extending chemotherapy beyond a year is required in only rare circumstances.

EXAMINER: What are the side effects of treatment?

CANDIDATE: Rifampicin: Rashes, hepatitis, orange discoloration of urine, sweat and saliva. Isoniazid: Hepatitis, peripheral neuropathy. Pyrazinamide: Anaemia, arthralgia, hepatitis, gout. Ethambutol: Optic neuritis (red–green colour blindness).

'There were much more interesting things to discuss about this topic: The characteristic MRI differences between secondary metastatic disease, infection and tuberculosis, the indications for surgery with tuberculosis of the spine, etc. The examiners were having none of this and more or less just concentrated on drug treatment of the disease. I must admit I did struggle a bit and the examiners would not let go of it and move on to something else.'

Basic science oral 1

Management of tuberculosis (including drugs).

Outcome measurements

A number of questionnaires have been developed to evaluate outcomes of interventions for OA hip. Six broad dimensions are important: Pain, ability to walk, level of activity, walking capacity, patient satisfaction and clinical examination.

Types of patient-based measures outcome

Disease-specific questionnaires

Most traditional hip outcome measures (e.g. Harris, D'Aubigne, Mayo and Iowa Hip Scores) are disease-specific. Most disease-specific outcome measures have not been validated. The WOMAC hip assessment is a newer, validated, disease-specific outcome measure. It consists of 24 items, assessing three dimensions: Pain, stiffness and physical function.

Patient-specific outcome measures

An example of a patient-specific outcome measure would be the MACTAR scale, in which the patient is asked to list the primary reasons why he or she is undergoing THA.

Region-specific questionnaires

The Oxford Hip Score is a questionnaire designed to assess the patient's perceptions in relation to outcomes of THA.

Functional capacity outcome

This measures functional capacity before and after a medical treatment. The 6-minute walk utilizing the same course and prompts has proven useful in assessing THA patients.

Global outcome measures (generic health status questionnaires)

The SF-36 is a typical global outcome measure.

Examination corner

Basic science oral 1

EXAMINER: Do you know any outcome measurements that can be used to assess the success of primary THA?

CANDIDATE: No.

EXAMINER: Have you heard of the SF-36 or the Nottingham Health Profile or the Oxford Hip Score?

CANDIDATE: I have heard of the Oxford Hip Score.

EXAMINER: What type of outcome measurement is it?

CANDIDATE: Sorry, I am not sure of your question.

EXAMINER: Let's move on. How does heparin work?

(Fail)

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Chapter 17

Knee oral core topics

Khaled M. Sarraf and Deiary F. Kader

Menisci

Anatomy

Menisci are crescent-shaped fibrocartilaginous structures that are triangular in cross-section. The lateral meniscus is more circular and covers 70% of the lateral tibial plateau, while the medial meniscus is C-shaped and covers 50% of the medial tibial plateau.

The menisci are composed primarily of type I collagen (90%). The fibres are arranged radially and longitudinally (circumferential). Longitudinal fibres help dissipate hoop stresses in the meniscus. The hoop tension is lost when a single radial cut or tear extends to the capsular margin. The extracellular matrix (ECM) consists of proteoglycans, glycoproteins and elastin.

By virtue of their specialized structure, high fixed-charge density, and charge-charge repulsion forces, proteoglycans in the ECM are responsible for hydration and provide the tissue with a high capacity to resist compressive loads (Figure 17.1).

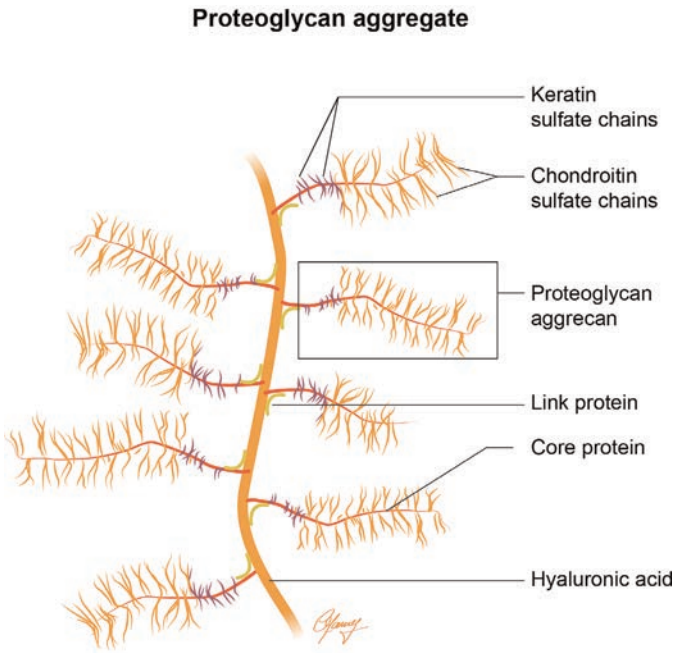


Figure 17.1 Proteoglycans. Aggrecan is the major large proteoglycan of the meniscus.Its main function is to enable the meniscus to absorb water, whose confinement supports the tissue under compression

Mensical tissue is considered biomechanically as a biphasic tissue. As the mensiscus is loaded,water is forced from the matrix and when the load is released, the negatively charged GAGs attract water back into the matrix, rehydrating the tissue.

During flexion the posterior excursion of the lateral meniscus is 11.2 mm, which is nearly twice that of the medial meniscus[1]. Therefore, medial meniscal tears are three times more common than that of the lateral meniscus (Figure 17.2).

Blood supply

- Branches of the lateral, middle and medial genicular arteries from the popliteal artery provide the major vascularization to the inferior and superior aspects of each meniscus
- The meniscus is vascularized by the perimeniscal capillary plexus (PCP), which is formed by branches of the lateral, middle and medial genicular arteries
- At birth the whole meniscus is vascular
- In adults only the outer 10–30% of the meniscus is vascular[2]. Hence, the outer 30% is called the red zone (PCP is present in the red zone)
- The white zone is the inner avascular portion of the meniscus
- The intermediate portion is called the red–white zone
- The red zone can heal via fibrovascular scar formation

Innervation

- Peripheral two-thirds of the meniscus is innervated by type I and II nerve endings
- The posterior horn has the highest concentration of mechanoreceptors

Functions of menisci

Load bearing

At least 50% of the compressive load of the knee joint is transmitted through the meniscus in extension, and around 85% is transmitted in 90° flexion. In the meniscectomized knee the contact area is reduced to approximately 50%. Partial meniscectomy also increases the contact pressures

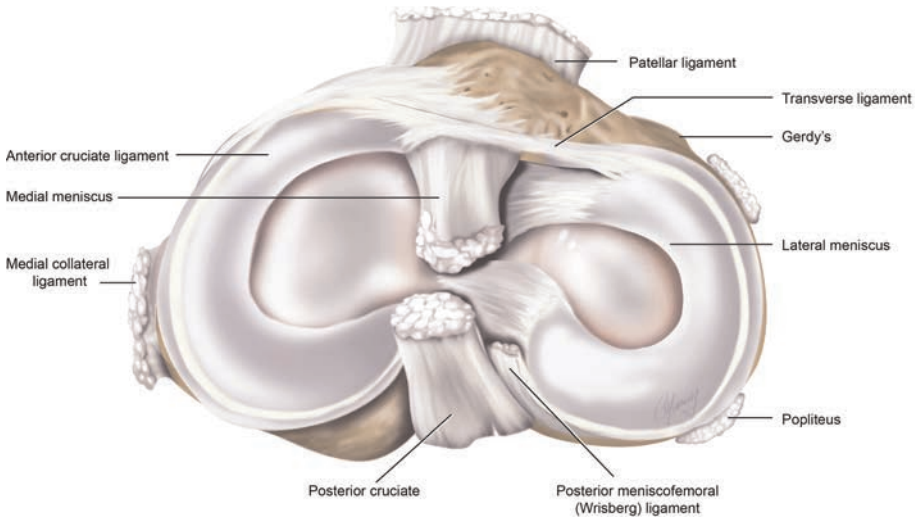


Figure 17.2 Axial anatomical view of the knee

Shock absorption

Menisci attenuate the intermittent shock waves generated by impulse loading during gait.The shock-absorbing capacity of normal knees is ~20% higher than in meniscectomized knees. The meniscal tissue has shock-absorption capacity because it has nearly half of the stiffness of articular cartilage (more elastic)

Articular conformity

The meniscus displaces in an anteroposterior (AP) direction as the knee passes through its range of movement. In addition, the menisci deform to remain in constant congruity to the tibial and femoral articular cartilage. Deformable properties of the meniscus aids load transmission and shock absorption

Knee joint stability

The medial meniscus in particular controls AP translation. Meniscectomy alone may not seriously affect stability. However, in association with anterior cruciate ligament (ACL) tears, meniscectomy significantly increases the anterior knee laxity

Lubrication

The menisci distribute synovial fluid and promotes a viscous hydrodynamic action needed for fluid-film lubrication

Proprioception

This has been inferred from the finding of type 1 and type 2 nerve endings in the anterior and posterior horns of the menisci

Prevention of soft-tissue impingement during joint motion

Biomechanics

The peripheral one-third of the meniscus plays a crucial part in joint stability and load transmission. The inner two-thirds of the meniscus plays an important role in maximizing joint contact area and increasing shock absorption. The menisci are attached peripherally by the coronary ligaments and are connected anteriorly by the transverse (intrameniscal) ligament (Figure 17.2). They move anteriorly in extension and posteriorly in flexion. The lateral meniscus is more mobile as it has less soft-tissue attachments.

Examination corner

Basic science

EXAMINER: What is the main constituent of the meniscus?

The meniscus is a fibrocartilaginous structure consisting of extracellular matrix (primarily water, collagen, proteoglycans, elastin and glycoproteins) as well as cells which are mainly fibrochondrocytes.

EXAMINER: What type of collagen is found in the meniscus?

The main collagen found is type I. There are small amounts of type II, III and V.

EXAMINER: Draw the shape of the meniscus including the orientation of the collagen fibres and describe how they aid with its function.

The menisci is microscopically arranged into three distinct layers: Superficial, lamellar and deep. The superficial layer exists on both the tibial and femoral surfaces.In the superior region there is an unorganized random arrangement of collagen fibrils, which contrasts with the inferior region in which the fibers are more radially orientated. The lamellar layer that also exists on both the femoral and tibial sides has fibers that are randomly orientated. The main part of the meniscal tissue is located between the two lamellar layers. A dense framework of circumferential coarse type I collagen fibres lie in this layer, cross-linked with radial fibers from the periphery. The radial fibres may act as a "tie" holding the circumferential fibres together, providing structural rigidity against compressive forces and resisting longitudinal splitting of the menisci (Figure 17.3).

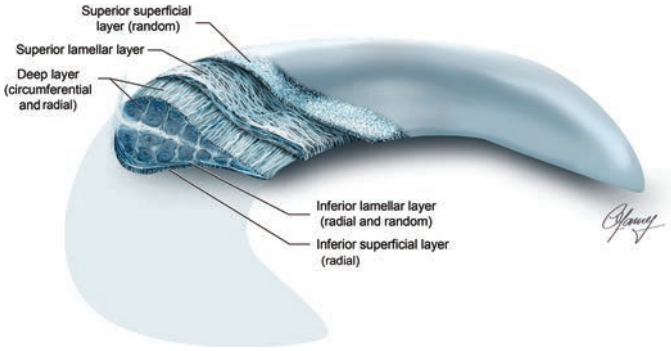


Figure 17.3 Diagram demonstrating the collagen fiber ultrastructure and orientation within the meniscus: Collagen orientations are of three main types: (1) circumferential, (2) radial and (3) random. Circumferential are mainly found in the deep zone, Radial fibers are spread throughout the deep zone and are also present on the periphery and horns of the meniscus in the lamellar zone. Despite the presence of radial fibers, random fiber orientation dominates the lamellar zone. In the superficial zone fiber orientation is random in the superior region and more radially orientated in the inferior region.

During weight-bearing, the compressive forces exerted are resisted by the hoop stresses in the circumferential fibres within the meniscus. The hoop tension is lost when a single radial cut or tear extends to the capsular margin. In contrast, shear forces within the meniscus are resisted by the radial collagen fibres of the meniscus.

The combination of the fibres allow the meniscal structure to expand under compressive forces and, hence, increase the contact area within the knee joint.

Biomechanical studies have shown that 85% of the compressive load is taken by the meniscus during flexion, while around 50% of the compressive load is transmitted in extension.

EXAMINER: What do you mean by hoop stress?

The development of 'hoop stress' within the meniscus depends on intact anterior and posterior attachments. Hoop stress also relies on the conversion of axial load into tensile strain through intact longitudinally oriented collagen fibers. The compression of the menisci by the tibia and femur generates outward forces that push the meniscus out from between the bones. The circumferential tension in the menisci counteracts this radial force. Hoop stress is the stress in a direction perpendicular to the axis of an item. As the thickness of the item decreases the hoop stress increases.

EXAMINER: What you have described above also relies on the meniscal attachments being intact. Could you please discuss the various attachments of both menisci?

The lateral meniscus which is more circular in its morphology covers a wider area of the tibial plateau it lies on in comparison to the medial meniscus. It is attached to the tibial plateau via the coronary ligament. The posterior horn is attached to the medial femoral condyle by two menisco-femoral ligaments. Ligament of Humphry which lies anterior to the PCL and ligament of Wrisberg which lies posterior to the PCL (see Figure 17.2). The lateral meniscus, unlike the medial, has no attachment to its adjacent lateral collateral ligament (LCL) and only has loose peripheral attachments to the joint capsule.

In contrast, the medial meniscus is more C-shaped. The posterior horn is attached to the posterior intercondylar fossa of the tibia, while the anterior horn attaches anterior to the ACL insertion and the medial tibial spine. The medial meniscus is firmly attached to the joint capsule throughout its periphery, including the deep medial collateral ligament which is a condensation within the capsule.

Meniscal tears

There are two main types of tears. **Traumatic tears** usually occur in younger patients owing to sporting injuries. **Degenerative tears** occur in the older age group – In 60% of the population over 65 years. The majority are asymptomatic and occur in degenerative joint disease and can have an insidious onset.

Medial meniscal tears are three times more common than lateral meniscal tears, however, lateral meniscal tears are more prevalent in acute ACL injuries.

Partial meniscectomy (50% excision) increases the peak contact stress by 43% and reduces the contact area by 20% while total meniscectomy increases the peak contact stress by 130% and reduces the contact area by 50%[3].

This will ultimately reduce the joint's shock absorbing capacity and load sharing ability. The effect of performing such procedures is more profound on the lateral side in comparison with the medial due to the morphology of the tibial plateau. The medial tibial plateau is concave while the lateral is convex, and, hence, the significant increase in contact stress in the latter.

Meniscal root tear is a complete disruption of the circumferential fibres posteriorly. Usually presents with a history of snapping knee in deep flexion and can be diagnosed by MRI scan, which usually shows root avulsion and meniscal extrusion. Posterior root tear and total meniscectomy have biomechanical similarities and both can cause significant change in contact pressure[4].

Tear orientation and appearance (Figure 17.4)

- Incomplete/complete longitudinal, bucket handle
- Displaced bucket handle
- Horizontal cleavage tear
- Oblique or parrot beak
- Flap, displaced flap and double flap
- Radial tear
- Complex tear is a combination of the above. Degenerative tears are usually complex in nature
- Zip tear meniscofemoral ligament tearing through the posterior horn of the lateral meniscus

Examination corner

Basic science oral 1

The examiner shows a photograph of torn meniscus and asks:

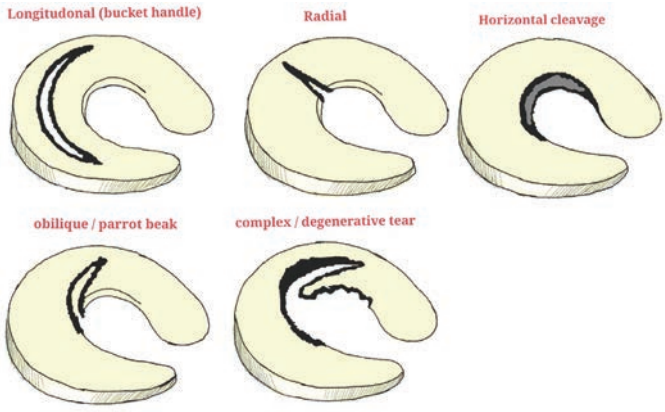


Figure 17.4 Different types of common meniscal tears

Tell us about different types of tears that can occur in the meniscus? (See Figure 17.4)
Which one would you repair?

There are various factors that I would consider before deciding whether to repair a meniscal tear. There are patient factors as well as local factors. Patient factors include young ages as patients under 30 do better than older the 30. I would also want to know the level of activity of the patient including their occupation; this is in order to counsel the patient appropriately. Some tears are more amenable to repair than others and this depends on the time since the tear – Early repair is better than late; the type of tear – Red on red as opposed to red on white or white on white; and whether or not there is a concurrent ligamentous injury – Ligament injuries need to be addressed to ensure stability and restoration of normal load onto the meniscus. There is also studies showing that meniscal tears are more favourable when performed with an ACL reconstruction due to the presence of platelet-derived growth factors (PGDF)[5].

How does an ACL rupture affects the medial meniscus?

An ACL rupture usually leads to abnormal strain exerted onto the menisci upon weight-bearing and throughout the range of motion of the knee. This strain particularly affects the posterior horn of the medial meniscus . 40% of tears in ACL injuries are of the peripheral posterior horn of the medial meniscus[6].

Around 50% of ACL injuries have a concurrent meniscal injury or articular cartilage. Repeated episodes of instability secondary to an ACL injury predisposes to meniscal tears. In chronic ACL instability, up to 90% of patients will have meniscal injury after 10 or more years. Similarly, the prevalence of articular cartilage lesions can be as high as 70% in ACL-deficient patients after 10 years. Studies of meniscectomy confirm the importance of meniscal function and subsequent loss as a risk factor for the development of knee osteoarthritis[7].

Meniscal repair

The majority of repairable menisci are associated with ACL rupture. It is reported that there is a 93% healing rate in meniscal repairs who are undergoing an anterior cruciate ligament reconstruction compared to 50% in meniscal repair alone[8]. This is speculated to be due to the local growth and clotting factors including stem cells and platelet-derived growth factors that are released during injury and from the femoral and tibial bone drilling[5]. Generally only 30% of tears in patients younger than 20 years old are repairable[8]. Tears in the peripheral third have the highest potential for healing due to the presence of the blood supply.

Consider the following:

- Patient's age: Better results when patients are below 30 years of age
- Activity level and occupation
- Chronicity: Better results if carried out <8 weeks
- Blood supply, location, quality of meniscal tissue, type and length of tear (this is a primary determinant of healing)
- Associated ligamentous injury
- Leg axial alignment

Relative contraindications to repair

- Stable tear (partial-thickness tear)
- Peripheral tear <10 mm long that cannot be displaced
- Complex, degenerative and central/radial tears are best excised partially

Types of meniscal repair

- Inside-out technique with vertical mattress suture is still the gold-standard technique
- Outside-in: Versatile access, less expensive instruments and safe
- All-inside: Becoming very popular with the developments of new devices and reports of >80% success rate. They allow the sutures to be tensioned once inserted
- Open repair. Rarely performed

Vertical mattress sutures are more reliable than horizontal mattress, as they are perpendicular to the circumferential fibres and have a less chance of cutting out.

To optimize healing one could use:

- Fibrin clot
- Rasps and shavers are used to freshen both sides of the tear prior to repair (most common in regular practice)
- Trephination of the meniscus with a spinal needle
- Vascularized synovial flaps
- Autologous blood clot
- Parameniscal synovial abrasion
- Endothelial cell growth factor
- Fibrin sealants
- Notch (non-articulating) microfracture to produce bleeding
- These biological factors are an important part of the surgical intervention

Complications

- Excluding failure to heal which is usually quoted at 15–20%[9] and re-tearing, neurovascular injury is the commonest
- Medially: Injury to the saphenous nerve and its infrapatellar branch 1–2.5%
- Laterally: Popliteal artery and peroneal nerve injury 1%

Meniscal allograft transplantation

This is still regarded as an experimental procedure. It is carried out to prevent joint deterioration following total meniscectomy and to help improve knee stability in patients with ligamentous instability. It is more commonly performed on the young and on the lateral meniscus due to its biomechanical importance.

Indications

Consider:

- Patient's age (best results in those <20 years old)
- Symptoms (in the future may be done prophylactically)
- Knee stability, ACL competency and alignment
- Compartment wear (Outerbridge I and II better outcome). Diffuse grade III or the presence of grade IV lesions, with joint space narrowing, are considered relative contraindications

Graft

- Fresh-frozen
- Freeze-dried grafts
- Collagen or synthetic grafts

The meniscus is immunologically privileged owing to dense matrix isolating the cells. These grafts need to be appropriately sized to match the patients' native meniscus as much as possible.

Technique

- Open
- Arthroscopically assisted

Classic reference

Fairbank TJ. Knee joint changes after meniscectomy *J Bone Joint Surg Br*. 1948;30:664–70.

This was the first detailed article to characterize the radiographic changes in the knee post meniscectomy. Fairbank also offered an explanation for the changes to the articular surface. He deduced that the changes in the articular surface are caused by overload due to loss of the meniscal tissue. He was the first to suggest that the meniscus may have a load bearing function.

Classic reference

Tapper EM, Hoover NW. Late results after meniscectomy *J Bone Joint Surg Am*. 1969;51:517–26

This is a landmark paper, which was one of the first to report the deleterious effect of meniscal excision. At the time it was a common belief among surgeons that the meniscus could be totally excised without long-term harm to the patient. In fact some surgeons predicted that degenerative arthritis and disability were inevitable if a torn meniscus was not completely removed. This article stimulated many subsequent basic science and clinical studies that determined the many important functions of the menisci and the natural history of the post meniscectomy knee

Classic reference

Johnson RJ, Kettelkamp DB, Clark W, Leaverton P. Factors effecting late results after meniscectomy *J Bone Joint Surg Am*. 1974;56:719–29.

Total meniscectomy is associated with instability and rapid severe articular degeneration.
The long-term outcomes after meniscectomy are unsatisfactory for the majority of patients according to a range of measures including activity and pain ratings, knee joint instability, knee joint structure and knee biomechanics during level walking.
The findings of this study concur with Tapper et al. and reports on the consequences of meniscectomy. The authors used stringent criteria in their clinical assessment and compared the two legs using the unaffected side as a control. This work reinforced the importance of the meniscus in keeping the knee healthy and strongly recommended that the meniscus should be removed only when it is definitely abnormal.

Examination corner

Basic science oral 1

- What is the composition and structure of the menisci?
- Describe collagen fibres arrangement.
- What is the role of menisci in load distribution?
- When do you consider repairing a meniscal tear?
- How does meniscal root tear affect joint contact pressure?

The meniscal root has a major role in the properties of the meniscus within the knee. A meniscal root tear completely disrupts the circumferential fibres of the meniscus leading to extrusion of the meniscus. This behaves in a similar manner in regards to the load patterns on the knee to a complete meniscetomy. This is more pertinent in a posterior root tear. Total meniscectomies increase the peak contact stresses in the knee by 235%, reduce the joint's shock absorbing capacity and its load sharing ability.

Meniscal cyst

Aetiology

- Cause unknown
- Myxoid degeneration of stressed fibrocartilage
- Probably traumatic in origin

- Meniscal tear may create a one-way valve allowing for cysts to form

Pathology

- Contain gelatinous fluid, surrounded by thick fibrous tissue
- Nearly always associated with a small, horizontal cleavage tear in the meniscus
- Isolated cysts without meniscal pathology have been reported
- More likely to occur laterally

Clinical features

- Insidious onset of discomfort
- Point tender cyst on palpation
- Symptoms are intermittent or related to activity
- Lump is situated at or slightly below the joint line
- Usually anterior to collateral ligament
- Seen most easily with the knee slightly flexed (<45°)
- Lateral cysts are firm, medial cysts are usually larger and softer
- Pisani's sign (cyst size decreases with knee flexion)

Differential diagnosis

- Ganglia: Superficial, not as hard and unconnected to the joint
- Calcified deposits in the collateral ligament: Show on radiographs
- Prolapsed torn meniscus (pseudocyst)
- Sebaceous cyst
- Bursitis
- Various tumours: Sarcoma, lipoma, fibroma and histiocytoma
- PVNS

Management

- Depends on symptoms, size, location and relation to meniscal tear
- If contiguous with the meniscal tear the meniscus is debrided and the cyst is decompressed arthroscopically or with needle aspiration
- If the cyst is distinct or very large, an open excision is more successful

Congenital discoid menisci

Abnormal development of the meniscus can lead to a hypertrophic and discoid shaped meniscus. In children, most meniscal tears are caused by congenitally discoid menisci. The incidence of discoid meniscus is 5% in Anglo-Americans, but can go up to 20% in Asians. It is most commonly lateral (rarely medial: Incidence of 4–15% vs 0.06–0.03%) and 25% of patients have bilateral discoid menisci.

Aetiology

1. Possibly failure of resorption of the central portion of the meniscus during development
2. Secondary to instability of the meniscus during development, subsequent to failure of attachment of the meniscotibial (coronary) ligament to the posterior horn (type III)

Classification (Watanabe 1974)

Type I – Complete

The meniscus covers the whole tibial plateau, causing inadequate visualisation on arthroscopy

Type II – Incomplete

The central portion extends further across the tibial plateau than normal

Type III – Wrisberg variant

Involves deficiency of attachment to the posterior horn meniscotibial ligaments, so the posterior horn is only secured by the meniscofemoral ligaments

Clinical features

The discoid lateral meniscus is usually asymptomatic. Snapping knee syndrome (popping knee syndrome) in children <10 years old is usually associated with type III. The knee snaps spontaneously, causing momentary pain and apprehension. A characteristic clunk may be felt at 110° as the knee is bent, or at 10° as the knee is straightened.

A McMurray test may cause an obvious pop (referred to as popping knee syndrome), with temporary subluxation of the posterior horn and occasional locking.

In older children, the discoid meniscus usually presents with the symptoms of a meniscal tear. Types I and II commonly have longitudinal or horizontal tears. Type III usually have no tears, but may exhibit degenerative changes. All types may have radial, bucket-handle or complex tears.

Typical radiographic findings

- Widened joint space
- Squaring of the condyle
- Ridging
- Cupping of the lateral tibial plateau
- Hypoplastic lateral intercondylar spine

MRI appearance

- Visualisation of the meniscus across the entire compartment in three consecutive cuts (usually in the lateral compartment)

Management

- If asymptomatic or there is only a clunk (not associated with a tear) leave it alone

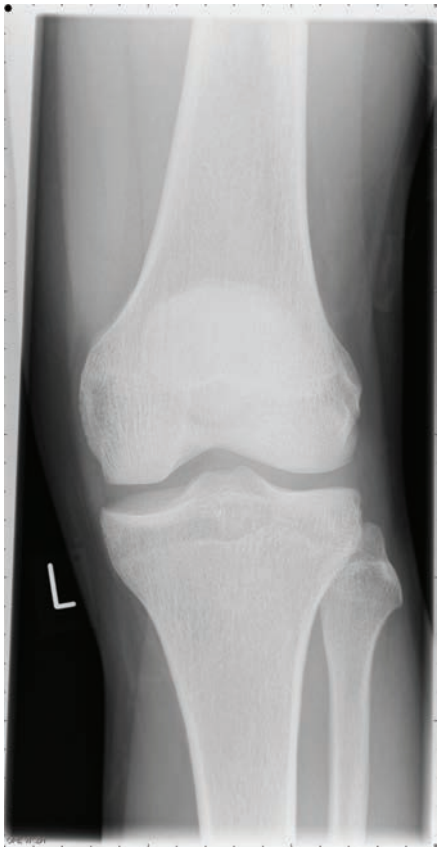


Figure 17.5 Less common discoid medial meniscus revealing the classic radiological features

- Symptomatic patients with a type I or II discoid meniscus may be treated with arthroscopic debridement and contouring of the central portion, leaving a rim of 6–8 mm (saucerisation). Peripheral tears in the vascularized zone should be repaired. Often the remaining rim is degenerate and may necessitate total meniscectomy
- Type III menisci, traditionally treated with total meniscectomy, are now usually managed with meniscal repair and reattachment of the posterior horn to the tibial plateau
- Further surgery is often required for recurrent tears
- OA is common following meniscectomy
- There is no increase in the risk of OA in asymptomatic patients

Osteochondritis dissecans (OCD)

Definition

- A lesion of subchondral bone that results in subchondral delamination and sequestration with or without articular mantle involvement (Stanitski)
- Subgroups: Juvenile, adolescent and adult types
- Peaks in preteen years
- Male : Female ratio is 5 : 3
- Bilateral 20%
- Medial:lateral condyle ratio is 80 : 20 – Most common on the non-weight-bearing lateral aspect of the medial femoral condyle. When found on the lateral femoral condyle it is more commonly located on the central portion of the condyle
- Patella lesion in 10%

Aetiology

Aetiology is unknown. It may be:

- Traumatic: lesion is thought to be due to macrotrauma or repeated microtrauma
- Vascular: possibly AVN (adult form more associated with vascular cause)
- Hereditary/congenital: abnormal epiphyseal ossification
- The condition tends to occur in children during increased physeal activity

Clinical presentation

- Non-specific, poorly localized pain
- Activity-related pain
- Stiffness
- Swelling
- Mechanical symptoms with locking
- Antalgic gait
- Effusion in unstable lesion
- Localized tenderness
- Wilson's sign (induction of pain as the knee is passively extended while the tibia is held in internal rotation: Tibial spine contacts OCD on the lateral aspect of the medial femoral condyle at 30° of flexion). Not very sensitive nor specific to osteochondritis dessicans

Pappas classification (according to age at detection)

- Category I: Below age 12 (excellent prognosis)
- Category II: Between 12 and 20 years
- Category III: Above 20 years

Prognosis

- Healing potential is high in juvenile (75%)
- Adolescent prognosis is unpredictable (50% heal)
- Healing is markedly reduced in those with a mature skeleton with possible premature OA especially if left untreated. It is usually symptomatic in adults
- Other than age, location of the lesion such as the lateral femoral condyle or the patella have poorer prognosis. Similarly, the appearance of synovial fluid behind the lesion on MRI correlates to a worse prognosis
- A systematic review by Harris JD et al.[10] to determine which surgical technique has improved outcomes and enabled athletes to return to their preinjury level of sports revealed there is little high-level evidence to support one form of treatment over another. However, cartilage repair and restoration appeared to fair better when compared to

microfracture when measured against speed of return to sport. The overall rate of return to pre-injury level of sports was 66%

Diagnostic imaging

- Preferred radiographic view is the tunnel view. The lateral radiograph typically demonstrates that the affected segment of the medial femoral condyle is contained within a region defined by the intersection of the Blumensaat line and the continuation of the line along the posterior femoral cortex (referred to as Harding area)
- MRI helps in staging the lesion

Examination corner

- An 11-year-old football player complains of ongoing vague left knee pain associated with activity. She denies any swelling, locking, clicking or giving way. She sustained a further acute mild injury while playing football that exacerbated her pain and brought her to the ED. On examination, you find anterolateral focal tenderness, mild effusion but no signs of instability. This is her x-ray (Figure 17.6), what do you see?

This is a horizontal beam lateral knee radiograph of a skeletally immature patient revealing a effusion, more specifically a lipohaemarthrosis with a bone fleck present just distal to the articular surface of the condyle. I am unable to determine whether this is from the more common medial condyle or lateral as per the patients focal tenderness. I would need an AP radiograph in the first instance to help me establish that this is indeed a bone fragment and also where it is coming from.

- What other investigation would you like to perform to obtain more information?

An MRI scan

- Here it is (Figure 17.7). Explain what you are looking for on an MRI scan in this condition in general, and then tell me how would you proceed in treating this child?

I would look at T2-weighted MRI sequences in order to highlight evidence of linear high-intensity signals between the lesion and parent bone which would indicate the degree of stability of the lesion. If the articular cartilage over the osteochondritis dissecans fragment is intact then no high signal (fluid) will be observed posterior to the fragment. However, if the fragment is separated from the underlying subchondral bone or the cartilage is fractured, then the synovial fluid would track around the perimeter of the fragment and indicate instability. Management is controversial as per the last recommendations from the ROCK group (Research on OsteoChondritis of the Knee)[11]. Generally treatment is determined on the age of the patient and the characteristics of the OCD in terms of its stability. In this case the T1- and T2-weighted sagittal MRI scans reveal a large articular defect on the lateral femoral condyle, I cannot visualize the fragment that is apparent on the lateral x-ray. Given the patient is 11 years old and this is an acute injury I would attempt to fix this loose fragment as it appears to be fairly large. I would perform this with a headless screw. If the fragment is completely detached and well rounded (chronic) then I would remove it and perform microfracture to the bed. I would consent the patient for all options and take an intraoperative decision depending on my findings.

- What are the other options of fixation in an unstable OCD?

There are other fixation devices such as absorbable headless pins or darts, bone grafting, fixation with autograft osteochondral plugs, and salvage procedures such as autologous or matrix-induced chondrocyte implantation (ACI or MACI) and fresh osteochondral allografts.

- You mentioned age as a risk factor. Please explain.

Patients younger than 12 years of age have an excellent prognosis as per the Pappa's classification. There is a high healing potential in the juvenile with an open physis. Healing becomes less predictable in the adolescent age of 12–20 and markedly reduced beyond skeletal maturity. Many OCD lesions in the juveniles heal spontaneously with activity modification[11].

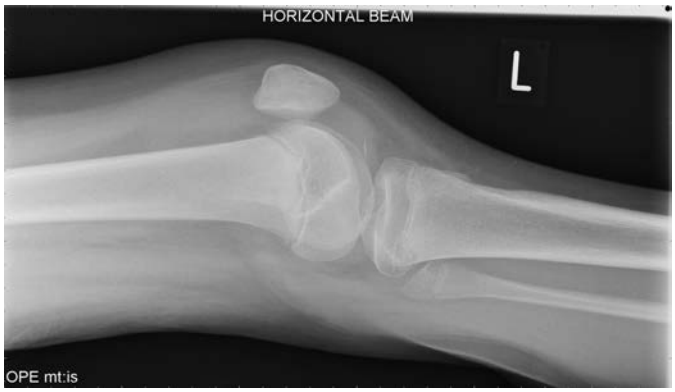


Figure 17.6 Lateral knee radiograph with OCD

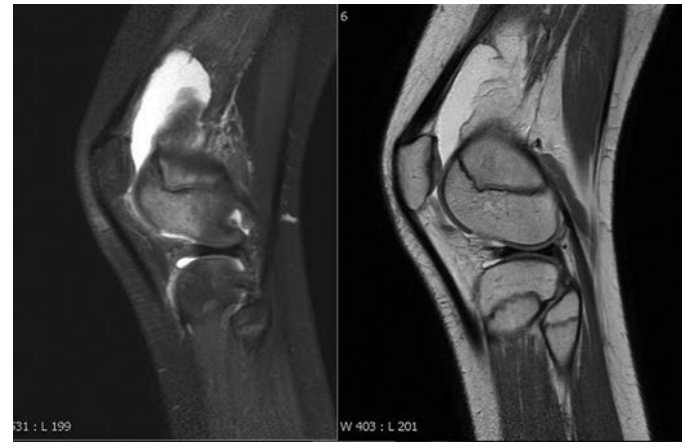


Figure 17.7 Sagittal knee MRI scan in T1 and T2 revealing and effusion with an OCD of the lateral femoral condyle

Guhl arthroscopic classification

- Intact lesion 1–3 cm (in situ, soft area covered by intact cartilage)
- Early separation (stable flap)
- Partially detached (flap attached by hinge)
- Complete detachment (full-thickness loss within bed or displaced)

Stage the lesion by radiographs, MRI and arthroscopy.

Management

- In young patients (open physis, category I, juvenile OCD), Which also includes asymptomatic lesions in adults → activity modification with restricted weight-bearing. 50–75% will heal without fragmentation
- OCD in young adults is usually symptomatic and almost invariably leads to early-onset OA if large enough, unless treated
- In situ lesions → retrograde or anterograde drilling (where indicated)
- Early separation stage → secure with headless screws, cannulated screws, bone pegs or equivalent. 85% healing rate in juvenile OCD. Metal work removal might be necessary (ensure head of device is buried within cartilage)
- Incompletely detached → remove underlying fibrous tissue, perform some form of chondroplasty and then fix the flap as above
- Completely detached → removal of the loose body (often too damaged to replace) possibly followed by:
  - Abrasion chondroplasty (microfracture): This usually leads to formation of fibrocartilagenous tissue covering the defect. There has been improved outcomes in skeletally immature patients
  - Osteochondral graft in the form of allograft plugs or autografts (OATS): This can be done either arthroscopically (lesions under 30 mm) or via an arthrotomy (lesions larger than 30 mm)
  - Autologous chondrocyte implantation (ACI) or matrix-induced chondrocyte implantation (MACI): This involves a two stage procedure, starting with articular cartilage harvest. This harvested cartilage is sent to a laboratory, where the chondrocytes are extracted from the matrix and are cultured. At 6 weeks the chondrocytes are reimplanted in the defect. The main difference between the two procedures is that with ACI the cells are injected under a prepared periosteal patch over the defect while in MACI the cells are embedded with a matrix or scafold. MACI evolved out of ACI and is not available in countries such as the USA yet

Table 17.1 Outerbridge arthroscopic grading system

Grade 0	Normal cartilage
Grade I	Softening and swelling
Grade II	Partial thickness defect, fissures 1.5 cm diameter
Grade III	Fissures down to subchondral bone, diameter > 1.5 cm
Grade IV	Exposed subchondral bone

Articular cartilage injury

- This is a separate entity from OCD. It is usually related to rotational force and direct trauma
- It is located in weight-bearing areas such as the medial femoral condyle
- Aritcular cartilage decreases the friction within the joint by fluid film formation, the presence of synovial fluid and the fact that is has elastic deformation properties. It also distributes the load within the knee
- It is avascular, aneural and has no lymph drainage
- Articular cartilage injury is classified as linear, satellite, flap, crater, fibrillation and degenerate lesions
- Any injury above the tidemark has a poor potential for healing due to the avascularity of this region
- Any injury extending below the tidemark that penetrates the subchondral bone has a better potential for healing as it causes an inflammatory response and may heal with fibrocartilage
- Not all defects are symptomatic
- Articular cartilage defects have no pathognomonic symptoms or signs and they frequently coexist with meniscal tears, patellofemoral dysfunction and early OA
- Femoral condyle lesions cause pain at or close to the joint line aggravated by running and descending stairs

Table 17.2 Classification of chondral lesions according to the ICRS system

Normal	Grade 0
Almost normal	Grade 1a – Superficial lesions/softening Grade 1b – As 1a and/or superficial fissures and cracks
Abnormal	Grade 2 – Extent < 0% of thickness
Severe lesion	Grade 3a – Extent >50% of thickness Grade 3b – Down to the calcified layer Grade 3c – Down to subchondral bone (without penetrating) Grade 3d – Includes bulging of the cartilage around the lesion
Very severe lesion	Grade 4a – Penetration of subchondral bone but not across the entire diameter of the defect Grade 4b – Penetration across the full diameter of the defect

Surgical treatment

- Multiple complementary procedures are available and still evolving
- The biomechanical environment needs to be corrected

Treatment options

- **Debridement**: Removing loose flaps only
- **Microfracture**: Usually the first line of treatment for full-thickness or near full-thickness articular cartilage injuries that measure 2 cm or less. It involves the creation of multiple subchondral perforations in the cartilage deficient areas to allow (in theory) sustained reparative response and allow healing with fibrocartilage from the mesenchymal stem cells (MSCs) that are sourced from the subchondral bone/blood vessels

  Mainly type 1 cartilage – Low stiffness and poor wear characteristics over time

  Technique: Obtain vertical borders, remove calcific bone bed and make multiple holes 3–4 mm apart with chondral pick. The defect and site of microfracture are not covered. This procedure was popularised by Steadman

  This should not be performed in uncontained defects and in the presence of axial mal-alignment.

Poor outcomes

- Obesity
- Smoking
- Inflammatory conditions
- Mal-alignment
- Ligamentous laxity
- **Rehabilitation**: NWB/Toe-touch weight-bearing for 6–8 weeks with full passive ROM (unless PF joint microfracture). No sports for 6 months
  - Better outcomes in patients under 40 years of age
  - Best results are in the femoral condylar lesions
  - Recently microfracture has been supplemented with barrier implantation to minimize leakage of the marrow elements
- **Autologous chondrocyte implantation (ACI)** continues to be under evaluation and has promising early results[13]. There is no size limitation and it produces hyaline-like cartilage
  - High cost
  - Requires two procedures: Arthroscopic biopsy and open implantation
  - Recently collagen membranes have been used instead of periosteum
- **Matrix induced autologous chondrocyte implantation (MACI)**

The principle is transplanting living viable cells that are capable of synthesizing and maintaining a cartilaginous implant. The end result is similar to hyaline cartilage.

This procedure can only be performed on a stable joint with no mal-alignment.

- **Autologous matrix induced chondrogenesis (AMIC)**
  - A one-step procedure combining microfracture with the application of a collagen I/III membrane to protect the without initial blood clot in order to serve as a scaffold for the developing chondrocytes. This clot is often descriped as a super clot that is rich in the essential factors (e.g. progenitor cells, mesenchymal stem cells (MSCs), cytokines and growth factors) necessary to form new cartilage
  - Described indications are symptomatic full-thickness chondral/subchondral defects in the major joints, post-traumatic or osteochondrosis dissecans, located in weight-bearing areas
  - This procedure should not be performed on kissing lesions, inflammatory disease, associated fracture, or on generalized OA
- **Autologous osteochondral transfer**
  - Also called mosaicplasty or osteochondral autograft transfer (OAT)
  - Involves transferring osteochondral plugs from relatively non-load bearing site to weight-bearing defect sites in the knee. Most common harvest sites include the superior trochlear ridge and the intercondylar notch area in the knee. The plug site should also have relatively thick hyaline cartilage, and easily accessible in an open or arthroscopic technique
  - Usually for small lesions <2 cm^2
  - Plugs should be cylindrical, at least 8 mm long and 4–12 mm diameter
  - The procedure is technique-sensitive and operator-dependent
  - Requires mini-open approach
  - Donor site morbidity remains an issue
- **Osteochondral allograft**
  - Transplanting fresh osteochondral allograft containing living chondrocyte into the defect
  - There is no size limitation
  - Used in post-traumatic reconstruction, OCD and osteonecrosis
  - Disease transmission is a potential hazard
  - Problems with graft availably and high cost

Synthetic osteochondral grafts (e.g. biomatrix, trufit plug)

- Remains controversial
- No long-term studies with good clinical outcomes
- Less morbidity to the patient

- Some older types negatively affected adjacent healthy articular cartilage
- **Focal resurfacing implants (HemiCAP)**
  - Indicated in middle-aged patients with symptomatic weight-bearing full-thickness defects
  - Flushed implants do not increase peak contact pressure.[14] Variable results reported

Examination corner

Basic science oral 1

You are likely to be asked by the examiner to draw the structure of articular cartilage and explain its function as you go along (Figure 17.8).

- What is the function of articular cartilage?

Its primary function is to provide a very low friction surface to allow smooth articulation of the joint. The coefficient of friction of articular cartilage is as low as 0.002. The biomedical industry has not been able to replicate this property to improve arthroplasty function. It also serves as a shock absorber.

- **What are its main components?**

Water (65–80% wet weight), collagen (10–20% wet weight, >50% dry weight), proteoglycans (10–15% wet weight) and chondrocytes (5% wet weight).

- **What is the primary collagen type found in articular cartilage?**

The collagen gives the articular cartilage its tensile stiffness. Type II constitutes 90% of the collagen present. Other types present include VI, XI and X (type X being only found in the calcified zone).

- **Describe zones of articular cartilage (you may use a diagram).**

Articular cartilage is divided into four zones: 1. Superficial(tangential): 10–20% thickness 2. Middle(transitional): 40–60% thickness 3. Deep(radial): 30% thickness then we have the Tidemark followed by 4. zone of calcified cartilage.

- **How do each zone differ in regards to their structure and content?**

- The superficial zone has relatively low proteoglycan content and contains no cells. It is, however, rich in collagen which is arranged in parallel to the joint surface and allows good resistance to shear forces. As the water concentration is high, it is squeezed out to provide lubrication.
- In the middle zone, the collagen fibres increase in their diameter and become less organized and more oblique in their arrangement. The middle zone has a high concentration of proteoglycans
- Collagen fibres in the deep zone are perpendicular to the tidemark. The highest concentration of proteoglycans are found in the deep zone. This is consistent with its main function which is to resist axial compression.
- Hydroxyapatite composes most of the calcified zone. This allows the cartilage to anchor itself into subchondral bone.

- **Draw a cross-section of articular cartilage? (Mention arcades of Benninghoff) (Figure 17.8)**

The three-dimensional structure of articular cartilage shows arcades of collagen that give rise to the appearance of the fibres in the three zones described above (tangential/horizontal; transitional/oblique; radial/perpendicular) all in relation to the joint surface and the tidemark.

- **Where does the nutrition of the cartilage come from?**

Nutrition of the articular cartilage comes from the synovial fluid that it bathes in. As the joint is loaded, the flux of water in and out of the cartilage allows the nutrients to diffuse through its matrix. This is supported by the viscoelastic properties of cartilage.

- **Talk me through what happens if you injure the articular cartilage with a scalpel while performing the arthroscopic portals?**

This type of acute trauma to articular cartilage is classified as either superficial or deep laceration, and this is in relation to whether the laceration is deep enough to cross the tidemark or not. If it is a superficial laceration, no adequate cellular response takes place macroscopically and, therefore, no cartilage repair occurs. This is due to the fact that the chondrocytes die and given that the cartilage is avascular no migration of chondrocytes occur and the defect remains. On the other hand if the laceration is deep and crosses the tidemark it penetrates the subchondral surface leading to fibrin clot formation and an inflammatory process which includes the release of growth factors and fibroblasts. This allows for fibrocartilage scar formation which is unorganized and has poor loadbearing properties. This is the theory behind abrasion chondroplasty.

Spontaneous osteonecrosis of the knee (SONK)

- Osteonecrosis of the knee with no identified cause
- More common among females who are middle-aged or elderly

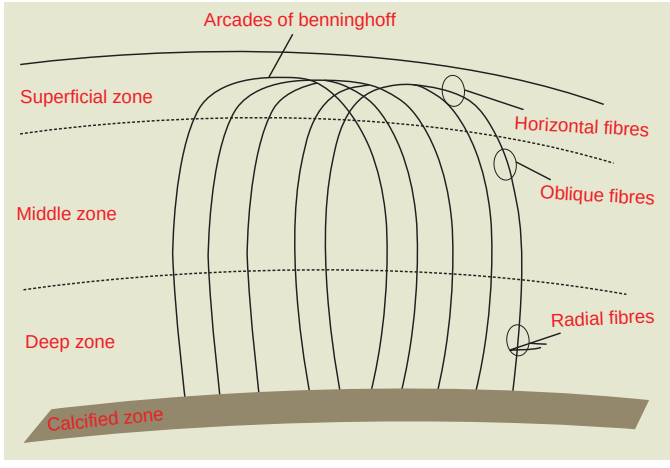


Figure 17.8 Schematic diagram of the cross-section of articular cartilage showing the Archades of Benninghoff

- Most commonly found in the distal aspect of the medial femoral condyle, and is almost always involving one joint
- Associated with meniscal root tear
- May represent a subchondral insufficiency/stress fracture

Clinical presentation

- Sudden onset of severe knee pain (usually non-specific). Can be focused over the medial femoral condyle
- Decreased range of motion with no mechanical block
- Effusion present in the acute stages
- Pain worse on activity

Diagnostic imaging

- Weight-bearing AP and lateral x-ray of knee (include hip and ankle if indicated)
- MRI can confirm the diagnosis and delineate the extent of the lesion
- Extensive bone marrow oedema seen on T2 images (Figure 17.9)
- Differential diagnosis include: OCD, transient osteoporosis, occult fracture

Treatment

- Non-operative: Most are treated successful in this manner
  - Activity modification
  - Rest and nonepartial weight-bearing
  - Analgesia including NSAIDs
  - Targeted physiotherapy focusing in range of motion and quadriceps strengthening
- Operative: Only considered after an extensive attempt of all non-operative measures as success is variable
  - Retrograde drilling
  - Arthroplasty (in larger lesions and bone collapse)
  - High tibial osteotomy (if mal-alignment present, a trial with an off-loader brace is recommended preoperatively)

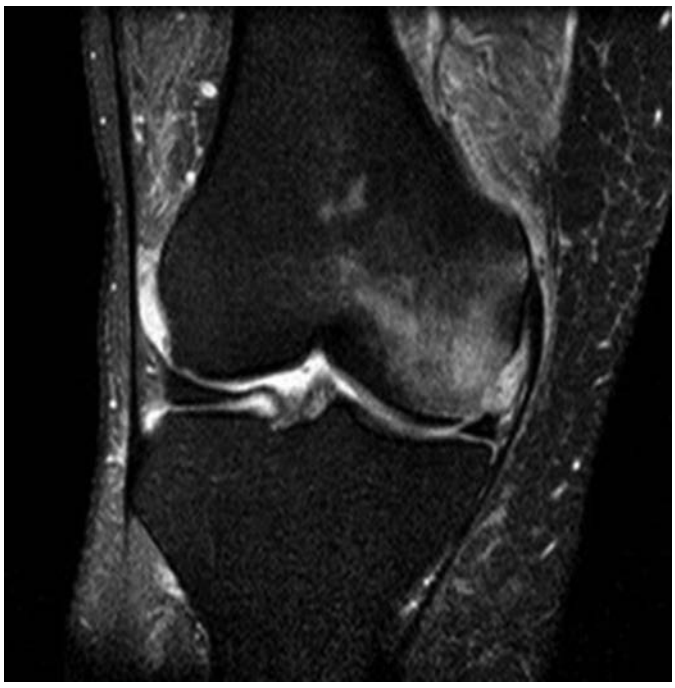


Figure 17.9 Spontaneous osteonecrosis of the medial femoral condyle revealing extensive bone marrow oedema within the condyle

Anterior cruciate ligament (ACL) injury

Anatomy

ACL is a primary resister to internal rotation of the tibia at <35° of flexion while the anterolateral ligament is a stabiliser of internal rotation >35° of flexion.

- Intra-articular ligament
- Originates just anterior to and between the tibial intercondylar eminences
- The ACL femoral attachment lies posteroinferior to the lateral intercondylar ridge ('resident's ridge') which runs at approximately a 30–35° angle with respect to the long axis of the femoral on the posteromedial aspect of the lateral femoral condyle
- It is 33 mm long and 11 mm in diameter
- The names of the two bundles of the ACL describe their tibial origins: The anteromedial bundle tightens in flexion (an anterior restraint); the posterolateral bundle tightens in extension (a rotary restraint)
- Supplied by the middle geniculate artery
- 90% type I and 10% type III collagen
- Prevents anterior translation and primary resister to internal rotation of the tibia at flexion angle <35° while the antrolateral ligament is a stabiliser of internal rotation in >35° of flexion[15]
- Has a proprioceptive role with the presence of mechanoreceptors within the ligament. ACL innervation come from the posterior articular nerve which is a branch off the tibial nerve
- Tensile strength of the native ACL is 2200 Newtons

Clinical features

- Mechanism of injury
  - low velocity, deceleration and pivotal injury, usually non-contact
  - valgus external rotation or hyperextension force in contact injury
  - high-energy RTA
- Audible or feeling of 'popping'
- Acute haemarthrosis in young within 1–2 hours; less dramatic in older patients
- Inability to continue playing sport

- Females more susceptible than males 4 : 1
- 20% of ACL injury is associated with MCL injury
- Meniscal damage occurs in 15–40% of acute ACL tears and becomes much more common with chronic ACL deficiency[16]
- There is an 80% incidence of lateral meniscal injury with combined ACL–MCL injury
- In chronic ACL deficiency, medial meniscal injury is more common
- Osteochondral lesions (bone bruising) are present in 80% of ACL ruptures.[17] This includes damage to the articular cartilage from the crush effect of the tibial plateau and the femoral condyles
- Chronic ACL deficiency causes posterior femoral subluxation and posterior tibiofemoral contact, leading to erosion of the posteromedial tibial plateau and subsequent fixed varus deformity. However, intact ACL has been associated with anteromedial arthritis pattern and correctable varus deformity (see also section on UKA)

Classic reference

Daniel DM, Stone ML, Dobson BE, Fithian DC, Rossman DJ, Kaufman KR. Fate of the ACL-injured patient: A prospective outcome study *Am J Sports Med*. 1994;22:632–644

Daniel et al. showed that ACL reconstruction does not always yield improved outcomes compared to the natural history and pointed out that patients who were able to 'cope' with ACL deficiency may have better outcomes is some respects than do patients who have reconstruction.

In ACL injured patients, sports participation reduced following rehabilitation both with surgical reconstruction and conservative treatment.

Joint arthrosis was more severe in patients undergoing surgical intervention. Pre-injury volume of sport participation, age, and amount of joint displacement measured by the KT-1000 arthrometer correlated with the need for late surgery.

This was one of the first papers that prospectively documented the outcome of conservative treatment in ACL injured patients and the factors that correlate with a greater risk of functional impairment and joint arthrosis. Daniel described a population of individuals who coped satisfactory with ACL deficiency over an extended period of time.

Classic reference

Noyes FR, Butler DL, Grood ES, Zernicke RF, Hefzy MS. Biomechanical analysis of human ligament grafts used in knee-ligament repairs and reconstructions *J Bone Joint Surg Am*. 1984;66:344–52.

This is a landmark article published by Noyes et al. on the structural and mechanical properties of both native ACL and the various tendon grafts used in reconstruction. The authors found that the ACL had a mean ultimate tensile strength of 1725 N and a stiffness of 182 N/mm.

Noyes also tested for the tensile strength and stiffness of patella, semitendinosus, gracilus and quadriceps tendon as well as fascia lata and iliotibial band grafts. A 14 mm bone patella tendon graft measured 168% of the tensile strength and almost 4 times the stiffness of a normal ACL. The semitendinosus and gracilis tendons were measured individually and were found to have 70% and 40% of the normal ACL strength. The patella graft was the only construct found to be stronger than the native ACL. These values represent the graft strength at implementation and do not account for the in vivo incorporation process.

The choice of harvesting site for grafts used in ACL reconstruction influences both the strength and stiffness of a graft. Bone-patellar tendon-bone grafts provide the greatest strength and stiffness compared to other sites. However, surgeons must consider that a range of factors will influence appropriate graft choice. Thigh stiffness could lead to limitations in a patient's ability to extend the knee.

Performing biomechanical testing on various ACL grafts and understanding graft strength and stiffness was an essential step in developing ACL surgical techniques and rehabilitation protocols. The results of this work are still quoted in the literature. The study, however, did not include quadrupled semitendinosus tendon.

Examination corner

- **Are ACL injuries more common in female or male athletes?**

ACL rupture are most common in female athletes in comparison to male athletes playing the same support. It is increased by a ratio of 4–5 : 1 in comparison with male athletes.

- **Why is that? What is the suggested explanation?**

This is multifactorial, and includes intrinsic factors such as increased valgus mal-alignment, a smaller intercondylar notch, reduced ligament diameter. There has been theories in regards to the hormone levels and many studies have looked into the relationship between menstruation and ACL rupture with no conclusive evidence. Landing biomechanics where females more commonly land with more extension and valgus plays a large part in the increased rate of rupture. Neuromuscular conditioning has been shown to reduce this rate[18].

Examination corner

How do you do the Lachman's test?

- Check PCL sag and medial tibial step-off before the test
- Maintain the knee in neutral rotation during the test.
- The most sensitive test for detection of an ACL tear. Knee is placed in 20–30° of flexion. The femur is stabilised with the non-dominant hand. An anteriorly placed forced is applied to the proximal tibia with the dominant hand. The amount of transaltion of the tibia on the femur, and the firmness of the 'endpoint' should be compared to the contralateral knee

How do you do the pivot shift for a non-functioning ACL?

- Place a valgus stress, axial load and internal rotation on the tibia as the knee is slowly flexed. In full extension, gravity pulls the femur posteriorly, resulting in anterior subluxation of the tibia. With further flexion, posterior pull by the iliotibial tract reduces the tibia at about 20–30° (shift).
- Partial ACL tear and lax MCL lead to positive Lachman's and negative pivot. Complete tear leads to positive Lachman's and pivot. The knee cannot be pivoted if there is complete disruption of the iliotibial tract or MCL giving a false negative pivot shift test (see pivot shift analysis in Chapter 9).

Differential diagnosis of acute haemarthrosis

- ACL rupture
- Intra-articular fracture
- Patella dislocation
- Capsular tear
- Peripheral meniscal tear
- Beware of patient on warfarin with haemarthrosis secondary to minor trauma

Imaging

- AP x-ray of the knee may reveal a Segond fracture (now thought to possibly be an avulsion of the anterolateral ligament – ALL) – Pathognomonic for an ACL injury. The ALL passes anterodistally from an attachment proximal and posterior to the lateral femoral epicondyle to the margin of the lateral tibial plateau, approximately midway between Gerdy's tubercle and the head of the fibula (Figure 17.10)[19]

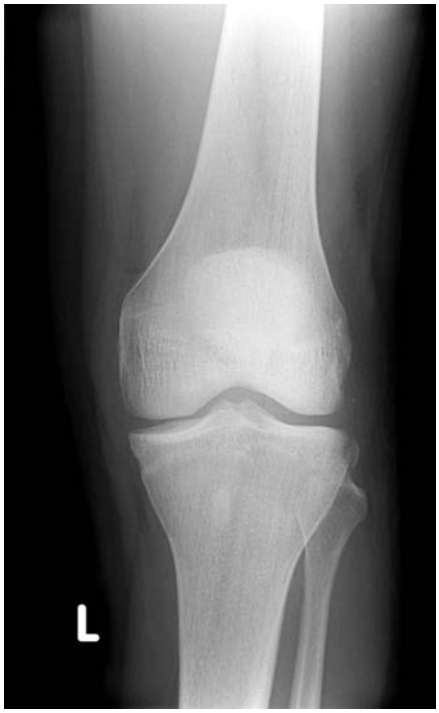


Figure 17.10 A Segond fracture seen occasionally on plain AP radiographs of the knee in ACL ruptures

- Lateral x-ray of the knee may reveal and haemarthrosis or lipo-haemarthrosis. It may also reveal tibial subluxation on the femur
- Sagittal MRI (T2) of the knee usually demonstrates disruption of the oblique ACL fibres, and bone bruising (bone marrow oedema with increased signal) of the middle third of the lateral femoral condyle and posterior third of the lateral tibial plateau (Figure 17.11)[17]
- Coronal MRI (T2) images show an empty lateral wall of the intercondylar notch

Outcome of injury

McDaniel – Rule of thirds

Patients with ACL-deficient knee

- One-third are able to compensate and can pursue normal recreational sports
- One-third are able to compensate but will have to reduce their sporting activities
- One-third do poorly and develop instability with simple activities of daily living
- However, a few are able to compensate and pursue normal recreational sports and most patients try to keep their activities within 'the envelope of stability' to avoid recurrent giveaways[21,22]

Management

Management should be individualized based on age, activity level, laxity, instability, associated injuries and other factors.

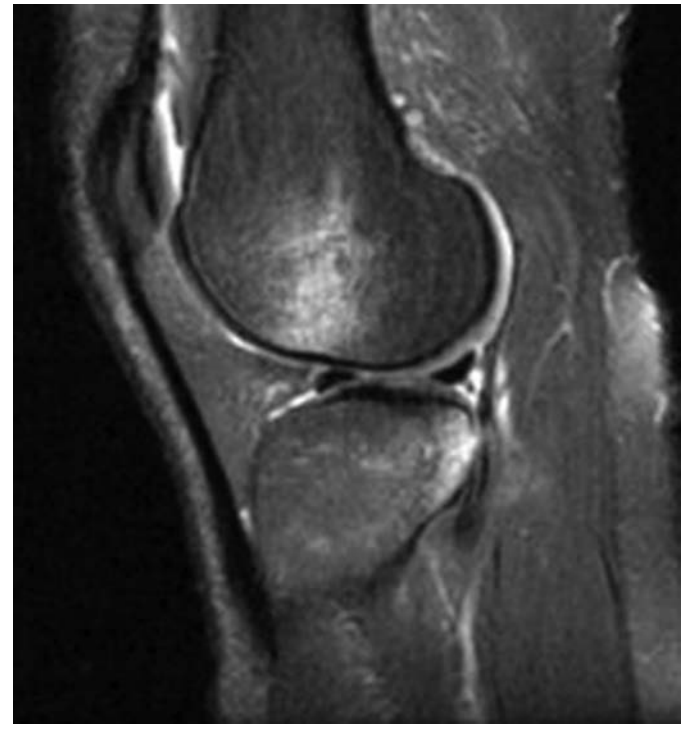


Figure 17.11 Sagittal MRI T2-weighted demonstrating the bone bruising in an ACL injury (ligament rupture not visible on this sequence)

Non-operative

- Commonly requires restriction or modification of activity level
- Extensive physiotherapy to rehabilitate and strengthen the quadriceps and hamstrings
- Proprioception training
- ACL brace
- Associated with high incidence of instability in younger patients
- Potentially may lead to meniscal tear, articular injury and subsequent degenerative changes
- Fallen out of favour as advances in surgical technique and physical therapy have reduced operative morbidity and improved outcome

Surgical

1. Primary repair of bony avulsion lesion in young patients
2. Primary repair ± augmentation. Obsolete
3. Extra-articular reconstruction (MacIntosh, Ellison)
   - Involves tenodesis of the iliotibial tract
   - Pass a mobilized strip made up of the posterior third of the iliotibial band to the PLC of the knee through a tunnel deep to the LCL
   - Reduces or eliminates pivot shift but there is concern regarding its effectiveness in addressing anterior translation
   - Has been used recently to augment intra-articular reconstruction especially in patients with severe ligamentous laxity or failed primary surgery
4. Intra-articular reconstruction: This is current best practice
   - Arthroscopic ACL reconstruction (open reconstruction is now in decline)

Graft types

- **Autografts**: bone–patella–tendon–bone (BPTB), hamstring (semitendonosis and gracillis), quadriceps. Ipsilateral hamstring autografts are the most commonly used graft in the UK
- **Allograft**: BPTB, achilles tendon, hamstring, tibialis anterior
- **Synthetic**: LARS®, Gore-Tex®, Dacron® or polyester
- Xenograft

BPTB autograft

Advantages:

- Patient's own tissue
- Easy to harvest
- Bone-to-bone healing
- Direct rigid fixation
- Faster biological integration in 6 weeks

Disadvantages:

- Donor site morbidity from graft harvest
- Anterior knee pain 30–50%
- Patellar tendonitis 3–5%
- Fracture patella, rare
- Patella tendon rupture, rare
- Patella baja (shortening of patellar tendon – Seen when the remaining tendon after harvest is sutured together)
- Development of late OA

BPTB allograft

- Slower incorporation
- Biologically inactive
- Less stability at 6 months
- Risk of disease transmission
- Essential role in revision surgery
- Weaker after having been irradiated and not biologically active
- Radiation affect the structural and mechanical properties of the graft

Hamstring graft autograft

This graft is usually quadrupled.

Advantages:

- Patient's own tissue
- Small incision
- Large cross-sectional area of tendon
- Relatively easy passage of graft
- Less donor site morbidity

Disadvantages:

- Slow tendon-to-bone healing in the tunnel in 8–12 weeks
- No bone graft in the tunnels – 'windshield wiper' effect (can occur with the use of suspensory fixation which can lead to tunnel abrasion and expansion when the knee is in motion)
- Deep flexion weakness after surgery
- Possibility of injury to saphenous nerve (poor technique)

Synthetic grafts (LARS/Gore-Tex®/Dacron®)

- Higher failure rate
- Expensive
- Osteolysis
- Risk of infection
- Atraumatic effusions
- No disease transmission
- No harvest site morbidity

Quadriceps graft

Thick tendon but short, with good biomechanical properties. The graft is taken with a patella bone plug. It is associated with decreased anterior knee pain. However, graft harvest weakens the quadriceps and can be technically difficult. In most centres,

it is rarely used as a primary graft but more commonly used in revision surgery.

Graft fixation

- Aperture fixation: Interference screw at the level of the joint (metal or bioabsorbable). Note that these screws interfere with the 360° circumferential healing of the graft in the tunnel
- Suspensory fixation

  Cortical: EndoButton®, tightrope, screw posts, staples
  Cancellous: transfixion pin/cross-pin
- WasherLoc™ (tibia)
- None (Press-fit)

Ideal fixation is strong enough to avoid failure, stiff enough to restore knee stability by resisting displacement under load, and secure enough to avoid slippage of the graft from its initial position. It is preferable for the fixation to be biocompatible, MRI safe and allow for easy revision.

Examination corner

Adult and pathology 1 oral

The examiner shows a radiograph of Segond fracture of the knee and asks (Figure 17.10):

- **Describe the radiograph**

This is an AP radiograph of a knee which appears to be non-weight-bearing. The most obvious abnormality is an avulsion fracture from the lateral aspect of the tibial plateau. This represents a Segond fracture.

- **What does the fracture suggest?**

A Segond fracture is pathognomonic for an ACL injury. It was previously thought to be an avulsion off the lateral capsule of the knee but recent studies have indicated that this is actually a rupture of the anterolateral ligament (ALL) from its tibial insertion[19].

- **How many bundles does the ACL have?**

The ACL is made up of two bundles. Then anteromedial (AM) and the posterolateral (PL) bundles. The name of bundle describes its femoral origin.

- **When does the anteromedial bundle get tight?**

The anteromedial bundle is tight in flexion. It is primarily an anterior restraint and is tested using anterior drawer or the Lachman's tests. While the posterolateral bundle is tight in extension, and its primary role is a rotary restraint. This bundle is evaluated with a pivot shift test.

- **Is double bundle reconstruction clinically superior to single bundle?**

A recent prospective randomised controlled trial revealed that ACL reconstruction using the transtibial approach, double bundle reconstruction was significantly better than single bundle reconstruction in relation to anterior and rotational stability during a mid to long-term follow-up (3–12 years). However, there were no differences in the subjective findings[22]. A 2013 Cochrane review concluded that there was insufficient evidence to determine the efficacy of double-bundle vs single-bundle ACL reconstruction in adults. There was limited evidence in the 17 trials included, that double-bundle reconstruction had some superior results in objective measurements of knee stability and increased protection in preventing re-rupture and further meniscal injury[23]. Double bundle reconstruction is technically more demanding, thus, leading to a higher risk of complication with tunnel placement.

- **What is the ideal ACL tunnel placement in the femur and the tibia?**

This is controversial and no consensus is present in regards to the gold standard for femoral tunnel placement. Again the advocates of the so called anatomic, more anterior, femoral tunnel placement have now reverting back to the posterior placement of femoral tunnels. The femoral tunnel should not be placed too anterior (as that would limit flexion and extension) or too posterior (as that would make the knee lax in both flexion and extension). The projection of the femoral tunnel can also lead to problems: If the tunnel is too vertical you risk rotatory instability or even blow out of the posterior femoral cortex. Tibial tunnel placement is less controversial, and should be placed midway between the anterior horn of the lateral meniscus and anterior aspect of the medial tibial spine. If tibial tunnel is placed too anterior there is a risk of notch impingement which leads to a fixed flexion deformity.

- **Which graft would you use? And why?**

I would use autologous ipsilateral hamstrings graft using gracilis and semitendinosus. I would use this as this is the graft that I am most familiar with harvesting and using. The results are consistent and the graft itself has good tensile strength and has low donor site morbidity. The incision is small and cosmetically acceptable, and the risk of anterior knee pain, patella tendonitis and patella fracture that you can get with patella tendon (BTB) grafts is avoided. I would not use any of the synthetic grafts until there are more robust research and data in regards to their long-term risks including the possibility of osteolysis which occurred in previous generations of synthetic grafts.

- **How would your management differ with an ipsilateral MCL sprain?**

With an MCL injury, I would delay the ACL reconstruction to allow the MCL to heal. I would place the patient in a hinge brace until that date especially if the patient has signs and symptoms of instability.

- **If your MRI revels a PLC injury, what would you reconstruct first the ACL or PLC?**

A PLC injury is commonly a missed injury in ACL reconstruction failure. If it is a grade III PLC injury then this will require reconstruction. This should occur either at the same time as the ACL reconstruction or performed as a first stage of a two-stage procedure.

Principles of ACL reconstruction

- Graft: Best to use a biologically active graft
- Tunnels: 'anatomically' and isometrically placed tunnels. Exact tunnel position is controversial with no consensus yet in the orthopaedic literature or the knee community
- Fixation: The graft should be adequately tensioned
- Rehabilitation should respect fixation choice

Surgical technique

- Femoral tunnel placement: the optimum tunnel position in anatomic single bundle ACL reconstruction remains controversial: Recent evidence suggests that placing the femoral tunnel through the anatomic centre of the femoral origin of ACL may further improve the rotatory stability compared to antromedial bundle femoral tunnel position[24]
- Tibial tunnel: the tibial tunnel aperture should be anterior to the PCL and within the footprint of the ACL. It is usually between the medial tibial spine and the anterior horn of the lateral meniscus. The trajectory of the tunnel should be less the 75°
- Care should be taken in calculating the length of the tibial and femoral tunnels taking into account the length of the graft available as well as the method of graft fixation
- Notchplasty is usually unnecessary if the graft is correctly placed. Remove osteophytes if present as they may cause impingement of graft
- To tension the graft appropriately, it is common practice to apply 40 N or 10 lb of tension on the graft while it is secured in 20–30° of flexion

Complications (Table 17.3)

- Tunnel placement technical errors (please see Figure 17.11 and ACL Examination corner)
  - Anterior placement of the femoral tunnel limits flexion
  - Anterior placement of the tibial tunnel limits extension
- Tunnel widening – Secondary to graft motion within the tunnel (both biological and mechanical factors) and found more with non-aperture fixation methods. More than 3 mm of motion interferes with graft incorporation within the tunnel
- Three types of graft motion
  1. Bungee cord effect – Longitudinal motion
  2. Wind-wiper effect – Horizontal motion
  3. Creep of the graft – This leads to tissue elongation
- Graft rupture from notch impingement
- Graft failure from mal-aligned limb (coronal and sagittal mal-alignment need to be corrected either prior or during ACL reconstruction commonly with a high tibial osteotomy)
- Flexion contracture and arthrofibrosis
- Failure of fixation – Fixation is the weakest link in the early postoperative period

Table 17.3 The effect of tunnel malpostioning on graft strain and knee range of motion. Malpostioning the femoral tunnel is less forgiving as it lies closer to the knee center of rotation. The effect of femoral tunnel malpostioning on graft strain is also dependent on the knee flexion angle at the time of graft tensioning

Femoral tunnel placement	Graft tensioning angle	
	Tensioning the graft in extension	Tensioning the graft in flexion
Anterior	The knee is tight in flexion	The knee is lax in extension
Posterior	Lax in flexion	Tight in full extension
Tibial tunnel placement	**Extension**	**Flexion**
Anterior	Notch impingement	Tight knee (graft strain)
Posterior	Tight knee (graft strain)	Loose knee (lax graft)

- Cyclops lesion from the residual tissue anterior to the ACL which blocks extension. Some surgeons prefer tunnelling the graft into the native ACL stump at its tibial attachment
- Infection
- DVT and PE
- Secondary osteoarthritis

Considerations for ACL injuries in the paediatric population

- Techniques are intended to avoid violating or minimizing injury to the physis which could lead to growth disturbance. This is more relevant in an open physis and patients under 14
- Reconstruction could be performed in a physeal sparing manner or transphyseal technique. No significant difference in growth disturbance has been found in either technique[25]
- Certain considerations need to be taken during reconstruction to minimize the risk of physeal injury. This includes limiting the tunnel diameter to <8 mm (which means graft diameter not >8 mm), drill tunnels at a lower speed, avoid oblique tunnels (i.e. more vertical tunnel placement need to be considered) and avoid interference screw fixation within the tunnels
- All inside technique using Arthrex Flipcutter is an alternative way

KT 2000 Arthrometer

- This instrument is used to quantify anteroposterior knee displacement. It measures AP translation of the tibia in relation to the femur
- Manual maximum anterior displacement of 30 lb can be used,
- Side-to-side difference of >3 mm is significant[20,21]

Examination corner

Adult orthopaedics and pathology oral 1

Shown sagittal MRI of Ruptured ACL (Figure 17.11)

- **What are the indications for ACL reconstruction?**

The indications for ACL reconstruction are patients that present with symptoms and signs of instability following an ACL rupture. Reconstruction is more pertinent for the younger and active patient as that reduces the incidence of further injury to the knee leading to the possibility of early OA. Meniscal and chondral injuries are not uncommon in an ACL deficient knee. Children with ACL ruptures should also be strongly considered for reconstruction to avoid further knee injuries as their compliance to activity limitation and modification will be poor. High demand middle-ages patients who are symptomatic also do warrant reconstruction (age is not a contraindication unless osteoarthritis is present).

- **What are the principles of ACL reconstruction?**

The principles are to reconstruct tibial and femoral bone tunnels in an anatomic and isometric manner, with the use of a biologically active graft that is adequately tensioned and fixed to allow early rehabilitation and provide stability.

- **Tell me what are the types of grafts and which would you use?.**

Grafts can be autologous, allogenic, synthetic or xenografts. Autografts can be: 1. Hamstrings 2. Patella tendon (BTB) 3. Quadriceps tendon. Allografts include hamstrings, patella tendon, quadriceps tendon as well as Achilles tendon. There are various synthetic grafts on the market which previously failed but new generation grafts have shown promising results (they are mostly being used for extra-articular ligament reconstructions such as MCL, LCL, MPFL). Xenografts are undergoing clinical trials and have potential in the future but are not currently used in clinical practice.

I would use a hamstrings autograft from the ipsilateral leg as I am familiar with this procedure. This graft type has good and reliable long-term results. It also involves a relatively small incision with low donor site morbidity. It also offers a relatively easy passage of the graft in the tunnel.

- **If a patient asks you about other options of grafts and wants more information about allografts, what would you tell them about their advantages and disadvantages?**

Allografts have their advantage; no risks, pain, or scars from the harvest site. Operation time is less as no harvesting is involved and patient has less postoperative discomfort with lower incidence of joint stiffness and quadriceps wasting. They are also useful in multi-ligament reconstruction when there is a need for several grafts. Their disadvantages include the risk of infection from the cadaveric tissue. The dilemma occurs in ascertaining the balance of sterility and radiation vs the alteration of the collagen tissue within the graft reducing its tensile strength.

Unlike organ transplantation, allografts aren't usually at risk for tissue rejection by the host. This is due to the minimal presence of protein antigen in these washed grafts (the bone ends are completely cleansed of any marrow elements).

A number of studies have found measurable but not statistically or clinically significant in the longevity of allografts. A study by Noyes et al.[26] looked at 4 and 7 year follow-up of 70 allograft patients and found no significant deterioration of the allografts over the measured time period.

A further disadvantage in allografts is cost which can be anything between 1000 and 5000. Given the current economic climate and the state of the NHS this would be difficult to justify.

Tibial eminence fracture

These are most commonly seen in skeletally immature children and adolescents aged 8–14 years. The tibial eminence is the non-articular portion of the tibia between the tibial plateaus and is in close proximity to the anterior cruciate ligament insertion. The mechanism of injury is similar to ACL injury in adults. This fracture frequently occurs as a result of a fall off a bicycle.

Meyers and McKeever classification (1959)

Type I: Non-displaced

Type II: Partially displaced or hinged

Type III: Completely displaced

Type IIIA (Zifko) involves the ACL insertion only

Type IIIB (Zifko) includes the entire intercondylar eminence

Type IV (Zaricznyj 1977): Comminution of the fracture fragment

Treatment

- Casting in extension for type I
- Open reduction and internal fixation
- Arthroscopic reduction and fixation
- Rarely, ACL reconstruction is necessary (ACL laxity is found in 10% of surgically treated and 20% of non-surgically treated injuries)

Type I fractures can almost always be treated by closed means, whereas types II, III and IV fractures frequently require surgical intervention. Arthroscopic reduction and fixation has become popular because of its lower morbidity. The fracture fragment can be fixed by using screws, bent K-wires or sutures. Beware of ACL laxity secondary to stretching in Type I injuries.

Examination corner

Trauma oral 1

The examiner shows a lateral x-ray of a type II fracture of the tibial eminence in a skeletally immature knee (Figure 17.12 a and b) and asks:

(a) (b)

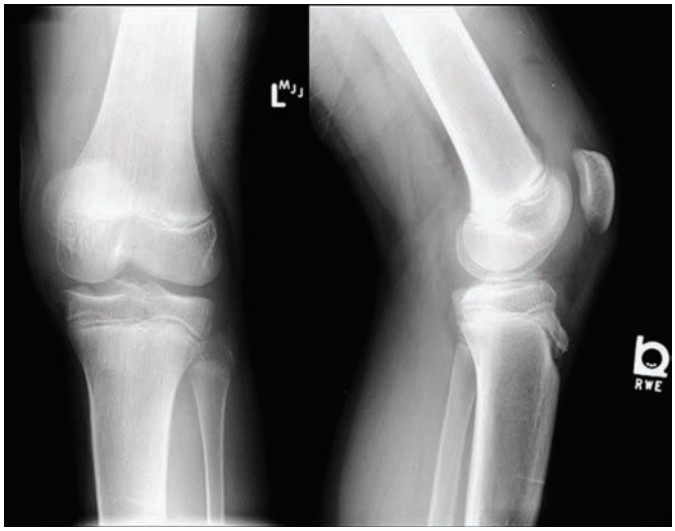


Figure 17.12 (a, b) AP and lateral knee radiograph revealing a tibial eminence injury

- **Describe what you see?**

AP and lateral radiographs of a skeletally immature knee, probably over the age of 10, with a tibial eminence fracture. The fracture has a hinge to it and is displaced slightly.

- **How do you classify this fracture?**

According to the Meyer Mckeever classification and this would be a type II. Type I is an undisplaced fracture and the a type III is completely displaced. Type IV which was an addition and not in the original description is a comminuted fracture.

- **What other radiological investigations would you ask for?**

I would request an MRI scan f this child's knee to delineate any associated intra-articular injury to the soft tissues of the knee. A CT scan would aid preoperative planning but given we are dealing with a child an MRI scan would be more appropriate.

- **What other structures could be damaged in the knee?**

These fractures especially the types III and IV are associated with 30–40% risk of injuries to the menisci, chondral cartilage and collateral ligaments within the knee.

- **How do you manage this type of fracture?**

After having taken a full history from the patient and any witnesses of the injury, assessed the patient and obtained all appropriate investigations and I would proceed into counselling the patient and his family in regards to open reduction internal fixation of this fracture.

- **What are the options of fixation and which fixation method is stronger and has less risk of causing growth disturbance?**

This fracture can be fixed with a screw, K-wires or sutures. This can be done open or arthroscopic-assisted. The older the child is the more likely a screw fixation would be performed. Screw fixation is stronger and more reliable. There is debate in regards to the degree of screw projection (whether to should remain within the epiphysis or cross into the metaphysis). If it does cross into the metaphysis then the risk damage to the physis and possibility of growth disturbance. Screw fixation is technically less demanding and allows earlier mobilization. Suture fixation is technically more demanding but does avoid phsyeal injury. It is important to ensure that the meniscus is disengaged from the fracture at the time of fixation.

Posterior cruciate ligament (PCL) injury

Anatomy

- This is the strongest ligament in the knee
- It is regarded as 'a central stabiliser'. It is the primary restraint to posterior tibial translation
- Posteromedial bundle (PM): tight in extension
- Antrolateral (AL): long and thick part, twice the size of PM bundle tightens in flexion
- Originates from a broad crescent-shaped area in the posterolateral medial femoral condyle
- It inserts centrally posteriorly 1.0–1.5 cm below the articular surface of the tibia
- It has an average length of 38 mm and a diameter of 13 mm
- PCL and quadriceps are dynamic partners in stabilising the knee in the sagittal plane.
- There are three components

  Anterolateral: Long and thick part, twice the size of the posteromedial bundle; tightens in flexion
  Posteromedial: Tight in extension
  Meniscofemoral ligaments: Mechanically very strong

  Anterior: Humphrey's ligament
  Posterior: Wrisberg's ligament

- Vascular supply from the middle geniculate artery

Mechanism of injury

- 3% of all knee injuries
- Direct injury against the proximal tibia when the knee is flexed 90° is the most common (dashboard injury)
- Falling on a flexed knee with foot in plantar flexion
- Forced hyperextension (>30°) is associated with multi-ligament injury. Most instability is experienced with the knee in 90° of flexion
- High association with periarticular fractures around the knee. It is recommended that the PCL is examined after fracture fixation as there is 7.8% incidence of PCL injuries[27]. This is also a 2–5% rate of PCL injury with femoral shaft fractures[28]

- Also associated with posterolateral corner (PLC) injury and knee dislocations

Diagnosis

- Injury is often missed in the acute knee
- Clinical examination is more reliable than MRI scan
- The PCL may be dysfunctional despite normal MRI
- MRI scan is a confirmatory study for a PCL injury in acute injury (Figure 17.13 a and b). However, it is only 50% of the time diagnostic in chronic cases; therefore, it should be used with caution
- Lateral stress view radiographs reveal increased posterior sag on posterior drawer in comparison with the contralateral knee (becoming gold standard)
- Kneeling stress x-ray shows the degree of posterior translation

Clinical examination

- Tibial step-off sign/posterior sag sign (medial tibial plateau is anterior to the femoral condyle at 90° flexion in a normal knee)
- Posterior drawer test at 90°
- Quadriceps active drawer test. Flex the knee to 60° and control the foot by applying downward force onto bed, then ask the patient to contract the quads. The test is positive when the tibia reduces
- Posterolateral rotatory instability (dial test prone – Requires two people to perform test accurately):
  Performed at 30° and 90°. Considered positive if there is a difference >10° of external rotation of the foot. If positive at 30° but not at 90° then it is an isolated PLC injury. If positive at both 30° and 90° then this indicates a PCL and PLC injury
- External rotation recurvatum test

Grading of PCL instability

- Normal tibial step-off is 10 mm at 90° flexion
- Instability could be mild, moderate or severe
- Grade I laxity is when there is a 5-mm step-off
- Grade II laxity is when there is no step-off (flush)
- Grade III laxity is when there is –5-mm step-off
- There is a high association between grade III PCL injury and PLC injury. This highlights the importance of the dial test

Management

In isolation, PCL injury often causes little long-term instability. However, it may lead to medial or PF joint pain at a later date. It is more troublesome in soccer players owing to difficulty in deceleration.

Acute isolated PCL injury is commonly missed as it may present with very little pain in the knee without haemarthrosis. There may be only bruising at the popliteal fossa. Chronic PCL injury on the other hand may present with pain in the medial compartment or anterior knee pain.

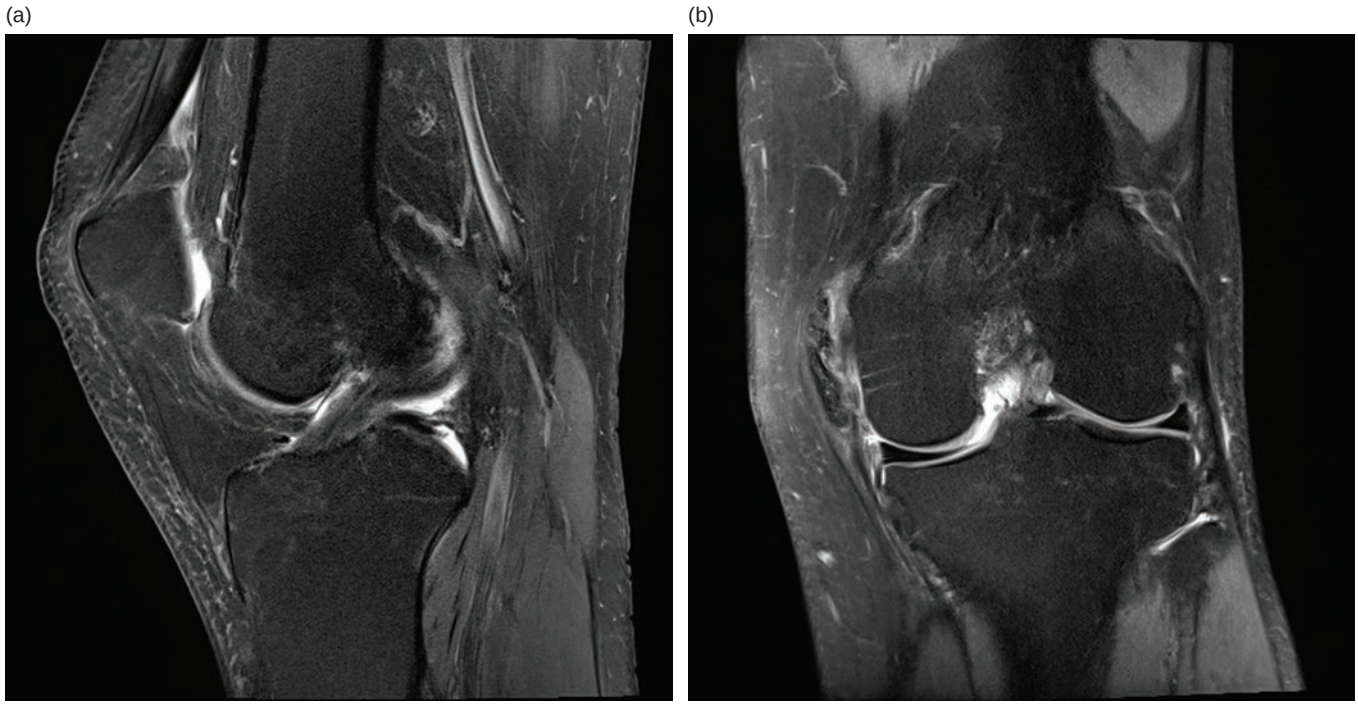


Figure 17.13 (a, b) Sagittal and coronal T2 MRI scan revealing a ruptured PCL

It is acceptable to treat an acute, isolated PCL injury conservatively. The knee is kept in extension in a brace with calf support (posterior tibial support, PTS brace) until the pain subsides (4–6 weeks) with quadriceps rehabilitation. Start early passive motion only in the prone position to maintain anterior tibia translation.

Outcome is poor after meniscectomy, or with patellar chondrosis, gross laxity and weak quadriceps. If associated with posterolateral or posteromedial injuries, knee stability is dramatically reduced.

Surgical reconstruction

PCL (open/arthroscopic) reconstruction is recommended:

- Acute combined ligamentous injuries
- Acute isolated injury with bony avulsion
- Symptomatic chronic PCL injuries that failed rehabilitation

Arthroscopic reconstruction, although technically demanding, is safe and commonly performed nowadays. Single bundle and double bundle PCL reconstruction can be performed. Double-bundle reconstruction is technically more demanding. Although both techniques resulted in similar patient satisfaction in a level II RCT as measured by outcome assessment, the double bundle procedure significantly improved knee stability[29].

Complications

Immediate

- Vascular injury to popliteal vessels: Posterior to PCL insertion on tibia (close to tunnels) with only the posterior capsule separating it
- Infection
- Technical error → imprecise tunnel placement, graft tensioning, insecure fixation

Delayed

- Loss of motion
- Avascular necrosis (medial femoral condyle)
- Recurrent or persistent laxity (common) when a combined injury is not adequately addressed

Outcome (Figure 17.14 a and b)

- Good clinical outcome seen in acute primary PCL repair with bony avulsions
- Mid-substance ligament repair are not advised as they are typically not successful
- PCL reconstructions are less successful than ACL reconstructions
- Key is to identify and address all other concomitant ligament injuries. Surgical technique is upon surgeon's preference. Surgical reconstruction can be performed using an arthroscopic transtibial technique of an open tibial inlay technique. This can be either a single or double-bundle reconstruction

Knee dislocation

ACL, PCL, MCL, LCL and PLC are the main stabilisers of the knee. Any triple-ligament knee injury constitutes a frank dislocation. This is relatively rare but is a severe and potentially limb-threatening injury.

There is 3.3–18.0%[30,31] incidence of vascular compromise and a 20–30% incidence of nerve injury. The incidence of any fracture may be as high as 60%. It usually happens as a result of a high-energy injury such as an RTA. It may occur following lesser injuries, such as sporting accidents. It may be missed on initial assessment.

Recent CORR papers in 2014 revealed lower incidence of vascular injury compared to the previously quoted figure of 50%. The largest study of knee dislocations to date (including 8050 dislocations) from the USA revealed an overall rate of concomitant vascular injury of 3.3–13.0% requiring surgical intervention.[30] Furthermore, a systematic review[31] revealed an overall frequency of 18% for vascular injury – With 80% requiring surgical intervention – And 25% for nerve injury following knee dislocation. Knee dislocations with injury to the ACL, PCL and MCL (Schenck and Kennedy KDIIIL) had the highest rate of vascular injury (32%) followed by posterior dislocations (25%).

Classification

Knee dislocation is classified on the basis of direction of tibial displacement (displacement of the tibia with respect to the femur):

- Anterior (most common: 30–50% of dislocations, and associated with intimal tears) and posterior; also medial, lateral (highest rate of peroneal nerve injury) and rotatory (usually irreducible) or combined
- Alternatively the Schenck classification is based on the pattern of ligament injury of knee dislocation (KD)

Key factors also include whether it is:

- Closed or open
- High or low energy
- Dislocation or subluxation
- Neurovascular involvement

Mechanism of injury

Hyperextension leads to anterior dislocation. Dashboard injury leads to posterior dislocation.

Examination

The knee must be examined carefully, looking for:

- Valgus and varus laxity
- Anteroposterior translation

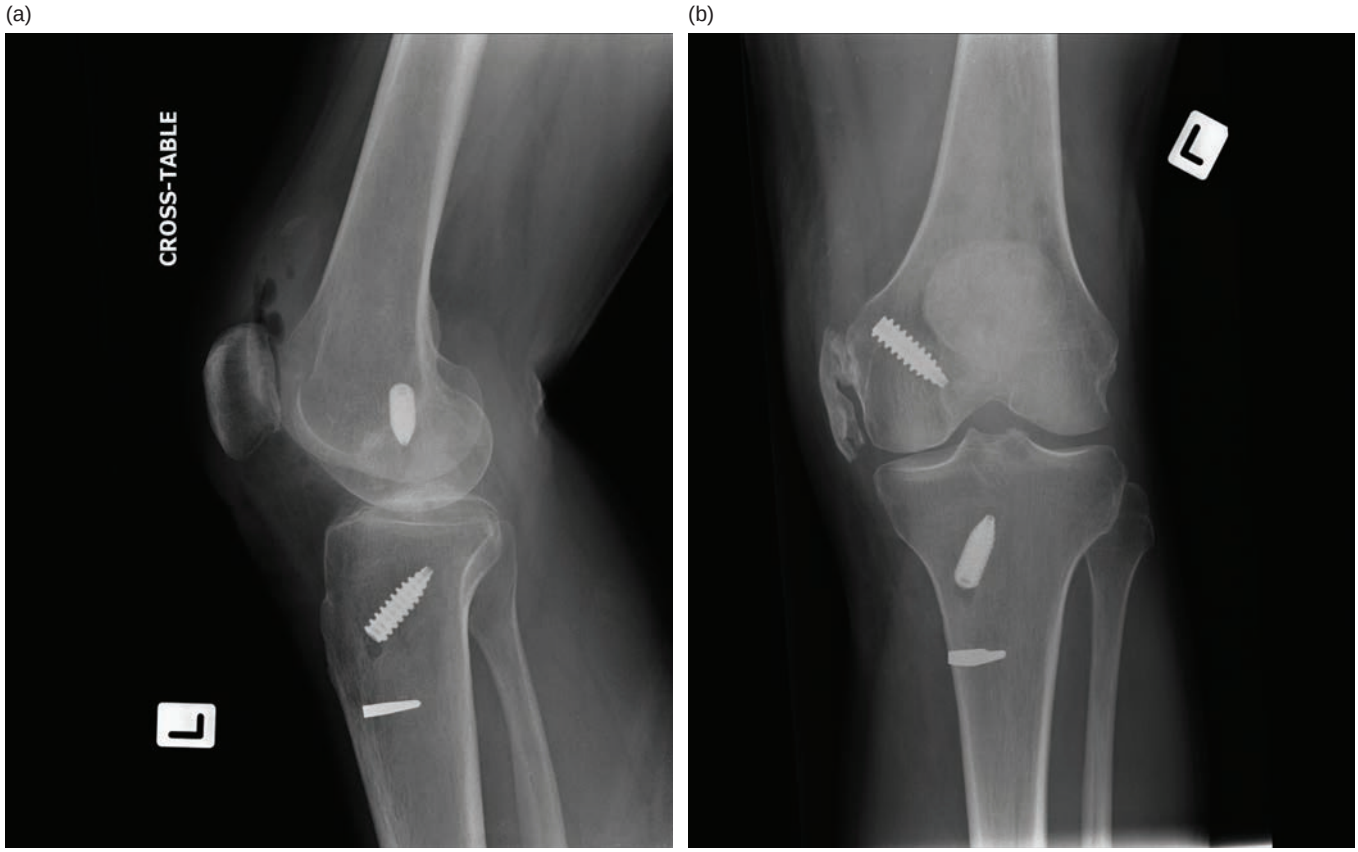


Figure 17.14 (a, b) Postop PCL reconstruction. Old MCL injury (Pellegrini–Stieda)

- Recurvatum
  - >10° hyperextension suggests ACL injury
  - >30° hyperextension indicates PCL injury
- Rotation indicates MCL and LCL injury

  Pulses need to be palpated: Popliteal, posterior tibial and dorsalis paedis

  Both sensory and motor nerves to the lower limb need to be assessed

  Clear and accurate documentation is paramount

Management

- Surgical emergency
- Deal with life-threatening injuries first
- Assess circulation by physical examination and/or Doppler in the emergency department
- Serial physical examination for at least 48 hours is sufficient to detect most of the clinically significant vascular injuries
- Ankle : Brachial Pressure Index (ABPI) can be used to assess the need for arteriogaphy/CT angiography. Arterial intimal tears are more common and easily missed, and pulses are commonly intact
- ABPI <0.9 is suggestive of significant arterial injury
- Involve the vascular surgeon with a view to arteriography/CT angiogram
- Radiography before manipulation (assess direction and associated fracture)
- Reduction as soon as possible in the emergency/operating room
- Check for the dimple sign medially, indicating posterolateral dislocation and medial condyle buttonholing, which preclude close manipulation
- Increased external rotation suggests popliteofibular ligament and popliteus tendon injury while increased external rotation and varus laxity suggest LCL injury as well
- Gross varus laxity and external rotation indicate extensive damage to the PLC, including the articular capsule
- Immobilization in an extension knee splint
- Check radiograph to confirm congruity; if not, consider external fixator
- Conservative management is going out of favour as it leads to gross instability
- Early surgical reconstruction and/or repair is currently recommended by the Knee Dislocation Study Group
- Timing of intervention is critical
- During the first week there is a likelihood of late vascular compromise

- Surgical dissection after 3 weeks becomes very difficult
- Ligament surgery is best performed as soon as the vascular surgeon allows
- Early motion is allowed to prevent arthrofibrosis if the integrity of the ligament and vascular reconstruction permit
- Most ACL/PCL/MCL can be treated with bracing the MCL followed by combined ACL/PCL reconstruction once range of movement is regained, usually after 6 weeks
- Alternatively, repair the capsule and repair or augment the MCL early and reconstruct the ACL 6–8 weeks later
- ACL/PCL/PLC can be treated by repairing or reconstructing the PLC acutely (within 3 weeks) and delayed ACL/PCL reconstruction 8 weeks later. PLC repair/reconstruction should be performed either prior to or in the same sitting (single-stage vs two-stage) reconstruction of ACL or PCL, otherwise the graft is likely to fail
- Open dislocation, fracture dislocation and vascular compromise require staged procedures

Indications for applying an external fixator

- If the tibiofemoral joint is incongruent after reduction
- Vascular injury (plus fasciotomy)
- Massive soft-tissue injury

Method of Ex-Fix application in a dislocated knee with an arterial injury:

- Position the patient supine for the ease of initial pin placement (lateral femoral pins and anteromedial tibial pins)
- Turn the patient prone for the popliteal shunt by the vascular surgeons (more recently many vascular and trauma surgeons have promoted vascular exploration and repair from a posteromedial incision which can be an extension of the fasciotomy incision which allows good visualisation and access to the popliteal artery as well as the femoral artery more proximally)
- Apply bars to the pins to achieve a solid construct with the knee joint reduced and the limb perfused (patient supine)
- Vascular anastomosis can be performed safely with the patient back in prone position
- Concurrent fasciotomies should be considered in these situations to avoid compartment syndrome secondary to reperfusion injury

Posterolateral corner (PLC) of the knee

The main stabilisers of the lateral and posterolateral aspect of the knee are popliteus, LCL, popliteofibular ligament, biceps femoris, iliotibial band, arcuate complex and capsular ligaments (Table 17.4).

Table 17.4 Structures of the PLC of the knee

Layer I	ITB and biceps femoris
	Common peroneal nerve lies between layer I and II
Layer II	Patellofemoral ligament and patellar retinaculum
Layer III	Superficial: LCL and fabellofibular ligament
	Deep: popliteus tendon, popliteofibular ligament, arcuate ligament, coronary ligament, lateral joint capsule
	Lateral geniculate artery lies between the superficial and deep layers of layer III

The LCL of the knee is a cord-like structure 5–7 cm in length. It is the primary static restraint to varus opening of the knee and secondary restraint to posterolateral rotation. It extends from an area proximal and posterior to the lateral epicondyle to the anterior aspect of the fibula head deep to the biceps femoris tendon. The LCL femoral attachment is 18.5 mm superiodorsal to the popliteus insertion. The popliteofibular ligament acts as a primary restraint to external rotation of the tibia on the femur at 30° of flexion. Similarly, the popliteus is a static and dynamic external rotation stabiliser.

The structures of the PLC function are to resist posterior translation as well as external and varus rotation of the tibia. They are the primary stabilisers of external tibial rotation at all knee flexion angles and the secondary restraints to anterior and posterior translation. Isolated PLC sectioning produces a maximal average increase of 13° of external rotation at 30° of knee flexion and only an average increase of 5.3° at 90°. Although isolated sectioning of the PCL has no effect on external tibial rotation, combined injury to the PCL and posterolateral structures leads to the highest increase in external rotation of 20.9°, especially at 90° of knee flexion[32,33]. Hence, the dial test is performed in the prone position at 30° of flexion to diagnose PLC injury and at 90° to diagnose combined PCL and PLC injuries.

Varus opening at 30° of flexion suggests LCL injury while varus opening at 0° is indicative of combined severe injury to the PLC and the cruciate.

Principles of surgical intervention

- Early repair (within 3 weeks) of torn and detached ligaments, tendons and capsule in acute injuries.
  A combination of early repair and reconstruction has been shown to provide better results
- Late reconstruction of two or three of the main stabilisers of the PLC of the knee, i.e. the LCL, popliteus tendon and popliteofibular ligament in chronic cases
- The reconstruction can be fibula-based, such as the modified Larson technique, or combined tibia- and fibula-based, such as the LaPrade anatomical reconstruction

- Combined ACL/PCL and PLC injury must be treated by reconstruction of all injured ligaments. Isolated ACL or PCL reconstruction without addressing the PLC will ultimately fail

Examination corner

Trauma oral 1

The examiner shows a clinical photograph of a dislocated knee (Figure 17.15) and asks:

- **Describe what you see.**

This is a lateral radiograph showing a dislocation of the right knee. I would like to see a AP radiograph to determine whether this is a posteromedial or posterolateral dislocation. There is also evidence of a bone fragment just anterior to the tibia which indicates an associated fracture. Associated fractures are seen in 60% of knee dislocations.

- **How do you classify this injury?**

This injury is usually classified on the basis of tibial displacement – This is only applicable if the knee is dislocated at the time of imaging. Anterior dislocation is the most common, followed by posterior dislocation as in this case. There can be medial and lateral as well as rotary (combination of the above A/P with M/L).

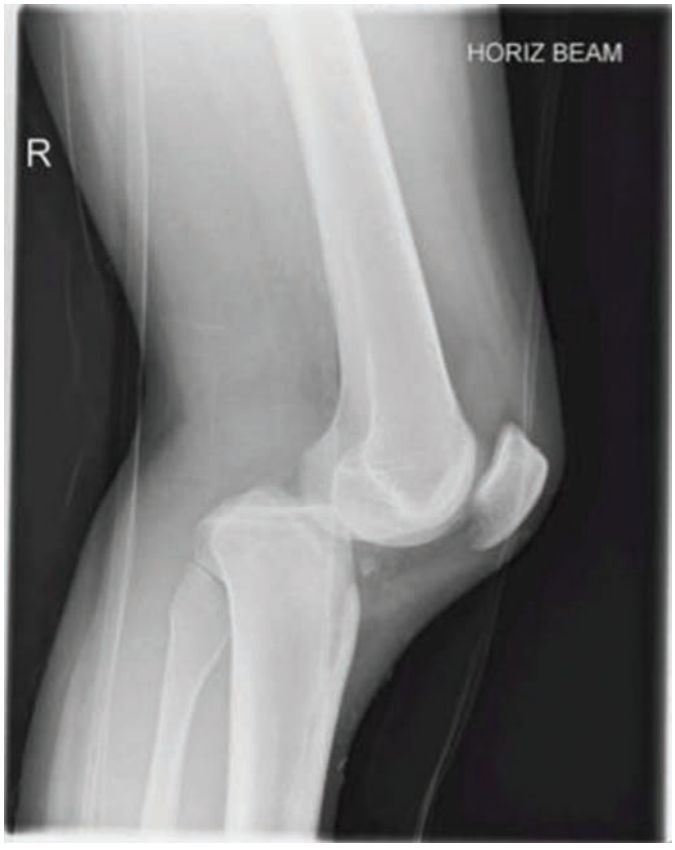


Figure 17.15 Lateral knee radiograph of a dislocation

The Schenk classification describes the dislocation according to the ligaments injured.

- **What is your initial management of a knee dislocation?**

This is a high-energy injury and should be approached using an ATLS® protocol. There is a considerable incidence of vascular and/or neurological injury. This is described in the literature at a rate of 20–50%. Compartment syndrome is also a risk factor in these injuries. Once the primary survey has been completed and the neurological and vascular status of the affected leg has been documented, I would administer sedation and attempt to reduce the knee. Once reduced, I would reassess the limb (neurological and vascular status) and document my findings.

- **Examiner interrupts: The knee was successfully reduced and remains so, but you are unable to feel a pulse. The foot remains warm and pink. What are your thoughts?**

Suspicion of a vascular injury warrants immediate intervention. I would discuss this with the vascular, plastic or if neither are available then the general surgeons (depending on what services are available in my hospital). I would alert theatres and prepare the patient for a spanning external fixation to stabilise the knee and popliteal fossa arterial exploration ± repair. An on table angiogram can be performed in theatre.

- **And what if there is a pulse?**

As this is a high-energy injury, I would discuss this with the radiologist and arrange a CT angiogram. If there is any difficulty in obtain the CT, I would perform an ankle-brachial pressure index. An index <0.9 in the context of this injury warrant surgical exploration. The benefit of the CT in such injuries is that it can detect intimal tears in the popliteal artery which might be masked by a normal pulse. The risk on an unidentified intimal tear is that it progresses or the artery forms a thrombus leading to ischaemia. If the foot had any signs of ischaemia, prompt vascular intervention is required.

- **Which types of dislocations are vascular injuries most commonly seen in?**

Around 20% of all dislocations have a vascular insult, with 50% being in the anterior or posterior knee dislocations. Anterior dislocations generally have an intimal tear from the traction applied on the artery, while posterior dislocations more commonly lead to complete tear of the popliteal artery. This is association of vascular injury with anterior and posterior dislocations are due to the anatomical trifurcation of the popliteal artery and its anchorage within proximal and distal soft tissues. Anterior and posterior dislocations lead to the artery tethering at the popliteal fossa. The artery proximally is within a fibrous tunnel at the adductor hiatus and then continues in the fibrous tunnel at soleus.

- **What is your order of ligament reconstruction in a multi-ligament knee injury?**

It all depends on the ligamentous injuries found on the MRI scan. If I decide to perform early reconstruction which has been shown to provide improved outcomes, I would reconstruct the PLC and PCL primarily (and perform a delayed ACL reconstruction). Neglecting to identify a PLC injury or not reconstructing/repairing it adequately leads to failure of knee stability.

- **How else can this injury be treated?**

If the dislocation has been reduced and is relatively stable, it can be braced with early rehabilitation. Once the scaring has occurred clinical as well as radiological assessment can be performed and at that point, any areas of instability that haven't resolved can be addressed. The commonest complication of knee dislocations (pre or post reconstruction) is arthrofibrosis leading to stiffness. In fact a large proportion of multi-ligament knee reconstruction require an MUA to improve the range of motion.

Patellofemoral (PF) joint disorders

This is regarded as the 'black hole of orthopaedics'. The international patellofemoral group explanation is that:

- The aetiology is complex and multifactorial in origin
- There is poor correlation between clinical symptoms, signs and radiological findings
- There is a lack of clinical interest
- Widespread terminological confusion
- Myths about anterior knee pain (AKP)
- AKP is self-limiting
- AKP is related to growth
- It is an expression of a psychological problem
- Vastus medialis obliquus (VMO) is responsible for patellar instability
- High Q angle indicates surgical realignment
- Lateral release improves AKP/instability
- Tissue homeostasis theory[34]
- PF joint pain can be caused by supraphysiological loading of the anatomically normal knee

Examination corner

Adult pathology oral 1

The examiner shows a clinical photograph of valgus knees and asks:

- **What is the Q angle and how do you measure it?**

The Q angle is an angle between (1) a line drawn from the anterior superior iliac spine to the midpoint of the patella (axis of the extensor mechanism) and (2) a line drawn from the midpoint of the patella to the tibial tuberosity (axis of the patella tendon).

The normal Q angle for males is 10–13° while is it 15–18° in females (i.e. females are more genu valgum than males).

- **What is its significance?**

It is very important in understanding patella bony malalignment. An increased Q angle leads to patella instability and this can be caused by femoral anteversion, genu valgum and external tibial torsion or pronated feet. Tight ligamentous structures can contribute to an increased Q angle.

In terms of arthroplasty, abnormal patella tracking is the most common complication of TKA and the key to normal patella tracking is to restore the Q angle. An increased Q angle leads to increased lateral forces on the patella femoral articulation which could lead to anterior knee pain, mechanical symptoms, accelerated polyethylene wear (in resurfaced patellae) and in extreme case patella dislocation.

- **What are the technical errors that need to be avoided in order not to increase the Q angle?**

One should avoid:

- Medialisation of the femoral component
- Internal rotation of the femoral component
- Internal rotation of the tibial plate
- Lateralizing the patella button on the patella (in resurfaced cases)

- **What is the best way of assessing component malrotation leading to patella maltracking after a total knee replacement?**

CT scan

Anatomy and biomechanics

Patellar articular cartilage is the thickest in the body. It has two main facets separated by a ridge. The medial facet is convex and the lateral facet is concave. The femoral trochlea has a higher and longer lateral facet compared with the medial side. The patella increases the efficiency of the extensor mechanism by 1.5 times and the muscles about the knee absorb more than three times the energy generated. Fifty per cent of the quadriceps tendon inserts into the upper pole of the patella and the rest blends into its anterior surface.

Ground Reaction Forces (GRF)

- Walking on level ground causes GRF of 0.5–3.0 times body weight (BW)
- Cycling: 1.2 times BW
- Stairs (up or down): 3.3 times BW
- Jogging and squat: Rise 6 times BW at 140°
- Squat: Descent 7.6 times BW at 140°

Typical anterior knee pain

- Pain (mostly dull/occasionally sharp) during: stair climbing – squatting – prolonged sitting – rising from a chair
- Giving way (quads inhibition)
- Catching or pseudo-locking
- Mild swelling caused by synovial irritation

Sources of anterior knee pain

The articular cartilage is avascular and aneural; therefore, the possible theories are:

- Synovial irritation
- Subchondral bone deformation
- Intramedullary pressure changes
- Ischaemia-induced neural proliferation and pain

- Lateral neuroma formation
- Tissue homeostasis theory

Causes of anterior knee pain

Local

- Muscle imbalance
- Soft-tissue imbalance
- Tightness: Quads, hamstrings, ITB or lateral retinaculum
- Laxity: Hypermobility syndrome
- Retinacular pain from overload or neuroma
- Patellar or quadriceps tendinopathy
- Synovial – Synovitis – Plica – Tumour (PVNS, chondromatosis)
- Articular damage – Patella/trochlea
- Traumatic
- Degenerate
- Inflammatory
- Fat pad syndrome
- Stressed bone, Osgood–Schlatter's disease/Sinding–Larsen–Johansson syndrome

Distant

- Mal-alignment (high Q angle)
- Core pelvic muscles
- Femoral anteversion 'leaning forward'
- Tibial torsion
- Foot hyperpronation
- Referred: Spine or hip (SUFE!) via the obturator nerve.

Examination

- Standing: Valgus/varus alignment, gait, leg length inequality, Q angle
- Sitting: VMO/quads atrophy, lateral patellar tilt, patellar tracking, J sign
- Supine: Patellar glide test, patellar tilt test, Clarke's test (commonly painful – Examiner likely to stop you from performing it), apprehension test, compression test and trochlear depth in hyperflexion

A patella that deviates laterally in terminal extension (J sign) suggests significant mal-alignment that may benefit from a distal realignment. Patellar tilt associated with lateral patellar compression, if severe, can be treated with lateral retinacular release.

Patellar instability

Recurrent patellar subluxation or dislocation can be very disabling.

Risk factors

1. Bony factors (static)
   - Trochlear dysplasia
   - Hypoplastic femoral condyle
   - Patellar shape
   - Patella alta
2. Mal-alignment
   - Patellar mal-alignment is an abnormal rotational or translational deviation of the patella along any axis
   - External tibial torsion/foot pronation
   - Increased femoral anteversion
   - Genu valgum
   - Increased Q angle or abnormal tibial tuberosity–trochlea groove (TT–TG) distance
1. Soft tissue (dynamic)
   - Ligamentous laxity (medial patellofemoral ligament rupture)
2. Abnormal gait
   - Walking with valgus thrust

Medial patellofemoral ligament (MPFL)

This is the primary static soft-tissue restraint to lateral patellar displacement. It provides 60% of the total medial restraining force[35]. The MPFL is most effective between 0° and 30° of flexion, as the trochlea, which is a primary restraint, provides stability with further flexion. MPFL sectioning can lead to substantial changes in patellar tracking. It originates from an

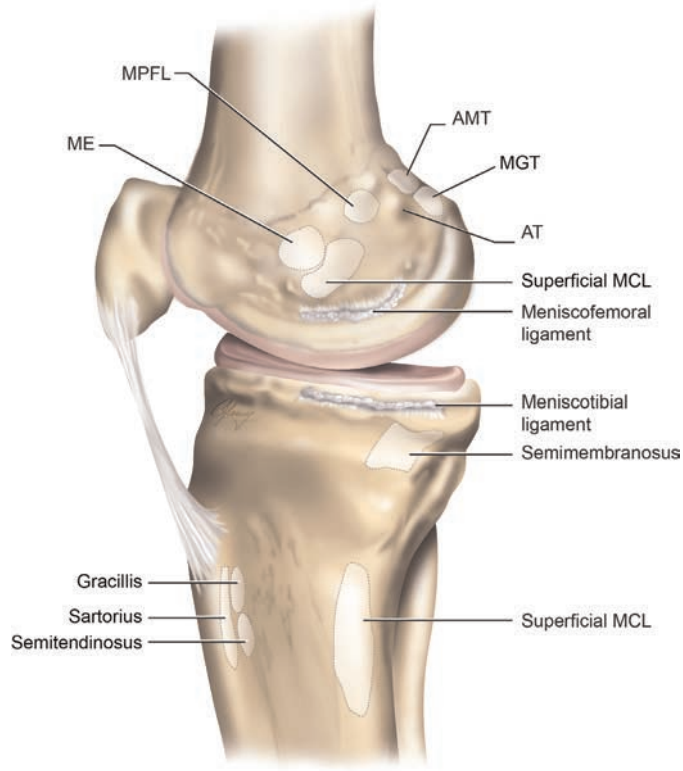


Figure 17.16 Medial structures of the knee. AMT, adductor magnus tendon; AT, adductor tubercle; GT, gastrocneumius tubercle; ME, medial epicondyle; MGT; medial gastrocnemius tendon; MPFL, medial patellofemoral ligament; POL, posterior oblique ligament; sMCL, superficial medial collateral ligament

area between the medial epicondyle and the adductor tubercle and inserts onto the proximal two-thirds of the patella. The average length of the ligament is 5.5 cm. During acute patellar dislocation there is a 90–95% incidence of damage to the MPFL. Nomura et al. reported almost every acute knee dislocation has resulted in an MPFL rupture[36]. Femoral attachment is commonly affected. In the past 10 years, MPFL reconstruction has become a popular procedure for treatment of recurrent patellar dislocation.

Investigations

- A lateral radiograph is the most helpful view for assessment of patellar tilt and trochlear depth
- Axial radiographs (merchant's view) to assess patellar tilt angle (normal <10°), congruence, sulcus angle (normal 138°) and trochlear dysplasia
- MRI for articular lesion. This also confirms the site of avulsion. Rupture is most commonly at the femoral origin but occasionally can be at the patella insertion
- CT scan to assess
  - Femoral anteversion (normal 5–15°)
  - Tibial torsion
  - TT–TG distance >15–20 mm is significant
  - Patellar tilt
  - Trochlear depth
- Isotope bone scan to measure bony activity and homeostasis within the knee can sometimes be useful

Management

Non-operative

The first line of treatment should always be non-operative measures. The patient's education and intensive rehabilitation under the supervision of a skilled physiotherapist plays a major role in the success of non-surgical treatment. Acute first-time patellar dislocation is treated conservatively in an extension splint for 2–4 weeks. Occasionally, surgical intervention is necessary to fix or remove an osteochondral fracture. Rarely, the medial patellofemoral ligament is repaired or reconstructed acutely. Despite intensive rehabilitation there is a 20% risk of recurrent dislocation. This figure increases to 50% after the second dislocation.

Surgical

This is only carried out when intensive rehabilitation fails to prevent further dislocation.

MPFL reconstruction

- Indications: Recurrent patella instability (lateral) with no underlying structural mal-alignment or minor mal-alignment
- Gracilis hamstrings autograft are most commonly used in this procedure. Harvest technique as per ACL reconstruction. Some have also used synthetic grafts as this procedure
- Femoral attachment should be identified using fluoroscopy. The tunnel is classically placed midway between the medial epicondyle and the adductor tubercle, and just posterior[37] to a line extending from the posterior cortex of the femur contrary to what is called (Schottle's point) which is more anterior and lies anterior to the line extending from the posterior cortex of the femur . The graft should be fixed at 60° of knee flexion applying only 2 Newton of force[38]
- This procedure offers very good outcomes if performed with the appropriate indications and can be combined with bony alignment procedures[39]

Proximal realignment procedure

- Lateral release (open/arthroscopic) is rarely performed nowadays in isolation. It is only indicated when there is pain and lateral retinacular tightness or when the patella is chronically dislocated
- Medial imbrication (open/arthroscopic). Indicated in mild to moderate maltracking, especially in the skeletally immature
- Quadricepsplasty

Combined proximal and distal realignment procedures

These procedures are indicated for tubercle mal-alignment and traumatic incompetency of the medial restraints.

Distal realignment procedures

Direction of tibial tubercle (TT) transfer:

- Medial transfer to treat mal-alignment
- Anteromedial transfer for mal-alignment and PF joint chondrosis
- Anterior when there is distal PF joint chondrosis

Elmslie–Trillat: medialisation without posteriorisation of the tibial tubercle

Fulkerson: medialisation with anteriorisation of the tibial tubercle in the arthritic patella. The obliquity of the cut depends on the degree of mal-alignment and arthrosis. A steep cut up to a 60° angle maximizes anteriorisation and is useful in patients who have more arthrosis than mal-alignment

Historical/abandoned procedures (these are still relevant for clinical practice as patients might have had them done or require revision procedures, and an understanding of what has been done is essential for any surgical planning):

Hauser: transfer of the TT to a medial, distal and posterior position. It increases the PF joint reaction force and causes patellofemoral degenerative joint disease

Goldthwait 1899–Roux 1888: medial transposition of the medial half of the patellar tendon, lateral release/medial reefing. Now the lateral half is placed under the medial half and medially

Maquet: anterior transportation of TT, which decreases patellofemoral contact forces. Not performed nowadays as it has a high incidence of skin necrosis, compartment syndrome and no effect on the Q angle

Summary

Identify the cause of instability and take the following actions accordingly:

- Lateral release when there is lateral patellar compression syndrome
- Tibial tubercle medialisation when there is abnormal TT : TG distance
- Tibial tubercle distalisation when Caton–Deschamps or Insall–Salvati patella index ratios >1.3 (Figure 17.17)
- Tibial tubercle elevation when there is patellar chondrosis
- Manage the immature knee by soft-tissue realignment procedures

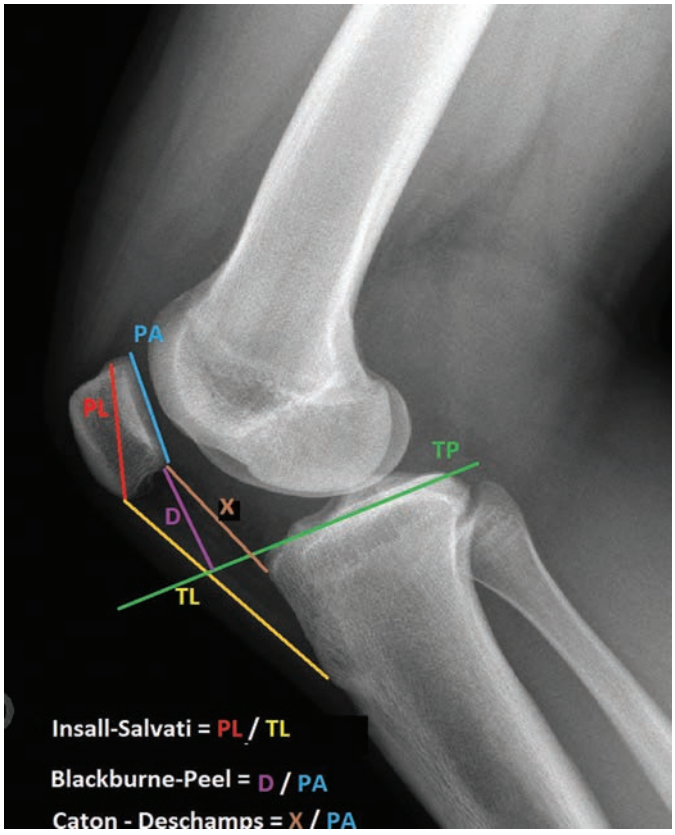


Figure 17.17 PL: Maximum patella length. TL: patella tendon length. PA: patella articular surface. TP: tibial plateau line. D: Shortest distance between the patella articular surface and the tangential line of the tibial plateau. The Insall–Salvati ratio is PL/TL, the traditional number used is <1.2 (0.8–1.2) with patella baja being <0.8 and patella alta is >1.2. The Blackburne–Peel ratio is measured on a lateral radiograph with 30° of flexion. It is a measure of patella height taking the patella articular surface (PA) divided by the distance between the horizontal line at the level of the tibial plateau and the inferior aspect of the patellar articular surface (D): (D/PA). A ratio of 0.8 is considered normal, with patella alta being a ratio of >1.0. Caton–Deschamps index is measured by X/PA. A ratio >1.3 is highly suggestive of patella alta

- Medial patellofemoral ligament reconstruction is indicated in recurrent dislocation when other parameters are within normal or near normal
- Manage trochlear dysplasia (true lateral x-ray and CT scan proven) by trochleoplasty

Patellofemoral joint arthroplasty

This is effective in isolated PF joint arthritis (Figure 17.19a and b), post-traumatic arthrosis and severe chondrosis after an extended period of supervised and non-operative measures.

Contraindications

- Inflammatory arthritis
- Chondrocalcinosis of menisci or tibiofemoral surface
- Patients with inappropriate expectations
- Considerable patellar mal-tracking or mal-alignment
- Patellar tendonitis, synovitis and patellar instability

Outcome

- Some studies reveal up to 90–95% good and excellent results in isolated PF joint arthrosis at mid-term follow-up: This includes the Avon patellofemoral arthroplasty[40,41]
- NJR reports revision rates of PF joint implants of 14.7% at 8 years
- Obesity and ACL deficiency do not seem to increase the failure rate
- It is an excellent alternative to patellectomy and accepted alternative to TKR in patients younger than 55 (in centres that do them regularly)

Examination corner

Adult and pathology oral 1

EXAMINER: A 44-year-old female presents to the knee clinic with knee pain that has been going on for some years, getting progressively worse. Her GP has tried multiple therapies but she is no longer coping with her symptoms.

Describe the radiographs (Figure 17.19).

These are skyline views of both knees.

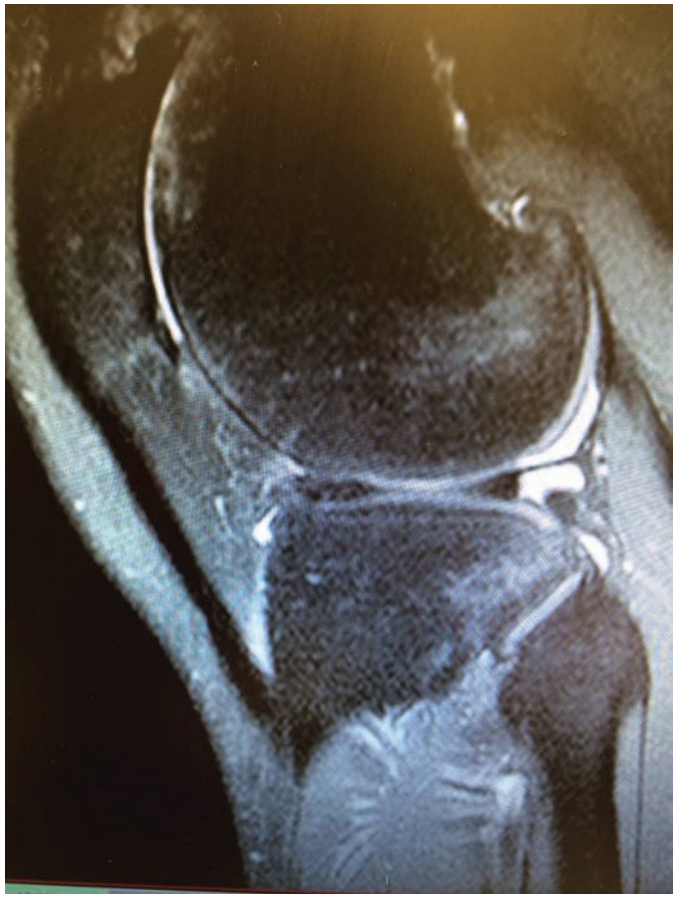
In a skyline or merchant's view what would you comment on?

I would look at the patella position and comment on whether there is evidence of a tilting patella, joint space within the patellofemoral joint, the trochlear morphology and the pattern of OA or compression of the patella onto the torchlea.

- **If the rest of the knee joint was normal what would be your diagnosis?**

Isolated patellofemoral joint osteoarthritis.

(a)



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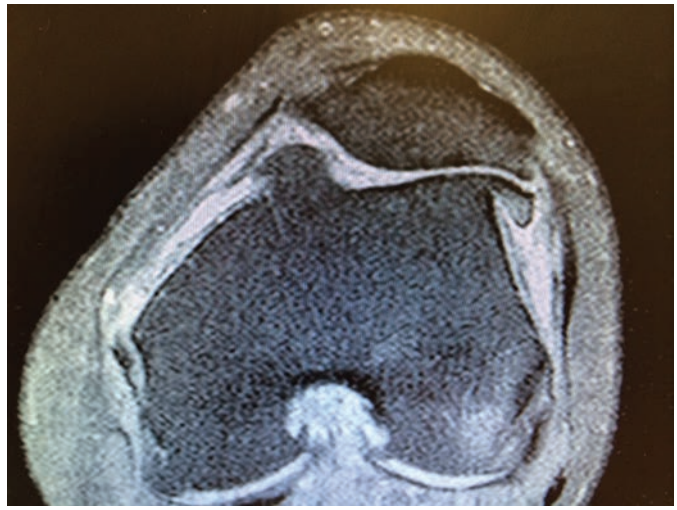

Figure 17.18 Sagittal and axial T2-weighted MRI of an isolated PF joint OA affected knee in 43-year-old man

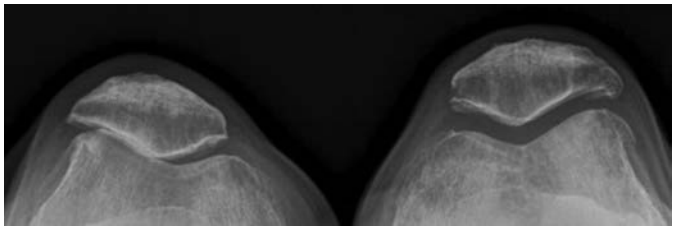

Figure 17.19 Knee radiograph with PF joint OA

- **What is the aetiology of this condition?**

Aetiology is complex and multifactorial and relates to any factor that increases PF joint pressure. It can be divided into three main factors:

- Alignment: This can affect the position of the patella in either rotational or translational plane leading to an increased Q angle or abnormal tibial tuberosity–trochlea groove (TT–TG) distance. This can also be affected by genu valgum, increased femoral anteversion or external tibial torsion. Valgus mal-alignment is associated with increased risk of disease progression
- Static (bony): Abnormalities within the trochlear groove depth or patella abnormality (lateral tilt)
- Dynamic (soft tissue): Tight lateral structures (lateral retinaculum) or incompetent medial structures (VMO/MPFL) both contribute to PF joint OA especially in chronic cases that have progressed beyond instability

Other factors include trauma – 28% of patients with isolated PF joint OA report previous patella instability compared with none in the tricompartmental knee OA[42]. Obesity is also associated with PF joint OA.

What do radiological findings determine?

Radiological findings correlate poorly with clinical symptoms and signs.

What are the management options for a patient with PF joint OA?

I would initially investigate what treatment the patient has had already.

Non-operative measures:

- Weight loss
- Activity modification
- Physiotherapy (VMO strengthening might improve symptoms)
- Analgesia

- Intra-articular injections (corticosteroid – NICE does not recommend hyaluronic acid)

Surgical measures: these need to be tailored to the patient and the clinical/radiological findings.

- Arthroscopic debridement/microfracture/chondroplasty/osteochondral graft
- Lateral patella facetectomy
- Distal ateriorizing procedures (Fulkerson or Maquet procedures)
- Patellofemoral arthroplasty (PFA)
- Total knee arthroplasty (TKA)

When would you decide to perform a PFA? And what are the contraindications?

PFA is indicated for isolated PF joint OA with correlated severe knee symptoms.
Contraindications to this procedure include inflammatory arthritis, medial/lateral tibiofemoral OA, chondrocalcinosis, patella instability or patella mal-tracking.

What type of arthroplasty would you offer this patient if you had to choose and why?

With careful patient selection I would offer this patient a PFA – Avon implant. I would refer it to a centre that does this procedure on a routine basis.
NJR results reveal that the median age for the patients undergoing PFA is lower than TKA.
Eight-year survival of PFA is 14.7%, which is considerably higher than TKA (2.82%), but the Avon patellofemoral implant has an improved revision rate of 10.4% at 8 years.
PFA vs TKA: a systematic review by Van Jonbergen[43] showed that the clinical results reported on PFA outcome studies are related to prosthetic design, surgical technique, patient selection and length of follow-up. Two-thirds of patients have shown good to excellent results in their 3- to 17-year follow up.
Van Jonergen in another study revealed that patellofemoral arthroplasty does not have a negative effect on the outcome of later TKA[44].

What are the most common reasons for the failure of the PFA implant?

Most common reason for revision is pain and aseptic loosening and progression of femorotibial OA. Progression of OA will require conversion to a primary non-constrained TKA.

Patellectomy

This is salvage last-resort surgery – It may not eliminate pain. It reduces extension power by 30–50%. The tibiofemoral joint reaction force may increase by 250%, causing OA. Although rarely performed, there are satisfactory results in 77%[45].

Lateral patellar compression syndrome

There is pain caused by a tight lateral retinaculum. Usually the patella has normal alignment and mobility. Imaging shows an abnormal patellar tilt without subluxation. Arthroscopic lateral release is generally very effective.

Patellofemoral arthritis in young patients

Non-surgical

- Patient education, physiotherapy, activity modification
- Optimize body weight

Surgical

- Arthroscopic cartilage debridement (gentle), lateral release (tilt and arthritis), Fulkerson tibial tubercle elevation for distal focal lesion, patellar resurfacing, patellofemoral joint arthroplasty, TKR (older patients) and patellectomy (rarely)[45]

Knee osteoarthritis

Unlike hip arthritis, knee osteoarthritis does not always present with night pain. Women have a higher overall prevalence of OA and have more severe OA of the knee than men. Despite that they are three times less likely to undergo TKR. Women have thinner distal femur articular cartilage. They also have a thinner patellar articular cartilage and are more susceptible to isolated PF joint arthritis[46,47].

Differential diagnosis of painful swollen knee

- Mechanical pain occurs when the joint is stressed/loaded (e.g. degenerative arthritis)
- Inflammatory: Occurs mainly at rest such as:

Inflammatory polyarthropathy (e.g. rheumatoid arthritis)
Crystal-induced
Spondyloarthropathy (e.g. ankylosing spondylitis and psoriasis
Infectious (e.g. staphylococcal, gonococcal and Lyme arthritis)
Neuropathic: Related to nerve distribution
Psychosomatic: Poorly defined pain that is disproportionate to clinical signs
Benign synovial disorders
Pigmented villonodular synovitis
Synovial chondromatosis
Synovial haemangioma
Lipoma arborescens

Non-surgical management of arthritis

- Exercise, muscle strengthening and aerobic exercises
- Weight loss – Reduce the progress of arthritis and reduce pain
- NSAIDs – GI side effects with chronic use (consider addition of PPI or COX-2 inhibitors)
- Bracing and orthotics – Bracing and orthosis for passively correctable unicompartmental disease, $<10°$ angulations (off-loader valgising brace for isolated medial OA)
- Steroid injection, corticosteroid and lignocaine are considered toxic to chondrocytes as shown in some studies, and, hence, should be used with caution in early OA[48,49]

Surgical management of arthritis in young patients

- Arthroscopy: This is controversial, and is only used for debriding chondral flaps and symptomatic meniscal tears, as arthroscopic surgery for OA of the knee has been shown provides no additional benefit to optimised physical and medical therapy[50]
- High tibial osteotomy was popularized by Coventry and Insall in the 1970s
- Unicompartmental knee replacement
- Total knee replacement

Proximal or high tibial osteotomy (HTO)

This is an excellent operation for relatively young or highly active individuals, with isolated medial compartment OA and varus alignment of the knee.

The principle is to realign the weight-bearing axis from varus to slight valgus i.e. offloading the medial compartment.

Realignment can be achieved either by closing wedge lateral HTO (commonly used), opening wedge medial HTO or dome osteotomy, which is more technically demanding.

Prerequisites for HTO

The place of osteotomies in the management of osteoarthritis of the knee was formulated by the International Society of Arthroscopy, Knee Surgery and Orthopaedic Sports Medicine (ISAKOS) in 2004.

The ideal patient and the not suitable patients for HTO according to ISAKOS

Ideal candidate

- Age 40–60 years
- <15° varus mal-alignment of the limb
- >100° flexion in knee
- None smoker
- BMI <30
- No PF joint arthrosis
- Stable knee
- Normal lateral compartment and arthrosis grades I–III in the medial compartment
- No meniscectomy
- No cupula: dished-type defect in the posteriomedial tibia. Leading to a fixed anterior subluxation of the tibia. This defect is associated with ACL deficient knee and act as a resting place for the femur
- Patients should be able to use crutches and have no major varicose veins or peripheral vascular disease

Contraindications (unsuitable patients)

- Severe OA changes in the lateral compartment or PF joint
- Severe medial compartment arthritis
- Incompetent MCL
- Coronal knee subluxation (tibial subluxation >1 cm)
- Inflammatory arthritis
- Severe osteoporosis
- Extra-articular deformity
- Crystal arthropathy
- Near total lateral meniscectomy
- Obesity (BMI >40): Valgus knee is poorly tolerated because of medial thigh contact
- Large varus thrust
- If >20° correction is needed
- Patellofemoral OA is a relative contraindication

Li et al.[51] revealed that among the 11 studies they looked at in their systematic review, the clinical outcome of simultaneous HTO and ACL reconstruction for medial compartment OA in the young patient with an ACL-deficient knee provides a satisfactory restoration of the knee stability, improvement in pain, more predictable return to sport, and alleviation of medial OA.

Planning

- Standing, long leg radiographs in neutral rotation
- Measure the mechanical axis (normal = 1.2° varus)
- Anatomical axis (6–7° valgus)
- Measure the degree of deformity and plan the size of wedge necessary
- Change the overall alignment to transfer the load to the unaffected side
- Sixty-four per cent across the tibial plateau from the medial side (Fujisawa point)[52]
- Varus arthritis can be treated with laterally based closing wedge osteotomy above the level of the tibial tubercle or medial opening wedge HTO
- Final alignment should create 10–13° valgus in medial OA. Overcorrection of 3–5° above the 6–7° normal valgus angle
- The medial tibial cortex represents the apex of the bony wedge in lateral osteotomies and should be left intact

Methods of osteotomy fixation

- Cast immobilization
- Staples
- Osteotomy specific plate and screws (most common method)
- External fixator
- Distraction osteogenesis: Correction can be adjusted after surgery. However, pin tracts create a potential problem for subsequent TKA

Closed wedge HTO

Surgical technique

- Computer-aided measurement of the wedge size can be used
- A 10-mm wedge excision leads to 10° corrections in a 57-mm-wide tibia
- An angular jig is more accurate
- Curved incision from the head of the fibula to 2 cm below the tibial tubercle

- The tibia is dissected subperiosteally anteriorly and posteriorly
- The proximal fibular head is excised at the superior tibiofibular joint or the proximal tibiofibular joint is separated using a Cobb elevator protecting the peroneal nerve
- Identify the bare area of the fibular head (safe landmark)
- A calibrated osteotomy guide must be used for the bone cut
- Leave 15–20 mm of tibial plateau to avoid fracture
- Fix with a plate or staples
- Rigid fixation and early mobilization eliminates patellar ligament contracture (leading to patella baja)
- DVT prophylaxis is similar to that after TKR

Complications

- Inadequate valgus correction: Aim for tibiofemoral angle of 11–13° valgus
- Overcorrection: PF joint derangement
- Recurrence of deformity
- Alteration in patellar height (patella baja)
- Intra-articular fracture
- Osteonecrosis of the tibial plateau
- Vascular injuries: Anterior tibial artery, popliteal artery
- Peroneal nerve palsy
- Delayed or non-union
- Compartment syndrome
- A more challenging TKR procedure when needed in the future
- Varus laxity (loose LCL)

Open wedge HTO

- The open wedge HTO gained recognition after the encouraging reports by Professor Hernigou in 1987[53]

Advantages

- Preserves bone stock (subsequent TKR is technically easier)
- Makes tightening of the MCL easier
- Preserves the lateral side for LCL or posterolateral reconstruction if insufficient
- No risk to peroneal nerve
- Less dissection
- Easier to achieve precise angular correction
- Better control over the posterior tibial slope

Disadvantages

- Requires a bone graft (substitute, autograft, allograft)
- Increased incidence of non-union and delayed union
- Large correction may affect leg lengthening
- Loss of fixation and recurrence of varus deformity
- Worsens patella baja by raising knee joint line
- Slow rehabilitation
- Plate fixation makes TKA harder

The outcome of the various high tibial osteotomies strongly depend on the amount of correction achieved. Under-correction leads to recurrence of the varus deformity. On the other hand over-correction leads to valgus overload and subsequent lateral compartment degeneration[54].

Open wedge HTO

Surgical technique

- Medial longitudinal incision or an oblique incision
- The MCL is mobilized posteriorly
- Use two 2.5-mm Kirschner wires to mark the oblique osteotomy
- Starting proximal to the pes anserinus 4–5 cm distal to the medial joint line
- Drive the wires to the tip of the fibula 10–15 mm below the lateral joint line
- The osteotomy of the posterior two-thirds of the tibia should hinge on the lateral (not posterolateral) side of the tibia
- Leave a 10-mm lateral bone bridge intact laterally
- The second osteotomy begins in the anterior one-third of the tibia at an angle of 135° while leaving the tibial tuberosity intact
- To compensate for an ACL-deficient knee one can decrease the posterior tibial slope, i.e. open a bigger gab posteriorly
- To compensate for PCL-deficient knee the slope should be increased, i.e. creating a bigger opening anteriorly

Distal femur osteotomy

Varus-producing HTO can be used to correct lateral compartment arthritis and valgus deformity $<12°$; however, a deformity of 12° or more needs distal femoral varus-producing osteotomy to address a lateral femoral condyle deficiency and to prevent joint line obliquity and gradual lateral tibial subluxation.

Either lateral distal femur opening wedge osteotomy using a Puddu plate/Tomofix or a medial distal femur-closing wedge osteotomy are undertaken.

Coventry et al.[55] reported a 5-year survival of 87% and a 10-year survival of 66%. However, the 5-year survival was reduced down to 38% when valgus angulation at 1 year was $<8°$ in a patient whose weight was >1.32 times the ideal weight.

Examination corner

The examiner showed a radiograph of PCL avulsion and asked:

- Describe the injury
- How do you attach the avulsed fracture?
- Describe the posterior approach to the knee

Knee arthritis and arthroplasty

The primary aim of total knee replacement (TKR) is to achieve: Pain relief which would lead to improved mobility and increased range of motion. This is achieved by obtaining

- A weight-bearing line through the centre of the knee or a postoperative mechanical axis of 0 ± 3°
- A joint line perpendicular to the weight-bearing line
- Soft-tissue balance
- Restoring normal Q angle and joint alignment

Anatomical and mechanical axes

The valgus cut angle is the angle between the femoral anatomical and mechanical axes. The normal anatomical axis or tibiofemoral angle measures 5–6° of valgus.

The mechanical axis, or weight-bearing line, is the line from the centre of the hip to the centre of the tibiotalar joint; it typically measures 1.2° of varus. Hence, 60% of the weight goes through the medial compartment.

Femoral roll-back

Femoral roll-back is the posterior shift in the femoral–tibial contact point in the sagittal plane as the knee flexes.

Aetiology of arthritis

- Idiopathic
- Post-traumatic
- Avascular necrosis
- Inflammatory arthritis

Contraindications to TKA

- Infection
- Neurogenic genu recurvatum
- Deficient quadriceps mechanism (polio)

Constraint ladder within knee implant design

- PCL retaining (cruciate retaining or CR)
- Rotating platform more constrained due to conformity
- PCL substituting (posterior stabilised or PS)
- Unlinked (non-hinged) constrained condylar implant (varus–valgus constrained or VVC)
- Linked (hinged), constrained condylar implant (rotating-hinge knee or RHK)

PCL retaining (CR)

The PCL is a major stabilising ligament in the normal and pathological knee. It tightens the flexion space and act as a secondary mediolateral stabiliser in flexion.

Advantages (compared to PCL-substituting design)

- Provides least constraint
- Lowered shear forces at the tibial component–host interface
- Preserves proprioceptive fibres (intact PCL)
- Greater stability during stair climbing (quadriceps strength)
- Fewer patella complications
- Preserve bone stock on the distal femoral cut
- Better kinematics but relatively less predictable (controversial)
- Avoids the tibial post-cam impingement
- Ease of management of supracondylar fracture (plate/nail)

Disadvantages

- Less conforming surfaces to allow roll-back
- Slide: Increases contact stresses and polyethylene delamination
- Technically more difficult to balance
- Loose or ruptured PCL can lead to flexion instability

PCL substitution/sacrificing

Indications

- Previous patellectomy
- Rheumatoid arthritis
- Stiff knee in post-traumatic arthritis
- Previous HTO
- Large deformity requiring the release of PCL
- Deficient or absent PCL

Advantages

- Conforming surfaces allowing roll-back
- No component slide
- Provides a degree of VVC
- Cam-post mechanism improves anterior-posterior stability
- Uses more congruent joint surfaces than CR, which reduces wear
- Facilitates any deformity correction
- Better range of motion
- Technically easier (to balance) than CR and reproducible
- Higher degree of flexion

Disadvantages

- Increased constraint associated with high stresses at fixation interface leading to increased loosening
- Femoral bone loss
- Tibial post increases wear
- Tibial post dislocation (Cam jump)
- Three times greater joint line alteration compared to CR
- Patella clunk/crunch syndrome

Mobile-bearing tibial components

Clinical trials have shown that the mobile bearing design does not provide any functional or radiological advantage over fixed-bearing prostheses[56,57].

Theoretical advantages

- Maximum conformity without an increase in component loosening
- Increased contact area in both sagittal and coronal planes
- Minimal constraint
- Reduced component sliding during flexion

- Reduced shear stresses on the polyethylene insert
- Allows self-correction of tibial component in rotational mal-alignment
- Facilitates patellar tracking
- Better kinematics in gait
- Low polyethylene wear

Theoretical disadvantages

- Bearing dislocation and spin out if the soft tissues are imbalanced
- Underside bearing wear creating small debris; hence, more osteolysis
- Technically difficult, less forgiving soft-tissue imbalance

Constrained unlinked (non-hinged) condylar implant (VVC)

Constrained prosthesis with a long tibial post without a link connecting the tibial and femoral components, e.g. constrained condylar knee (Legion by Smith and Nephew and TC3 by DePuy).

Indications

LCL or MCL minor deficiency
Excessive bone lose
Weak bone at the epiphysial interface

Advantage

- Provides anteroposterior and varus-valgus as well as some rotational stability (substitute for deficient collaterals or bone loss)

Disadvantages

- Increased femoral bone resection
- Aseptic loosening due to the large forces acting on the prosthesis (these are stemmed implants)

Constrained-linked (hinged) condylar implant (RHK)

- Rarely indicated

Indications

- Used for global instability (total collateral ligament disruption/recurvatum)
- Severe bone loss ± neuropathic arthropathy (Charcot joint)
- Tumour resection
- Hyperextension instability, e.g. in polio

Advantage

- Provides stability in cases of large bone loss and significant instability

Disadvantages

- Large bone resection
- High level of constraint leading to increased rate of aseptic loosening due to the large forces acting on the prosthesis (these are stemmed implants)
- Periprosthetic fractures for the stress risers created

Knee arthroplasty surgical technique

Skin incision

- Anterior longitudinal midline skin incision
- Skin blood supply is in the subcutaneous fat so avoid undermining
- Medial vessels are relatively large so in cases where there are multiple scars use the most lateral

Deep dissection

- Medial parapatellar in most cases
- Subvastus, midvastus
- Lateral parapatellar (very valgus knee, laterally subluxed patella)
- Tibial tubercle osteotomy (Whiteside)
- Rectus snip
- Quadriceps turn-down

Soft-tissue balancing

- Collateral ligaments are no longer isometric but act as a sleeve
- Sleeve release affects both flexion and extension gaps
- Medially, posteromedial release affects extension only
- Laterally, iliotibial tract and posterolateral release affect extension only
- In FFD release the tight posteromedial corner first

Equal flexion/extension gap (Table 17.5)

- If the flexion and extension gap is symmetrical, adjust the tibia
- If the gap is asymmetrical, adjust the femur (majority of cases)
- Downsize the femur, cut more off the posterior femoral condyle
- Resect the distal femur to increase the extension gap
- Increasing the tibial slope increases the flexion gap
- PCL excision increases the flexion gap by roughly 5 mm

Tibia cut

- Posterior slope 3–5° generally, but depends on knee design
- PS knee performs better with no slope
- CR knee performs better with a 3–5° slope

Distal femoral cut

- Valgus angle 5–7° from anatomical axis
- Perpendicular to mechanical axis
- Intramedullary alignment jig
- Cut less femur in CR knee
- Cut 1–2 mm more femur in PS knee

To make flexion gap rectangular

- Externally rotate the femoral cutting block 3° or align it parallel to the femoral epicondylar axis

Table 17.5 Balancing the flexion and extension gaps

	Flexion gap loose	Flexion gap OK	Flexion gap tight
Extension gap loose	Thicker insert	Augment femur/downsize femur	Convert to PS design/downsize femur and use thicker insert
Extension gap OK	Resect more distal femur and use thicker insert Oversize femoral component	Perfect	Downsize femur/increase tibial slope
Extension gap tight	Resect distal femur Release capsule posteriorly	Resect distal femur/release posterior capsule	Thinner plastic insert/cut more tibia

- Flexion/extension gaps should be rectangular and equal
- Never internally rotate the tibial component

Mal-alignment

- Coronal mal-alignment causes a 24% failure rate at a median period of 8 years[58]. The best implant survival has been in knees with overall alignment between 2.4° and 7.2° of valgus tibiofemoral angle[59]. Computer navigation has been advocated to decrease variability in surgical technique and improve implant durability, and ultimately hoping to lead to improved patient outcomes
- Recent evidence, however, has challenged this concept by showing that a postoperative mechanical axis of 0 ± 3° did not improve 15-year implant survival[60]
- The optimum AP position of the femoral component is in line with the anterior cortex of the femur
- A forward femoral implant leads to overstuffing of the patella and instability in flexion
- A medialized femoral component leads to patellar mal-tracking
- A posterior tibial slope of >10° leads to premature failure
- Tibial medial overhang causes impingement and pain (minor lateral overhang is acceptable)
- A slight posterior overhang is acceptable
- A posterolateral overhang may cause popliteus muscle impingement

Medial release for varus knee

1. Osteophyte excision
2. Deep MCL to posteromedial corner (pie crusting)
3. Semimembranosus aponeurosis
4. Superficial MCL
5. Pes anserinus insertion
6. PCL

Mostly 1 and 2, but if still tight proceed to 3–6. Check stability at each stage.

Lateral release for valgus knee

See 'Valgus knee', below.

Fixed flexion deformity

- <10° can be corrected by cutting bone
- May need to resect more bone from the femur
- Remove posterior osteophytes
- Severe FFD needs posterior capsular cutting (with great care) with the knee in extension and the capsule under tension (lamina spreader)
- For very severe FFD, use a Cobb to lift the posterior capsule of the femur while sticking to bone

Patellofemoral maltracking

This is potentially a major problem after TKA. If the patella is mal-tracking, it is advised to release the tourniquet and re-assess prior to taking any action. To prevent mal-tracking the surgeon should:

- Externally rotate the femoral component
- Lateralise the femoral component
- Avoid anterior placement or oversizing of the femoral component
- Avoid internal rotation of the tibial component (increases the Q angle)
- Avoid an excessive valgus angle
- Avoid raising the joint line
- Medialize the patellar button
- Avoid inferior placement of the patellar component

The following cause increased anterior displacement of the patella:

- Oversized femoral component
- Overstuffing the patella

Patella resurfacing debate

This issue is still contentious in the field of arthroplasty. Recent long-term follow-up of 78 knees showed no difference in outcome between resurfaced and non-resurfaced knees[61]. For resurfacing:

- Reduces anterior knee pain
- Improves knee strength in flexion (stair descent)
- Less likely to revise the knee for anterior knee pain

Against resurfacing:

- No difference in outcome
- Increase wear particles
- Long-term problems with patellar fracture

Historically, extensor mechanism problems occurred in up to 10% of patients and accounted for up to 50% of the long-term problems of TKA. Reported problems include:

- Patellar tendon avulsion
- Patella fracture and AVN
- Patellofemoral instability
- Component loosening

Indications for selective patella replacement:

- Advanced osteoarthritic patella
- Rheumatoid arthritis
- Preoperative patellofemoral pain
- Obese patients
- Overweight females
- Chondrocalcinosis

Examination corner

Adult orthopaedic and pathology oral 1

EXAMINER: While performing a total knee arthroplasty, what are the options in regards to the patella?

CANDIDATE: What to do with the patella during total knee arthroplasty is a controversial topic with no consensus in the orthopaedic literature. The options are to leave the patella as it is regardless of the amount of degenerate changes present, remove all patella osteophytes, circumpatellar electorcautery or perform patella resurfacing.

There has been a recent RCT published in the BJJ in 2014 of 300 knees revealed that the improved clinical outcome with electrocautery denervation compared with no electrocautery of the patella is not maintained at a mean of 3.7 years' follow-up[62].

Another prospective, randomised, double-blinded study of 350 primary total knee arthroplasty with selective patellar resurfacing with a mean follow up of 7.8 years demonstrated that satisfaction was higher in patients with a resurfaced patella. In patients followed for at least 10 years, no significant difference was found. No difference was found in KSS scores, survivorship and no complications of resurfacing were identified. The vast majority of patients with remaining patellar articular cartilage do very well with TKA regardless of patellar resurfacing. Knees with exposed bone on the patellar articular surface were excluded[63].

In view of the above, I would selectively resurface. If there is evidence of Grade III–IV osteoarthritic changes involving the patella then I would resurface it. Otherwise, I would perform circumferential electrocautery for the potential improved pain in the short term, accepting that it is temporary.

COMMENT: There is no right answer here, it is a personal preference – as long as you can back up your argument with evidence. Describing what you would do reveals to the examiner that you have thought about it and have formed an educated opinion. The examiner might not agree with you, but you have stated in the outset that it is controversial.

Patella baja

- Shortened patellar tendon, which is hard to evert
- Knee flexion is limited by patellar impingement on the tibia
- Seen most often following previous HTO, fracture of the proximal tibia, or tibial tubercle osteotomy
- Avoid cuts that raise the joint line
- Increases the difficulty of TKR

Managing patella baja

- Use a small patellar dome superiorly
- Trim anterior tibial and patellar polyethylene at the impingement points
- Lowering the joint line by cutting more off the proximal tibia and using distal femoral augmentation (rarely necessary)

Raising the joint line affects:

- PCL function
- Collateral ligaments tension
- Patellofemoral joint mechanics[64–66]

Valgus knee

- The normal tibiofemoral angle is 5–6°
- The normal knee mechanical axis is 1.2° varus
- The valgus knee can be defined as a tibiofemoral angle >10°
- Valgus knee is associated with bony and soft-tissue abnormality
- There are acquired or pre-existing bony deficiencies
- There is lateral subluxation of the patella
- There is lateral capsule and ligament contracture
- Elongated PCL may become dysfunctional in severe valgus
- There is distal femoral rotational deformity with externally rotated epicondylar axis up to 10°

Aetiology

- Mainly primary arthritis
- Inflammatory arthritis and osteonecrosis (small proportion)
- Post-traumatic arthritis (loss of lateral meniscus)
- Over-correction after HTO
- Childhood metabolic disorder (rickets)

Alignment

- No more than 5° femoral cut
- Component rotation is best achieved using the AP axis (Whiteside)
- Do not use additional 3° of external rotation, as the distal femur is externally rotated already
- Be careful not to internally rotate the femoral component by posterior referencing off a deficient lateral condyle

Approach and soft-tissue release

- The medial parapatellar approach gives good access to the whole knee and better soft-tissue cover (preferred approach)
- The lateral parapatellar is a direct approach. Theoretically it preserves the neurovascular supply to the extensor mechanism and enhances postoperative rehabilitation
- Make preoperative and intraoperative assessments of the deformity. If the deformity is passively correctable and the flexion–extension gaps are equal, then a lateral release is unnecessary
- There is no consensus regarding the sequence of soft-tissue release

Soft-tissue release in the valgus knee

- Osteophyte excision
- Lateral patellofemoral ligament release
- Release posterolateral capsule off the tibia
- Sacrifice PCL in moderate-severe valgus

Flexion and extension tightness

- Release (or pie-crust) lateral collateral ligament (LCL) from the femur

Extension tightness

1. Release (or pie-crust) the iliotibial band. A release would be performed at Gerdy's tubercle
2. Release popliteus (has a flexion component to it)

Flexion tightness

- Release posterolateral capsule off the tibia
- Cut PCL and recess posterior capsule
- If it remains tight, you rarely need to proceed to
  - Biceps femoris tendon – Z-lengthening
  - Detachment of lateral head of gastrocnemius

Complications

- Same as for varus knee
- There is a high risk of peroneal nerve stretching after severe valgus correction
- It is best to use a loose bandage postoperatively and to keep the knee in slight flexion

Examination corner

Adult orthopaedic and pathology oral 1

EXAMINER: Describe what you see (Figure 17.20).

CANDIDATE: This is an AP weight-bearing radiograph of bilateral lateral compartment degenerative change which are more significant on the left side. There are valgus deformities with loss of joint space in the lateral compartments. There are osteophytes in the lateral compartment, evidence of subchonrdal cysts and sclerotic margins all which are consistent with osteoarthritis of the lateral compartment of both knees, with severe changes on the left side.

EXAMINER: The patient is 76 years old with a history of hypertension and hypercholesterolaemia, which are controlled with medication. She is otherwise independent but her mobility has significantly been reduced due to continued pain in the left knee, which disrupts her sleep and affect her activities of daily living. What are you going to offer this lady?

CANDIDATE: I would take a detailed history.

EXAMINER: (Interrupts) done. What I told you is the history you will get from her. What are you going to do next?

CANDIDATE: I would like to examine her gait, do a full knee examination, including measuring the valgus angle with a goniometer as well as noting any evidence of fixed flexion deformity in the knee which is not uncommon in severe valgus OA. I will assess the integrity of the collateral ligaments to establish if she has an intact MCL and whether any of her valgus deformity is correctable. I would also examine her hip and ankle

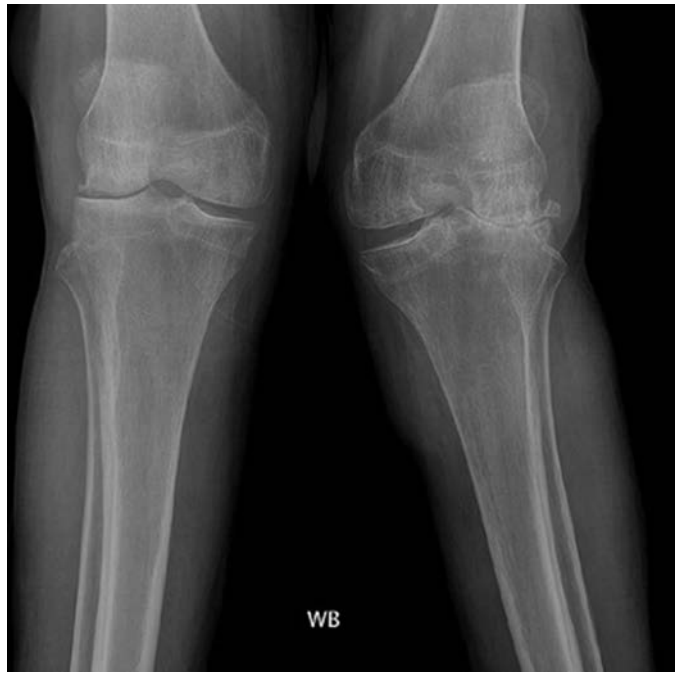

Figure 17.20 AP radiograph bilateral valgus knees

to check for any deformities or stiffness. I would then obtain a lateral and skyline radiograph of the knees and pelvis and hip x-ray if indicated. Long leg view would be very helpful to determine the mechanical axis and degree of valgus. Routine bloods including CRP, ESR, Hb and group and save as well as a CXR and ECG would be required if any surgical intervention is to be taken.

EXAMINER: Well she has tried all non-operative measures with her primary care physician and musculoskeletal physiotherapist and has come to you asking for an operation. What are you going to offer her? She has 10° of fixed flexion and 20° of valgus deformity.

CANDIDATE: I will offer her a total knee replacement.

EXAMINER: What approach would you use?

CANDIDATE: I would perform a medial parapatellar approach as this is the approach that I am most familiar with when performing at TKR. I am aware that some people would advocate the lateral parapatellar approach in cases of severe valgus deformity.

EXAMINER: Talk me through the approach and releases.

CANDIDATE: After the midline incision, medial parapatellar approach, excision of Hoffa's fat pad and everting the patella I would excise the osteophytes on both femur and tibia. I would then release the lateral patellofemoral ligament. I would perform a lateral and posterolateral capsular release from the tibia, while protecting the LCL and popliteus. I would routinely sacrifice PCL in moderate to severe valgus deformities. If the knee remains tight laterally in extension the I would pie crust the iliotibial band (ITB) or release it subperiosteally off Gerdy's tubercle. The next structure to release would be the popliteus (although it acts both in flexion and extension. However, if the knee remains tight the LCL will need to be released, usually subperiosteally off the tibia. I would also routinely recess the posterior capsule with extreme care if any fixed flexion remains. Rarely, which I have no experience with, one can perform a Z-lengthening to the biceps femoris or detach the lateral head of gastrocnemius in extreme case when the deformity is not corrected by conventional releases.

EXAMINER: Good. What are your thoughts preoperatively and what would you ask the theatre staff to prepare?

CANDIDATE: Given that this is a valgus knee with fixed flexion deformity, it will require soft-tissue releases to correct the alignment. As mentioned in my examination, I would want to assess the collaterals, specifically the integrity of the MCL. I would also want to assess whether these deformities are correctable or fixed. This will dictate the type of knee prosthesis required for this patient.

If the collaterals remain competent following both bone cuts and soft-tissue release to correct the deformity, then I would proceed with an unconstrained TKR. Given this is a valgus knee I would expect that there might be attenuation of the PCL and, therefore, a PCL-sacrificing prosthesis would be my preference.

If the MCL is too lax or the LCL had to be recessed/released during the procedure to achieve the necessary correction, then I would proceed to a more constrained knee prosthesis. This would be a non-hinged constrained prosthesis that provides AP and varus-valgus stability with the high central post as well as some rotary stability. This will compensate for the lack of collateral integrity. If the collaterals are completely deficient with global instability or bone deficiency (rarely the case), then I would consider a hinged prosthesis such as a rotating hinge. This has the disadvantage of increased rate of aseptic loosening secondary to the high level of constraint and the large forces acting on the prosthesis. The stems that used in both femoral and tibial side are present to increase stability and reduce the stress at the bone/cement interface. These implants also have a higher risk of periprosthetic fractures from stress risers below the stems.

Unicompartmental knee replacement (UKR)

It is important to understand that UKR is not 'half a total knee'[67]. It is a ligament-balancing procedure more than a realignment procedure and is not intended to correct an extra-articular deformity. The pattern of arthritis is usually anteromedial owing to ACL preservation. The intact ACL and the preserved posterior tibial plateau cartilage lead to stretching of the MCL every time the femur rolls back in flexion, preventing fixed varus deformity[68].

Advantages

- Avoids patellofemoral overload
- Retains knee kinematics
- Restores function and range of movement
- Rapid recovery: Three times faster than after TKR
- Less blood loss and, hence, transfusion
- Cost less than TKR (all factors considered)
- Quicker operation than TKR
- Quicker return to work than after TKR
- Lower infection rate (halved) compared with TKR
- Allows minimally invasive approach
- Easier to revise than HTO
- No patellar fractures or dislocations
- Maximizes the longevity of total knee arthroplasty
- Reduced incidence of DVT
- Reduced mortality from pulmonary embolism
- High flexion lifestyle

Prerequisites

- Intact ligaments (especially ACL and PCL)
- Correctable varus deformity
- $<10°$ FFD
- Flexion beyond 100°

- Preservation of the articular cartilage lateral compartment, as demonstrated on a valgus stress radiograph
- Clinically asymptomatic PF joint and contralateral compartment

Contraindications

- Inflammatory arthritis
- Sepsis
- Young age
- High level of activity

Relative contraindications

- ACL degeneration
- Chondrocalcinosis[69]
- Lateral meniscectomy
- Osteonecrosis
- Combined obesity and small bone size in some women

Principles

- Appropriate for 25% of osteoarthritic knees requiring arthroplasty
- Never release the MCL
- Polyethylene-bearing dislocation rate is 1/200 after medial compartment mobile bearing UKR
- Polyethylene bearing dislocation rate is up to 6% after lateral compartment mobile bearing UKR
- Dislocation rate can be reduced by using a fixed bearing UKR

Management options for medial compartment OA

- HTO suitable for high-demand, young patients
- UKA (better functional results, much better 10-year survival – 98% vs 66%)
- TKA

Examination corner

Adult and pathology 1 oral

EXAMINER: If you had a patient who has a UKA implanted in his knee 12 years ago with symptoms of gradual onset of pain, serial radiographs over the years with evidence of loosening which has been fully investigated and is aseptic, what implant would you chose to revise him to?

CANDIDATE: As long as the patient is fit and able to withstand a further procedure, and I am happy with the evidence that this is aseptic loosening, I would discuss the option of performing a single stage revision to a primary unconstrained total knee replacement. I would assess the potential bone loss preoperatively and intraoperatively. The tibial bone loss is usually more prominent and more concerning as this can dictate the level constrain needed. Although in most occasions a CR or PS TKR can be implanted, a studies have shown that the revision of UKA to TKA is a more complex procedure compared to primary TKA, with a higher incidence of using constrained implants (twice as likely in a revision vs primary TKA) and the use of thicker polyethylene inserts[70].

I would ensure that the theatre staff have constrained implants (non-hinged and hinged) as well as stems and augments available at the time of revision. I will also warn the patient of the slim possibility of converting to a two-stage revision, if unexpected intraoperative findings are faced.

Painful knee arthroplasty

Painful knee arthroplasty presents a major diagnostic challenge. In certain cases even a careful history-taking, meticulous clinical examination and numerous investigations may not help in reaching a diagnosis. Infection is particularly difficult to diagnose. To date there is no single preoperative investigation that can reliably diagnose an infection. Our inability to diagnose subclinical infection before revision surgery is still a major concern.

Possible causes of painful knee arthroplasty

- Infection
- Aseptic loosening
- Instability
- Stiffness
- Mal-rotation
- Mal-alignment
- Patellar pain or dislocation
- Extensor mechanism rupture
- Incompetent medial collateral ligament
- Periprosthetic fracture
- Implant breakage
- Complex regional pain syndrome
- Hip or spine pathology (referred pain)
- Unexplained pain (1/300)

Management

History

Date of index operation, postoperative pain relief/problems, wound leak, wound infection (and need for antibiotics), pain at rest, mechanical pain, stair climbing and descent, any injuries, medical problems, especially diabetes and rheumatoid arthritis.

Examination

Limp, walking aid, leg alignment, patellar alignment/tracking/tenderness, inflammation, effusion, quadriceps tone, CRPS (RSD) signs, joint tenderness localized/generalized, ROM active/passive, laxity in sagittal/coronal plane and finally assess the hip, spine and foot.

Investigations

Plain weight-bearing x-ray, bloods (including WCC, ESR and CRP – IL-6 (expensive) in specialist units), bone scan (not helpful until at least year after the index procedure), white cell-labelled bone scan, knee aspiration, fluoroscopic alignment check, CT scan to check rotation and long leg films to assess the overall alignment. SPECT bone scan and SPECT–CT has also been a novel imaging option to detect loosening/infection and highlight areas of maximal activity.

AAOS clinical guideline practice summary for diagnosis of periprosthetic joint infections of the knee[71]

In patients with suspected periprosthetic infection, the AAOS working group strongly recommended:

- Testing ESR and CRP
- Joint aspiration
- The use of intraoperative frozen sections
- Obtaining multiple intraoperative cultures (at least three but no more than six using different instrument for each sample and from different areas)
- Against initiating antibiotic treatment until after cultures
- Against the use of intraoperative Gram stain (as it is not helpful in ruling out infection)
- Nuclear imaging was weakly recommended as an option in patients in whom diagnosis of periprosthetic joint infection has not been established and who are not scheduled for re-operation

What is the definition of periprosthetic joint infection?

It has been defined by the workgroup on the Diagnosis of Periprosthetic Joint Infection at the Proceedings of the International Consensus Meeting on Periprosthetic Joint Infection in 2013 as:

- Two positive periprosthetic cultures with identical organisms

OR

- A sinus tract communicating with the joint

OR

- Three of the following minor criteria
  - Elevated CRP and ESR
  - Elevated synovial fluid WCC OR ++ change on leukocyte esterase test strip
  - Elevated synovial fluid PMN%
  - Positive histological analysis of periprosthetic tissue
  - Single positive culture

Examination corner

Basic science oral 1

EXAMINER: What is the material of the insert between the femoral and tibial component made of?

CANDIDATE: It is made of ultra-high-molecular-weight polyethylene (UHMWPE). It is a subset of thermoplastic polyethylene and has long hydrocarbon chains. These chains are bonded together by covalent bonds. The longer chain allows more effective load transfer to the polymer by strengthening intermolecular interactions. This leads to a very tough material with a high impact strength.

UHMWPE is highly resistant to corrosive chemicals, has a very low coefficient of friction and is highly resistant to abrasion.

It was first used clinically by John Charnley in 1962.

EXAMINER: How has the properties of the polyethylene been manipulated to improve its characteristics?

CANDIDATE: We now have highly cross-linked UHMWPE or XLPE. It is cross-linked by using gamma or electron beam radiation, which is then thermally processed to improve the material's oxidation resistance. This process is performed in an inert environment of vacuum or inert gas, to prevent oxidation. Antioxidants, such as vitamin E, have been infused into the XLPE in order to abolish the free radicals that are introduced during the irradiation process. The cross-linking of UHMWPE has reduced the rate of wear both in vivo and in vitro.

EXAMINER: Is highly cross-linked polyethylene currently used in clinical practice?

CANDIDATE: Yes, it is now commonly used as a bearing surface in total hip arthroplasty with positive results regarding less wear in comparison to conventional polyethylene, although showing similar amount of surface damage in retrieved acetabular liners[72]. However, this is not yet the case in knee arthroplasty although many studies are looking into that.

EXAMINER: What are the disadvantages of XLPE?

CANDIDATE: It is more brittle than conventional poly and, therefore, has a higher risk of fracture. It is also two to four times more expensive. A study from HSS showed that although material properties of XLPE reduce adhesive and abrasive wear, it does not reduce the risk of crack propagation, deformation, pitting and delamination found in TKR. Given that wear-induced osteolysis in TKR has not been found to be a major cause of failure at long-term follow-up and that mid-term follow-up studies show no difference in outcome measures between conventional PE and XLPE in knees, they currently cannot recommend the use. Conventional compression-molded polyethylene with its outstanding long-term results should remain the material of choice in TKR[73].

Polyethylene wear

The rate of polyethylene wear and osteolysis is determined by several factors:

- Patient factors: Age, size and activity level
- Surgical factors: Alignment, rotation, cementing, balancing
- Implant factors
  - Polyethylene thickness
  - Material, property and polymerisation

- Manufacturing method: Compression moulding preferred to machined component
- Sterilisation method: Avoiding gamma radiation in air
- Cross-linking: Moderately/highly cross-linked polyethylene – May offer improved resistance in the knee
- And whether the implant vacuum pack is still in date and not expired which would increase the presence of free radicals

Examination corner

Adult and pathology oral 1

EXAMINER: A patient comes in to your elective clinic as an add-on, as he was feeling generally unwell, and coming to see you because his knee has become suddenly more painful, swollen and red. He is an 84-year-old man with no significant medical history except a total knee replacement 3 years ago. What would you like to ask this patient?

CANDIDATE: I would initially like to establish the reason for total knee replacement and if possible find out where it was performed and obtain the operative details. I would ask him if there were any wound problems or evidence of infection during the postoperative period, such as a superficial wound infection that required antibiotics, or any more serious infections that required admission to hospital and IV antibiotic therapy. I would then want to establish more about his current state and medical background, if he is on any immunosuppressive therapy, or if he has had any trauma or recent infective processes that he is aware of. I would then move on to investigate if there has been any changes in regards to how the knee feels to him, and if there is any restriction in its movement.

EXAMINER: Good. What are you concerned about?

CANDIDATE: A prosthetic joint infection.

EXAMINER: What are your initial investigations, specifically your blood markers?

CANDIDATE: I would request an AP, lateral and skyline radiograph of the knee, looking for any signs of loosening, fracture, and compare it with the last postoperative radiograph available. In regards to blood tests, I would request a FBC – Looking at the WCC and differential as well as signs of anaemia for the possibility of anaemia of chronic infection. I would request a CRP, an ESR and, if I am in a specialist unit, IL-6, which is very sensitive and specific.

EXAMINER INTERRUPTS: So you mention sensitivity and specificity. Please tell me what are the sensitivity and specificity of the blood test that you requested, as you will need to know how much you can rely on the results of the tests that you have requested?

CANDIDATE: CRP has a sensitivity of 88% and a specificity of 74%, ESR has a sensitivity of 75% and a specificity of 70% and IL-6 has a sensitivity of 97% and a specificity of 91% according to a study by Berbari in *JBJS(AM)* in 2010[74]. These tests are not diagnostic independently.

EXAMINER: How would you go about confirming your diagnosis prior to any surgical intervention?

CANDIDATE: I would perform a joint aspiration in a sterile environment. I prefer doing this in theatre. The technique that I use is aspiration of the joint through a 3 mm stab incision of the dermis with a scalpel under local anaesthetic, so avoid any skin commensals appearing in my sample. I would obtain as much fluid as possible with a large bore needle and insert them directly into blood culture bottles as well as sending it in a microbiology specimen pot for urgent gram stain as well as well microbiology and culture including sensitivity and synovial white blood cell count and differential (synovial PMN $>$ 90% is positive for acute infection and $>$80% for chronic as per the Consensus Meeting on Periprosthetic Joint Infections).

C-reactive protein concentration, leukocyte esterase concentration in the joint fluid aspirate, and other molecular markers of periprosthetic joint infection are currently under investigation.

EXAMINER: If you were to embark into operative treatment what are your options and what is your preferred method?

CANDIDATE: The main principles are to eradicate infection and eventually implant a new prosthesis in a clean environment to restore knee function that is pain free. The two-stage exchange arthroplasty is currently the most accepted procedure for the treatment of periprosthetic joint infection. Two-stage revision involves resection of the infected implants, thorough debridement and irrigation, placement of a temporary antibiotic-impregnated cement spacer (static or articulating), and delayed component reimplantation once the infection is eradicated after a period of intravenous antibiotic. One-stage revisions are increasing in popularity in certain specialist centres. Its proponents focus on its advantages over two-stage procedures in it being a single operation for the patient, with decreased morbidity, lower cost and some argue that it provides improved functional results[75]. Strict criteria are usually applied in centres who perform single-stage revisions including: A healthy patient, infection being acute and postoperative, known organism and adequate soft-tissue coverage.

I would personally offer the patient a two-stage revision. I would support my decision with a recent systematic review that demonstrated an average success rate of 90% for two-stage revision in infected total knee replacements[76]. The study also reported that two-stage revision provided better outcomes than one-stage revision.

Knee arthrodesis (Figure 17.21 a and b)

Indications

- Failed knee replacement
- Uncontrollable sepsis
- Neuropathic joint
- Young patient with severe articular joint disease and ligamentous damage

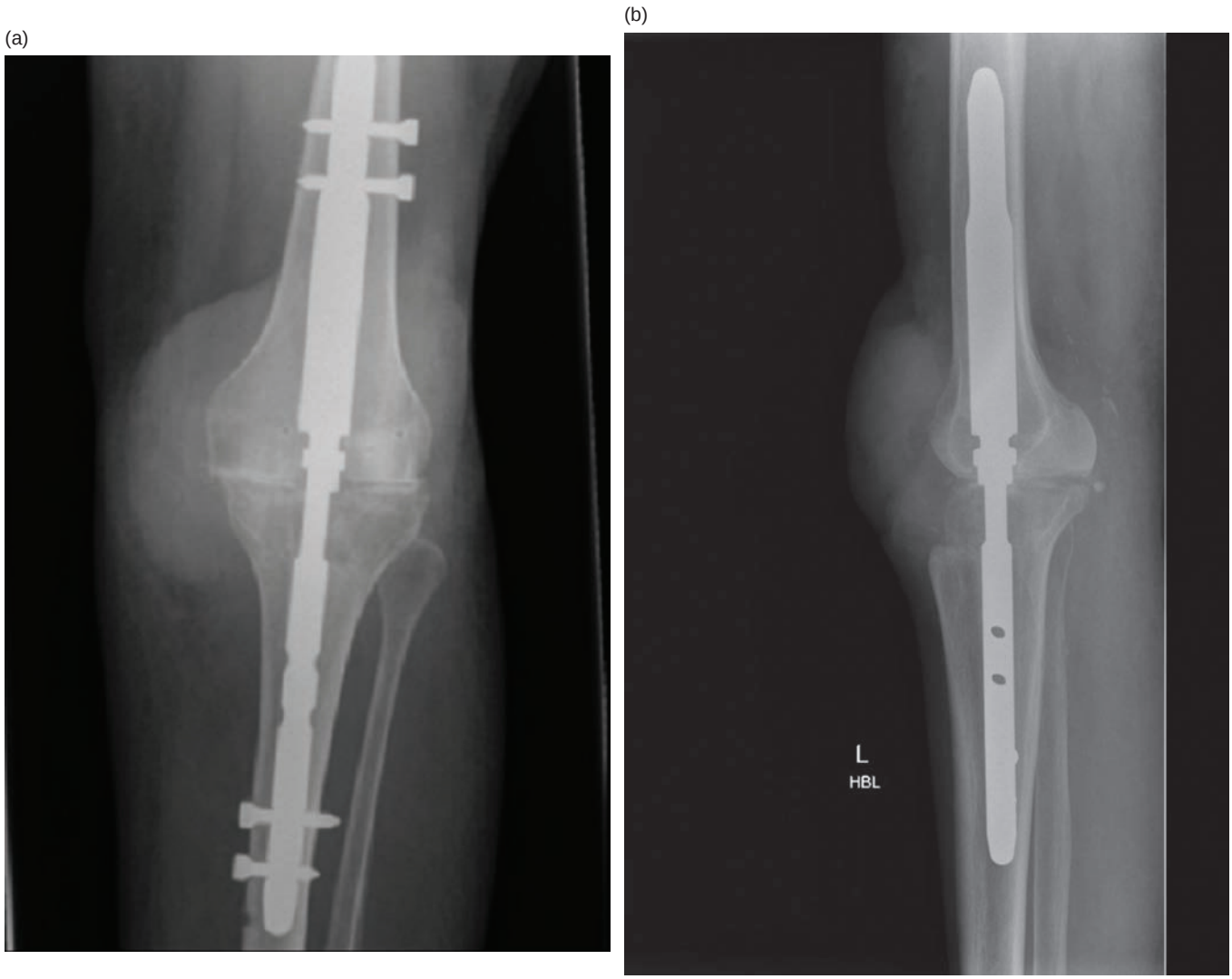

Figure 17.21 Knee arthrodesis (a, b: AP and lateral radiographs) secondary to loss of extensor mechanism following a severe 3% full thickness burn over her knee joint with concurrent infection and partial neuropathy in a diabetic 79-year-old woman. This was done using a Whichita Fusion Nail® and the patient achieved full union with very good function and resumed independence[77]

- Disruption of extensor mechanism
- Poor soft-tissue envelope
- Systemically immunocompromised
- Resistant microorganisms
- Post-traumatic arthrosis in a heavy manual labourer

Contraindications

- Bilateral knee disease
- Ipsilateral ankle or hip disease
- Ipsilateral hip arthrodesis
- Severe segmental bone loss
- Contralateral limb amputation

Optimal position for knee fusion

- 7–10° of external rotation
- Slight valgus
- 10–20° of flexion
- The above may be easier to achieve with external fixator rather than IM nail

Techniques

1. Intramedullary arthrodesis
    - Long custom-made nail through piriform fossa
    - Intramedullary fixation with modular and non-modular nails
    - Linked nail through the knee is easier to apply
    - More reliable in achieving union than external fixation
    - Technically difficult
    - Forty per cent complication rate (nail breakage and migration)
    - May cause widespread osteomyelitis in the tibia and femoral shaft

- The nail has to be inserted after removing implants and infection treatment

2. External fixation
   - Conventional or circular frames
   - Allows for arthrodesis in the presence of infection
   - Can be applied at the time of implant removal
   - A 20–60% complication rate (neurovascular injury, pin site infection and fracture through pin site)
3. Plate fixation
   - Main drawback is recurrent or new infection
   - Single anterior or dual plating

Complications

- Non-union
- Mal-union
- Delayed union
- Recurrent infection

Benign synovial disorders

- Pigmented villonodular synovitis
- Synovial chondromatosis
- Synovial haemangioma
- Lipoma arborescens

Examination corner

Adult orthopaedic and pathology oral

EXAMINER: What is the diagnosis? (Figure 17.22)

CANDIDATE: This is a weight-bearing lateral knee radiograph that reveals multiple small, well-defined, juxta-articular mineralized nodules of similar size. This is likely to be synovial chondromatosis.

EXAMINER: So what is synovial chondromatosis?

CANDIDATE: It is a benign mono-articular disorder of unknown origin that is characterized by multiple intra-articular cartilaginous metaplasia forming loose bodies, not all of which are ossified. Seventy per cent of cases are found in the knee followed by twenty per cent in the hip. It most commonly affects patients in the fourth and fifth decade and men are more commonly affected than women.

EXAMINER: Agreed, arthroscopy may not be appropriate here. Any other risks that you would like to mention to the patient in regards to this condition?

CANDIDATE: This is a benign condition, but I will be sending the samples for histological confirmation of the radiological and macroscopic diagnosis. There is a very small chance of malignant degeneration into synovial chonrdosarcoma but this is rare.

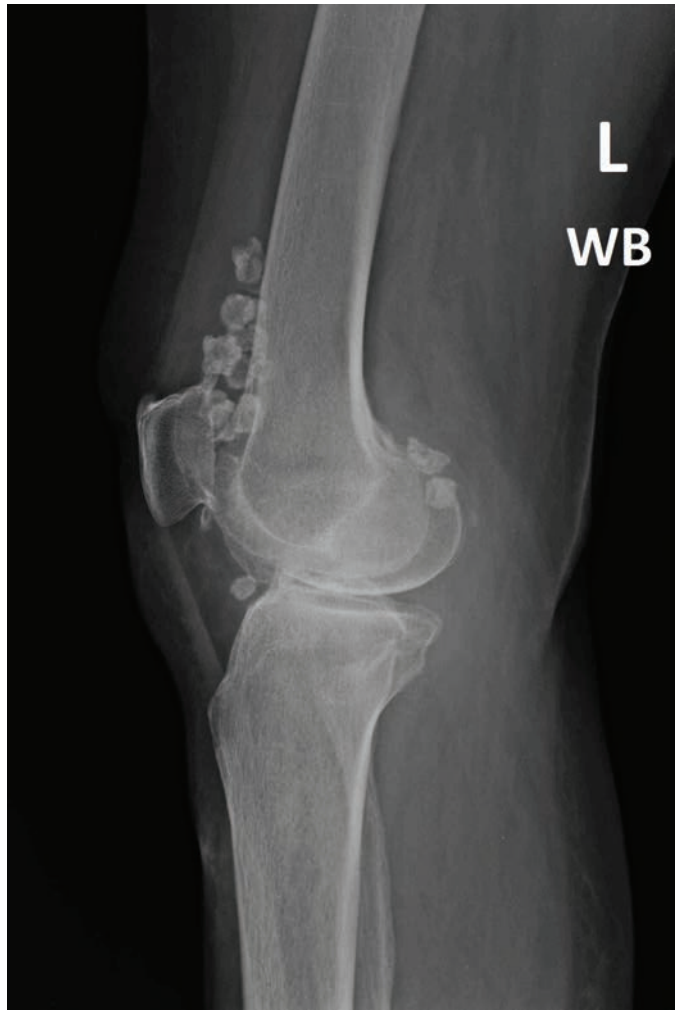

Figure 17.22 Lateral knee radiograph with synovial chondromatosis

Pigmented villonodular synovitis (PVNS)

PVNS is a benign proliferative condition of the synovial membrane and tendon sheath. Knee PVNS is usually monoarticular and affects young adults. It is characterized by synovial inflammation and haemosiderin deposits.

- Aetiology: Unknown, possible trauma, neoplastic process, chronic inflammation
- Incidence $<1/500\ 000$
- Usually painless knee swelling, but it may present with catching, multiple nodules, locking or instability
- Two main types: Diffuse and localized
- Diagnosis is mainly by MRI scan (Figure 17.23) and biopsy. Joint aspirate colour and cytology can be helpful
- The diffuse type is commoner and harder to eradicate
- Bone erosion, subchondral cysts and joint space narrowing may be seen in PVNS
- Treatment aims to eradicate all abnormal synovial tissue
- A combination of non-surgical and surgical intervention may be necessary

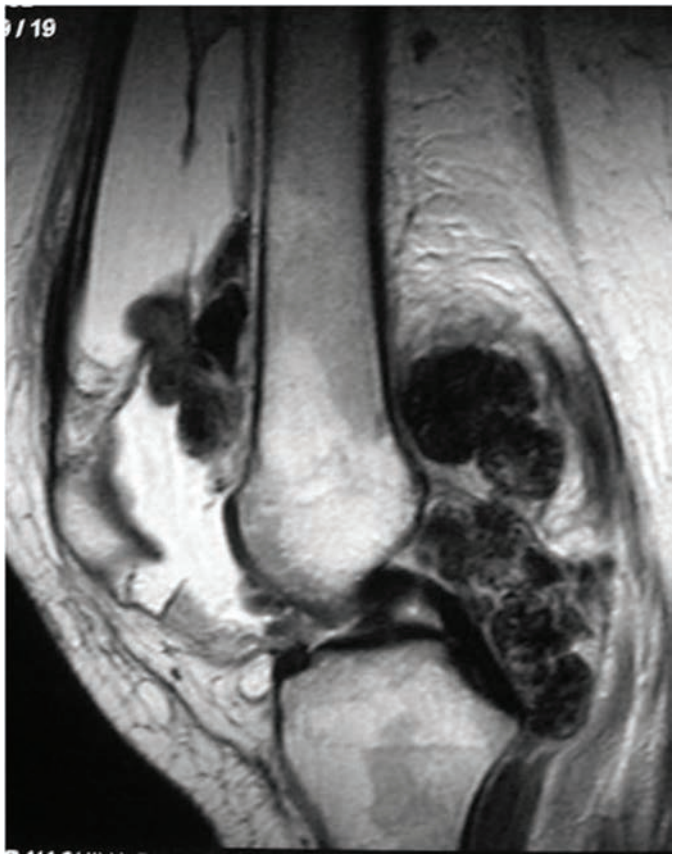

Figure 17.23 Multiple PVNS lesions shown on the T2-weighted MRI with low signal intensity in the posterior and anterior aspect of the knee with an effusion present

- Radiation therapy: External beam therapy is now less popular than intra-articular radioisotope yttrium-90 injection, which has beta emission only, causing less body irradiation
- Surgical treatment
  - Arthroscopic synovectomy has better functional results than open surgery, but higher recurrence rates. However, it can offer good access to the posterior compartment if performed by experienced arthroscopists. Multiple portals can be useful in these cases including a posteromedial and suprapatellar portal. It is best reserved for well-assessed patients (MRI/biopsy), less extensive disease with only intra-articular involvement
  - Open synovectomy is usually through combined anterior and posterior approaches. It is associated with a high rate of postoperative stiffness and slow rehabilitation
- Treatment plan
  - Localized PVNS and less extensive disease: Arthroscopic synovectomy alone can be sufficient
  - Diffuse PVNS involving the popliteal compartment: Arthroscopic synovectomy through anterior and posteromedial portals followed by yttrium-90 (6 weeks post-operation)
  - Recurrence (20–50%) can be treated by open radical synovectomy
  - Secondary arthritis is best treated with knee arthroplasty

Synovial chondromatosis (osteochondromatosis)

- This is a benign metaplastic disorder of synovial membrane, characterized by the formation of multiple cartilaginous nodules or osseous loose bodies inside the articular space. It typically affects adult men twice as often as women
- Patients present with pain, swelling, effusion, locking and limited motion
- Clinical examination reveals diffuse tenderness, crepitus, palpable nodules or loose bodies
- Can be localized or generalized
- X-ray shows calcified intra-articular loose bodies
- MRI shows synovial proliferation and calcified nodule or a large intra-articular soft tissue
- Synovial chondromatosis is generally a primary condition, but it may occur secondary to osteoarthritis
- In primary synovial chondromatosis the loose bodies are typically numerous, small, round and uniform in size
- In secondary synovial chondromatosis the loose bodies are few and variable in size
- Treatment is by arthroscopic removal of loose bodies alone or with synovectomy
- Recurrence after open/arthroscopic treatment ranges from 3% to 30%
- Recurrence can occur even after synovectomy

Knee disarticulation (through knee amputation)

Any indication such as infection, ischemia or traumatic
An alternative to ultrashort transtibial stump
Important alternative to transfemoral amputation
New indications is infected and loose knee arthroplasty

Advantages

- Superior compared to a transfemoral stump
- Thigh muscles are all preserved
- Preservation of adductor muscle insertion
- Hip motion is not limited
- Bilateral knee disarticulation can walk 'barefoot'
- Enhanced proprioception
- A long lever arm when sitting
- Decreased metabolic cost of ambulation

Nail–patella syndrome

Autosomal dominant genetic disorder Chromosome 9
Lean body build
Patella affected in 90% of patients, patellar aplasia in 20% (Figure 17.24)
Elbows: Limited pronation, supination, extension
Subluxation of the radial head may occur
General hyperextension of the joints can be present
Exostoses ('iliac horns') 80% of patients

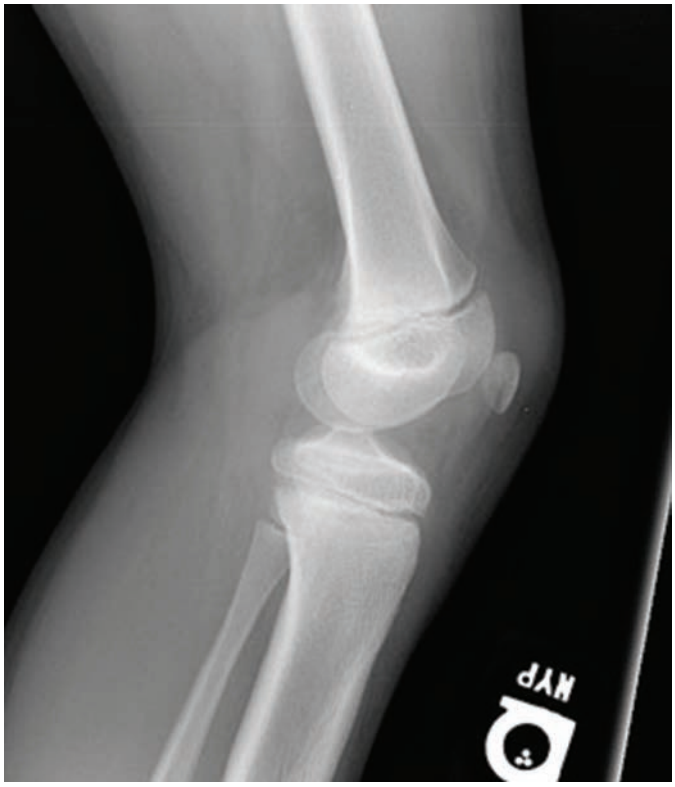

Figure 17.24 Lateral knee radiograph demonstrating patellar hypoplasia

Kidney failure and teeth weakness
The treatment of nail patella syndrome is mainly supportive with the majority of patients being asymptomatic.

Knee outcome measures

- **SF36** is the most popular general health outcome measure
- **WOMAC** – Western and Ontario McMaster University Osteoarthritis Index: Most commonly used condition-specific outcome measure for osteoarthritis owing to its efficacy and sensitivity to change
- **KOOS**: Developed to evaluate sports-related injuries such as ACLs and meniscal injuries
- **IKDC** – International Knee Documentation Committee, 1993: Joint-specific tool to evaluate symptoms, function and sporting activity
- **Lysholm Score, 1982**: Developed to evaluate knee ligament surgery
- **Cincinnati Knee Rating Scale, 1983**: Assesses subjective symptoms and functional activity level
- **Tegner activity level, 1985**: Designed to lend numeric scores to patient activity level. It is sport-specific; hence, it may limit its applicability cross cultures
- **MARX Activity Level Scale**: This is a functional activity questionnaire rather than a sport-specific questionnaire. Its strength lies in its measure of function rather than sport activity
- **Oxford Knee Score**: Designed specifically for patients undergoing knee replacements. It is joint-specific with good evidence of reliability of content and validity of construct[78]. It has also been shown that the Oxford Knee Score at 6 months is a useful predictor of early revision after TKRs. Scores <27 are associated with a risk of revision within 2 years[79]

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Chapter 18

Foot and ankle oral core topics

Kailash Devalia and Jane Madeley

Introduction

There is a large recommended syllabus from the British Orthopaedic Foot and Ankle Society for the FRCS (Tr & Orth) examination. This syllabus is very detailed and comprehensive. Whilst a candidate may not be expected to know all the details of every condition, he or she should at least be prepared to answer questions on most conditions and in particular on the more common foot and ankle disorders.

Most foot/ankle conditions are likely to be encountered in the short cases, although an intermediate case should not come as surprise. As with all cases, appropriate history and slick, targeted examination, go a long way to securing a pass even if you only know a little on managements available.

The BOFAS Educational Comittess hold courses aimed for trainees nearing the FRCS (Tr & Orth) exam. The emphasis is on clinical examination cases and day to day clinical scenarios. The content is taught to the standard of the FRCS (Tr & Orth) exam. The course has grown in complexity and can be up to 3 days in length even involving cadaveric workshops. The course has had excellent feedback and comes highly recommended.

Common cases

Several foot and ankle cases are known as to be examiner favourites as they are relatively common conditions and are easily available to recruit for the exam. These cases include:

1. Hallux valgus
2. Hallux rigidus
3. Rheumatoid foot
4. Pes cavus
5. Residual CTEV
6. Pes planus
7. Tarsal coalitions

These conditions are also relatively common oral topics for the adult pathology or paediatric orals. Good knowledge, stepwise examination technique, going through set answers for particular topics and being aware of where you can end up digging a hole for yourself, can compensate for a practical weakness in the subject.

Ankle arthroscopy[1]

Arthroscopes (1.9, 2.3 and 2.7 mm diameters, 30 and 70° scopes).
Non-invasive distraction technique.

Contraindications

Absolute: localized soft-tissue or systemic infection, severe rigid end-stage degenerative joint disease
Relative: moderate degenerative joint disease, severe oedema, RSD and suspect vascular supply.

Indications

Diagnostic

When MRI findings are in doubt

Therapeutic

Anterior and anterolateral ankle impingement

- Anterolateral synovitis lesion following plantar flexion and inversion injury
- Anterior ankle impingement with mild degenerative changes
- Effective outcome with both open and arthroscopic surgery (86% satisfaction rate)
- With arthroscopy: quicker recovery, reduced length of stay and smaller incisions

Osteochondral lesions

- Arthroscopic debridement associated with any technique of subchondral bone penetration (curettage, drilling or microfracture) in lesions <15 mm in diameter has fair evidence (level II and III studies) recommending intervention
- Evidence regarding lesions >15 mm is insufficient to make recommendations

Ankle arthrodesis

- Equivalent fusion rates to open arthrodesis
- Significantly quicker times to union, decreased pain, shorter hospital stay, faster rehabilitation and mobilization and a lower complication rate

- Arthroscopic ankle arthrodesis in ankles with coronal plane deformity <15° has fair evidence to support this mode of intervention (level II and III studies)
- Insufficient evidence for arthroscopic fusion in ankles with >15° of varus/valgus deformity
- Risk of non-union

Septic arthritis

- Effective intervention with cure rates over 90%, conversion to open washout if failure to improve within 2 days of arthroscopic irrigation

Arthroscopic landmarks

- A thorough knowledge of ankle anatomy is needed to avoid potential complications
- Before performing ankle arthroscopy it is important to mark out in pen on the surface of the ankle potential structures at risk: Dorsalis pedis artery, deep peroneal nerve, great saphenous nerve, anterior tibial tendon, peroneus tertius tendon and superficial peroneal nerve and branches
- The joint line is identified by inverting and plantarflexing the ankle

Joint Distraction

- Good distraction of the joint is necessary for adequate visualisation
- Skin traction with heel and foot strap. Avoid superficial midfoot placement of the foot strap, less soft tissue protection underlying nerves.
- Body traction: Lopped around surgeon. The surgeon applies more or less traction by leaning forwards or backwards. Simple, cheap and effective

Joint distension

Usually with 30 mm of normal saline

Portals

Five portals have been developed for use

Anterior scope

Patient is supine

For anterolateral impingements, most osteochondral lesions and arthroscopic ankle arthrodesis

Anteromedial

- Primary viewing port. Least dangerous port. Established first
- Just medial to the tibialis anterior tendon and lateral to the saphenous nerve and vein directly at level of joint line. Incise skin, bluntly dissect soft tissues and joint capsule
- Risks: Damage to tibialis anterior tendon, saphenous nerve and vein

Anterolateral

- Lateral to peroneus tertius tendon and extensors
- Working port. Created once arthroscopy is introduced
- Risks: Damage to superficial peroneal nerve
- Preoperative mark the SPN branch by plantar flexion of forth toe. Most commonly injured nerve in ankle arthroscopy

Anterocentral

- Rarely used as considered dangerous
- Medial or lateral to EDL tendon
- Risks: Damage to anterior tibial artery and deep peroneal nerve; to EHL and EDL; to terminal branches of the superficial peroneal nerve

Posterior scope

Patient is prone.

For posterior impingements, Os trigonum, Haglund's excision and, rarely, osteochondral lesions

Posterolateral

- Just lateral to the edge of the Achilles tendon, at the level of the tip of the lateral malleolus. The portal is usually at or slightly below the joint line
- Most commonly used and safest portal
- Risks: Damage to short saphenous vein and sural nerve

Posteromedial

- Rarely used
- Medial to Achilles tendon at the level of the tip of the lateral malleolus
- Enter joint capsule under direct vision from the posterolateral port
- Risks: FHL, posterior tibial vessels and nerve

Complications

Studies have reported variable complication rates, ranging from 7.6–17% including:

- Nerve injury (SPN most common)
- Infection
- Painful scars
- Synovial fistula
- Articular cartilage damage
- Complex regional pain syndrome
- Prolonged portal drainage

Rheumatoid foot

Introduction

The two predominant symptoms of the rheumatoid foot are pain and deformity. Approximately 15% of rheumatoid patients present initially with foot symptoms. Eventually 70–90% of those with long-standing rheumatoid arthritis have

foot involvement. The disease starts in the forefoot and, with time, advances to involve the hindfoot.

Practice describing the typical rheumatoid deformities using clinical photographs as props – You have 30 seconds to 1 minute maximum real time for this in the exam. This is more helpful for a viva question but is also useful for a clinical case.

Deformities

A. Forefoot – very commonly involved
   1. Hallux valgus is often present but rarely problematic
   2. First MTP joint erosive arthritis, sometimes associated with IP joint arthritis
   3. MTP joints develop dorsal subluxations/dislocations (synovitis of the MTP joints, weakening and stretching of the capsule and collateral ligaments, planar plate laxity/rupture) with the cushioning fat pads pulled forwards with atrophy (metatarsalgia and forming keratosis)
   4. The MT heads become prominent around the sole of the foot predisposing to pain, callosities and skin breakdown
   5. Hammer toe and claw toe deformities of the lesser toes (intrinsic muscle contracture)

B. Hindfoot – less commonly involved and less severe
   1. Valgus ankle, which often presents late with pain and instability
   2. Valgus hindfoot with synovitis and arthritis in the subtalar joint
   3. Talonavicular joint subluxation/dislocation causing flattening of medial arch and forefoot pronation
   4. Subfibular impingement

C. Soft tissues
   1. Tenosynovitis of the tibialis posterior and peroneal tendons presents mainly with swelling of tendons both medially and laterally around the ankle
   2. Collapse of the medial longitudinal arch of the foot occurs due to rupture or weakening of the tibialis posterior tendon and gradual disruption of the talocalcaneal interosseous ligament
   3. Distal migration and atrophy of the forefoot fat pad
   4. Prominent plantar metatarsal heads leading to plantar callosities
   5. Painful bursitis between the metatarsal heads
   6. Tarsal tunnel syndrome (valgus hindfoot)
   7. Morton's neuroma
   8. Retrocalcaneal bursitis

Clinical examination

- Proximal to distal
- Assess the hip and knee before foot surgery. This might need addressing first prior to foot surgery
- Assess the skin condition for risk of infection and wound healing
- Assess the vascular status of the foot
- Make a careful neurological assessment as there may be a neuropathic component
- Look for tendinopathy or ruptured tendons
- Assess whether the primary deformity is in the hindfoot or forefoot
- Determine which joint is causing pain; this is not always easy and may need a diagnostic injection
- Take a drug history, in particular steroid therapy and methotrexate, anti-TNF-α agents

Management

Multidisciplinary approach. Optimisation of medical treatment with involvement rheumatologists.

Conservative

- Special shoes/footwear
- Accommodating orthoses
- Steroid injections
- Immune-mediating drugs (TNF inhibitors)

Surgery (see Examination corner)

Goals of surgery are a stable pain-free plantar grade foot. There is a move towards joint preservation surgery.

If both the forefoot and hindfoot are involved, care is needed to decide which to operate on first. If the hindfoot is correctable, go for forefoot surgery first. If the hindfoot is severely deformed and rigid, it is often necessary to correct this first.

Forefoot

- Standard technique for forefoot correction in rheumatoid foot: MTPJ1 arthrodesis(10° dorsiflexion/10° valgus) with MT2–4 head resections (Fowler's procedure) and realignment of lesser toes deformities
- Synovectomy useful when severe synovitis present but no MTPJ subluxation/dislocation and no metatarsalgia
- Weil's shortening osteotomy of MT heads is an alternative to MT heads resection if no severe arthritic changes present in joint. This allows reduction of the MTP joints and return of the fat pad to the sole of the foot
- Stainsby procedure is where proximal phalanx base is resected with release of plantar plate and extensor tenodesis to flexors. This is an alternative to MT heads resection or shortening
- Mechanical offloading of MTPJ reduces synovits (through MT heads resection/MT shortening)
- If associated hallux valgus is present with lesser toes deformities, then MT head resections would accentuate the hallux valgus and MT-shortening osteotomies may be a better option

- Hallux valgus with no arthritic changes: better to perform modified Lapidus rather than standard MT osteotomy and soft-tissue correction
- If hallux valgus present with MTPJ1 arthritis, MTPJ arthrodesis is a better alternative
- Main complications with forefoot reconstruction is metatarsalgia and recurrence of deformity
- Althogh silastic joint arthroplasty could be a good option for the hallux, in rheumatoids results are not promising because of the risk of erosive synovitis and implant failure
- Lesser toe amputation could be an alternative with a severe isolated toe deformity
- Associated osteopenia justifies the use of stronger fixation with locking plates for arthrodesis

Hindfoot

- Arthrodesis is the main stay for correction of rheumatoid hindfoot and ankle deformity
- Try to avoid a single-joint arthrodesis (e.g. triple arthrodesis better than isolated talo-navicular arthrodesis)
- The most important aspects of hindfoot correction are alignment and stability
- Tibiotalocalcaneal arthrodesis is preferred over pantalar arthrodesis
- Total ankle replacement is successful; however, it is associated with increased incidence of subsidence, fracture and mal-alignment

Examination corner

Adult pathology oral 1
Radiograph of rheumatoid foot shown (Figure 18.1)

Describe how rheumatoid arthritis affects the foot?

Early radiographic features are soft-tissue swelling, widening of joint spaces and osteopenia. Later on marginal erosions involving the MTP joints, subchondral cysts whilst with advanced disease subluxation, dislocation, deformity and ankylosis are seen.

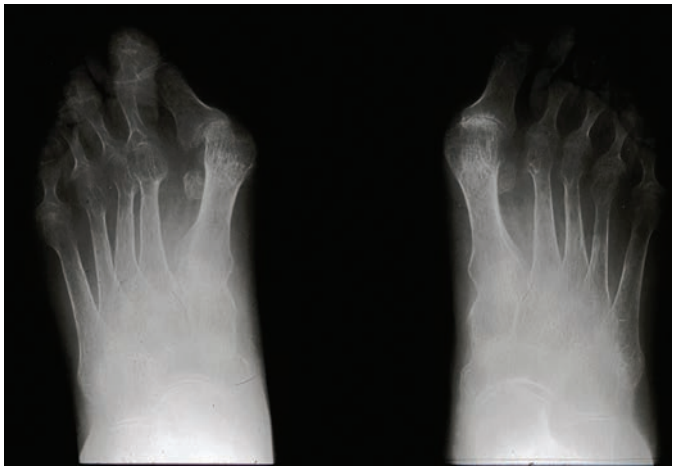

Figure 18.1 AP radiograph of rheumatoid foot

Adult and pathology oral 2: radiographs of a rheumatoid foot with dislocated MTP joints

Discussion about surgical management.

Pobble amputation. This operation involves amputation of all the lesser toes at the MTP joints. It is used for severe pain and deformity. 'I have been shown an old radiograph of a rheumatoid foot with the lesser toes missing and asked to comment on what operation was performed.'

Adult and pathology oral 3: a strange question

EXAMINER: A 53-year-old lady attends your orthopaedic clinic complaining of a painful and swollen second left MTP joint. The GP has mentioned a possibility of rheumatoid arthritis. How will you confirm the diagnosis?

CANDIDATE: I would take a history from the patient, find out how long she has had symptoms in the toe.

EXAMINER: Several months.

CANDIDATE: I would measure ESR, CRP and rheumatoid factor.

EXAMINER: All normal.

CANDIDATE: I am not sure I would do anything at this stage. Possibly I would send her for an x-ray of the foot.

EXAMINER: The point here is that you may want to refer her on to a rheumatology colleague for a second opinion. Reviewing her in 6 months is also a reasonable option – She won't come to any harm. The radiograph shows proximal resorption of the proximal phalanx of this second toe.

CANDIDATE: This can occur in rheumatoid disease.

EXAMINER: Are you just going to accept this?

CANDIDATE: There are other causes for this resorption such as gout and I would measure her serum urate level.

EXAMINER: Would you not want to biopsy the toe?

CANDIDATE: I would not want to jump in at this stage and biopsy without further information. *(I was struggling a bit here and wasn't sure what he was getting at.)*

EXAMINER: The point here is that other rare conditions can cause this appearance, e.g. PVNS. Let's move on to something else.

Adult and pathology oral 4: Clinical photograph of rheumatoid forefoot

Splayed foot
Hallux valgus with pronation and underriding, lesser toe hammering. Vasculitic lesions on shin. We discussed orthotic shoes, then Fowler's excision arthroplasty

Adult and pathology oral 4
Clinical picture shown of severe rheumatoid disease

EXAMINER: How would you treat the patient?

CANDIDATE: I would take a full history and enquire about mobility and functional limitations. It is important to use a

multidisciplinary approach involving rheumatologists, physicians, occupational therapists and orthopaedic surgeons.

A pyramid treatment approach is used starting with non-steroidal anti-inflammatory agents, progressing to steroids and may include cytotoxic drugs, e.g. methotrexate, or 'biologicals' (drugs which modify the immune system by blocking cytokines or their receptors, such as IL-1 or TNF-α). There may be a role for conservative management such as custom-made orthosis to accommodate the deformity, padded heels or foot/ankle orthotics. A patient may have considerable deformity in their feet but manage well with conservative treatment. Attempts should be made to minimize the doses of steroids used preoperatively. It is advisable to temporarily discontinue biological drugs before surgery, but continuation of methotrexate is thought to be safe.

Grennan et al.[2] showed no increased risk of infection or wound complications if taking methotrexate, but stopping the medication prior to surgery did lead to a flare up in rheumatoid disease in about 5% of patients.

I would offer her ankle arthrodesis as this has been shown to be the gold standard management option for ankle involvement in rheumatoid arthritis. Coronal and sagittal plane deformities can be corrected. The ankle is fused in neutral flexion, with 5° of hindfoot valgus and external rotation to match the contra-lateral leg.

EXAMINER: What are the complications of ankle arthrodesis?

CANDIDATE: Complications include infection, delayed or non-union (10%), neurovascular injury, wound healing problems and mal-alignment. Longer term there is the potential to develop degenerative changes in adjacent joints, reported as between 1% and 60%.

EXAMINER: What technique would you use?

CANDIDATE: Various techniques have been used including cannulated screws, plate fixation, retrograde nail, and external fixation. I would prefer arthroscopic arthrodesis using screws unless the deformity was very severe with poor bone stock.

EXAMINER: What about ankle arthroplasty?

CANDIDATE: Ankle arthroplasy is a good choice in rheumatoid patients as they have low functional demands and have other joints affected. There are a number of complications that can occur with ankle arthroplasty.

EXAMINER: Such as?

CANDIDATE: Infection, wound breakdown, aseptic loosening, mal-alignment, stiffness and impingement. There are worries about rates of osteolysis and revision for TAR although movement is allowed which allows walking with reduced stresses on adjacent joints.

EXAMINER: What are the results like for ankle arthroplasty?

CANDIDATE: The STAR prosthesis has a 10-year survival of 80.3% and is one of the most widely used prostheses.

EXAMINER: You seem mixed up. What are you going to offer this patient?

CANDIDATE: There is not enough evidence in the literature to give firm guidance as to the preferred option. Haddad et al.[3].

performed a systematic review of the literature up until 2005 comparing outcomes following second-generation TAR and AA. They concluded that both techniques gave comparable intermediate and long-term outcomes, reporting good or excellent outcomes in 68.5% of TAR and 67% of AA patients and mean AOFAS scores of 78.2 following TAR and 75.6 following AA. However, the majority of the 49 studies reviewed were single-centre case series and none directly compared the two treatments. A systematic review by Jordan et al.[4] identified four studies comparing TAR with AA in which two studies reported a significant improvement in functional outcome in favour of TAR but also a higher complication rate. All of these studies, however, demonstrated a lack of high quality evidence with methodological flaws and weaknesses.

Ankle arthritis

Symptoms

- Pain
- Stiffness
- Deformity
- Perceived or actual instability
- Limitation of ADL

Causes

Primary osteoarthritis of the ankle is rare. It is usually secondary to another predisposing cause. Look for features of a secondary cause on any radiograph studied[a].

- Post-traumatic arthritis: rising incidence especially in younger age group. Displaced ankle fractures, distal tibial pilon fractures, talar neck and body fractures with AVN, chronic ankle ligamentous instability
- Osteochondritis dissecans of the talus
- Inflammatory arthritis: Rheumatoid arthritis
- Others: Charcot joint, previous septic arthritis, haemophilia, pyrophosphate arthropathy, etc

Conservative management

- Modification of footwear, e.g. cushioned heel inserts with a stiff rocker bottom sole
- Splints and orthosis, e.g. moulded ankle-foot orthosis
- Intra-articular steroid injections or viscosupplements
- Non-steroidal anti-inflammatory medication, painkillers

Limited surgical approaches

Can be used to improve symptoms in the short term and buy time.

[a] Attempt to mention this subtly to the examiners when describing radiographs to them.

Arthroscopic ankle debridement

- Can be technically difficult to get into the joint
- May be useful if there is an obvious cause that could be corrected, e.g. anterior impingement osteophytes
- Resection of osteophytes, synovectomy, areas of impingement, loose osteochondral fragments
- Expectations must be reasonable
- Buys time, 90% good/excellent results reported at 2 years
- Poor outcomes in advanced arthritis

Open ankle debridement

- Use anterior/lateral approach based on future definitive procedures
- More invasive
- Can be complicated with cutaneous nerve entrapment, extensor tendon damage, wound dehiscence and formation of hypertrophic scar tissue

Joint distraction using Ilizarov fixator

- Improves symptoms of post-traumatic OA
- The fixator is applied for a period of 3 months. The articular surfaces do not come into contact with one another during this time. The patient is allowed to weight bear. At 6 weeks hinges are applied to the construct to allow movement whilst maintaining distraction
- The increased hydrostatic pressure within the joint is thought to stimulate proteoglycan production
- Improvements in range of movement and pain as well as increased radiological joint space are seen at 2 years
- Motion distraction better than static fixed distraction
- No improvement in range of motion
- Effect temporary rather than long lasting
- Common side effects: Pin site infection

Definitive surgical procedures[b]

Total ankle arthroplasty for ankle arthritis

- Early designs of ankle arthroplasty relied upon a cemented and constrained configuration and were subject to loosening, up to 85% in 1 series at 5 years
- Second-generation implants (cementless, semi-constrained, either fixed bearing or mobile bearing) have a reduced rate of revision
- Renewed interest with improved jigs providing reproducible results and mobile bearings improving mobility and reducing loosening. Appreciation that realigning the hindfoot and restoring ligament integrity improves survivorship, resulting in better intermediate-term outcomes
- Whilst ankle arthrodesis, for many surgeons, remains the gold standard treatment for end-stage ankle arthritis, there is enough survivorship data around to make a case for an ankle replacement
- Challenges of revision: Poor soft-tissue coverage, loss of bone stock, poor choice of off-the-shelf revision components. Fall-back position is arthrodesis
- Meta-analysis by Haddad et al. in 2007[1] gave equivalent outcome at 5 and 10 years for both groups

Indications for total ankle arthroplasty

- Low physical demand patient
- Age >55 years[c]
- Coronal mal-alignment <10° of varus and valgus[d]
- Competent (or reconstructable) deltoid ligament and lateral ligament
- Degeneration secondary to inflammatory, osteoarthritis or post-traumatic arthritis

Relative contraindications

- Age <55 years
- Diabetes mellitus
- Poor bone stock or bone loss
- Avascular necrosis of the talus
- High BMI
- History of ankle sepsis (consider if >1 year post infection, if inflammatory markers are normal and if preoperative biopsy and aspirate are negative)

Absolute contraindications

- High physical demand (e.g. construction worker)
- Peripheral vascular disease
- Peripheral neuropathy (including Charcot)
- Paralysis/neuromuscular disease
- Coronal mal-alignment >20° varus or valgus
- Previous infection
- Soft-tissue compromise (multiple previous incisions, flaps)

[b] Talking the talk: The options for management of ankle arthrosis would include conservative management, a limited surgical approach such as arthroscopic ankle debridement or a more definitive surgical procedure such as ankle arthrodesis.

[c] Many surgeons are extending the age indication for total ankle arthroplasty, performing surgery in young low-demand patients with inflammatory arthritis.

[d] For example, the original operating technique for mobility total ankle replacement recommends implantation where coronal plane tibial-talar tilt does not exceed 20°. However, many surgeons are extending the indications with respect to mal-alignment, balancing the hindfoot with appropriate calcaneal osteotomy and stabilising the ankle with deltoid ligament or lateral ligament complex reconstruction as required, either as a staged procedure or at the time of arthroplasty. In the exam it is wise to be safe and therefore conservative, but having an awareness of the evolution in practice will not hurt.

Design features TAR

- A successful and well-executed TAR provides the patient with a near normal gait pattern in relation to the kinematics of the knee, ankle and midtarsal joint
- **First-generation ankle arthroplasty:** All polyethylene tibia, metal talar component with cement fixation. High constraint led to loosening. In some cases surface incongruity led to poly wear
- **Second-generation ankle arthroplasty:** Two-component and three-component designs
- In the two-component design the tibial element is a metal-backed polyethylene prosthesis with a metal talar component; hence, a fixed-bearing component, e.g. the Agility™. The Agility™ is the most commonly used TAR in the USA. It relies upon a syndesmotic fusion at the distal tib–fib joint for tibial component stability. The design allows slight side-to-side and rotational movement as the talus component moves within the tibial component, dissipating rotational forces
- In the three-component design both talar and tibial elements are metal with a mobile polyethylene bearing. These may be press-fit or cemented components (e.g. BP, STAR and Mobility™)
- The LCS (low contact stress) ankle arthroplasty developed in the 1970s, evolved into the Buechel–Pappas (BP) arthroplasty, named after the designers, being the first three-component design
- The mobile-bearing implants, with their highly congruent bearing surfaces, overcome the problem of high contact stress and the resultant poly wear. At the same time they are not as constrained as the first-generation designs, which reduce shear at the bone/implant interface and, thus, reduce loosening
- The Buechels and Pappas[5] themselves reported 2–12 (mean 5) year follow-up with 88% good–excellent, 5% fair and 7% poor outcomes in a series of 75 BP ankle replacements with deep sulcus talar component design. Wood et al.[6] reported 79% survivorship at 6 years in 100 BP ankle replacements
- The STAR ankle has evolved from a cemented fixed bearing ankle to an uncemented mobile bearing three-component arthroplasty. It features two anchor bars to improve tibial fixation and a concave talar component which is reciprocally shaped to the poly meniscus. The designer reported 95% 5-year survivorship and these results have been reproduced by other authors
- Mobility ankle arthroplasty is a three-component mobile bearing press-fit ankle arthroplasty[7]. This has now been withdrawn from the market due to a high incidence of medial pain
- The rate of intraoperative and postoperative malleolar fractures is high especially during the learning curve but does not adversely affect the final outcome
- The most important surgical principle is to achieve the soft-tissue balance and congruent alignment of the component for long-term survival of the prosthesis

Benefits and complications of ankle arthroplasty

Benefits

- Maintains mobility
- Prolongs/prevents progression of arthritis in adjacent joints

Recent advances have made short- and intermediate-term results of second-generation implants more positive (78–80% survival at 5 and 10 years[1]).

Complications

A recent meta-analysis has characterized complications and the prevalence in total ankle replacement and their likelihood of causing failure.[8] The study reviewed 2386 implants with a mean failure rate of 12.4% at a mean follow-up of 64 months.

Nine main complications are reported at the rates below:

1. Subsidence (10.7%)
2. Aseptic loosening (8.7%)
3. Intraoperative fracture (8.1%)
4. Wound healing problems (6.6%)
5. Technical error (6%)
6. Implant failure (5%)
7. Non-union (4.4%)
8. Postoperative fracture (2%)
9. Deep infection (1.7%)

By grouping the non-union and aseptic loosening groups, eight reasons for TAR failure have been identified. Three of these complications (deep infection, aseptic loosening and implant failure) resulted in >50% of failures. Conversely, two of the more common complications (intraoperative fracture and wound healing delay) did not lead to failure in any case. Deep infection was an uncommon complication (occurring in only 1.7% of arthroplasties) and resulted in failure in 80.6% of the arthroplasties in which it occurred. Complications have, thus, been graded in terms of their likelihood of causing failure:

Low-grade complication – Very unlikely to cause failure

- Intraoperative fracture
- Wound healing problems

Medium-grade complications – Failure occurs <50% of the time

- Technical error
- Subsidence
- Postoperative fracture

High-grade complication – Failure occurs >50% of the time

- Deep infection
- Aseptic loosening
- Implant failure

Ankle arthrodesis for ankle arthritis

There is plenty of evidence that ankle arthrodesis yields good results in the alleviation of pain from ankle arthritis, but there

are important critical long-term adverse outcomes to consider, including the late onset of arthritis in contiguous joints[9].

Ankle arthrodesis

- This is historically the gold standard
- A fusion rate between 80% and 90% has variously been reported
- Relief of pain is usually excellent but most have limited hindfoot motion that makes walking on uneven ground difficult and few are able to run effectively
- Gait analysis shows that walking speed is decreased, as are step length and single stance duration

Types of ankle fusion

- Compression arthrodesis using rigid internal fixation, e.g. cross screw or parallel screw compression, anterior tension plate
- Arthroscopically assisted ankle fusion (only in the absence of gross deformity)
- Compression arthrodesis with external clamp (Charnley). High incidence of pin tract and superficial infections
- Ilizarov technique: Allows tibial lengthening at the same time
- Intramedullary nail for tibio–talar–calcaneal fusion

Position of fusion

- Neutral position: Dorsiflexion/plantarflexion (10° equinus if patient cannot stabilise the knee) (CP)
- 5° valgus
- 5° external rotation – Similar to contralateral limb
- Traditional teaching recommends slight posterior translation of the tibia

Complications

- Non-union, mal-union, infection, poor wound healing, pin tract infection, tibial fractures, amputation, painful neuroma, posterior tibia nerve injury, vascular injury
- Wound infection and breakdown with a reported incidence of up to 40% in some series. Non-union occurs in one-third of cases in some series

Chronic ankle instability

Introduction

This is a must-learn key topic for the oral part of the examination.

Ankle sprains are very common and the vast majority can be managed conservatively without any functional instability.

Anatomy

Lateral ligaments

- **Anterior talofibular ligament (ATFL):** Primary restraint to anterior translation of talus
- **Calcaneofibular ligament (CFL):** primary restraint to varus forces to talus
- **Posterior talofibular ligament (PTFL):** strongest, last to fail

Medial ligaments

Deltoid: Superficial fan-shaped from tip of medial malleolus to talus, navicular and calcaneus. Deep part is more important for stability and is attached to talus

Other important ligaments

Syndesmotic ligaments

AITFL: Anterior-inferior tibiofibular ligament
PITFL: Posterior-inferior tibiofibular ligament
IOTFL: Interosseous tibiofibular ligament, which extends into interosseous membrane

Peroneal stabilisers

SPR: Superior peroneal retinaculum
IPR: Inferior peroneal retinaculum

Mechanism of injury

A forced inversion injury leads first to damage to the ATFL. In more severe injuries this is followed by damage to the CFL. It is almost impossible to injure the CFL in isolation. Disruption of the PTFL is rare.

Acute inversion injuries can be graded as either unstable or stable.

Unstable injuries are further subdivided according to the degree of talar tilt and anterior drawer present under stress. A partial or complete tear of the ligament complex heals in a lengthened position, causing lateral joint laxity. If only the ATFL is involved, anterior subluxation of the talus in the ankle mortise will occur. When both the ATFL and CFL are injured, talar tilt will also be present.

History and examination

Giving way: usually painless and mechanical
Painless/painful: most instabilities are painless. If painful must exclude internal impingement, osteochondral fractures, peroneal injuries and arthritis
Acute/chronic: acute instabilities need conservative approach at least for 4–6 weeks
Functional/mechanical: functional instabilities are not true instabilities and are mainly due to internal impingement. This requires EUA and arthroscopy
Locking/clicking: arthritis, osteochondral fragments
Hyperlaxity (Beighton's score): may lead to recurrence and, hence, needs more robust non-anatomical repairs

Key points

- **Exclude hindfoot varus, forefoot overpronation and subtle cavus deformity:** most important predisposing factor to recurrent lateral instability. This may lead to failure of surgical repairs if not corrected simultaneously. Hindfoot varus/first ray overpronation can be addressed

with lateral displacement calcaneal osteotomy and dorsiflexion osteotomy of the first metatarsal
- Most chronic lateral instabilities are painless. If painful, exclude associated occult pathology (OCLs, synovitis, OA, peroneal tendon pathology)
- Anatomical repairs restore joint biomechanics, but weak as scarred tissues used for repair
- Non-anatomical repairs are potentially more robust, but do not restore normal joint kinematics and stiffen the subtalar joint
- Recurrence is associated with generalized ligament laxity, high functional demand and missed hindfoot varus. Robust non-anatomical repairs may be preferred at the expense of a stiff subtalar joint

Management

Acute injuries and instabilties

Most sprains improve with conservative management including rest, ice, analgesia and early rigorous rehabilitation

Indications for surgical reconstruction

- Failure of conservative management (>4–6 weeks). MRI scan is useful to confirm the presence of chronically ruptured or chronically attenuated ankle ligaments, and is useful to exclude occult ankle osteochondral lesion or ankle synovitis
- Chronic instability with positive clinical examination and positive stress radiographs
- Anterior drawer sign: Absolute drawer >10 mm or >5 mm on contralateral side., positive varus tilt test: Absolute talar tilt of >10° or >5° on contralateral side

Surgical approaches

Surgical reconstructions are grouped broadly into anatomical repairs of the ligaments and non-anatomical repairs using tenodesis, and, more recently, anatomical reconstruction with tenodesis.

Anatomical repairs

Anatomical repair (e.g. Brostrom–Gould) utilizes the local tissue to restore normal anatomy and joint kinematics, and maintains ankle and subtalar joint movement. Within this principle lies its weakness in the respect that the native tissue may be scarred or attenuated by recurrent sprain and, therefore, suboptimal for purpose. It has a very good, long-term reproducible result and is the first line of surgical treatment.

Brostrum

Direct repair of ATFL and CFL (anchors/fibre-wires/ Ethibond™)

Gould

Augment the repair with inferior retinaculum

Steps

- 'J 'shaped incision over the distal fibula, protecting the superficial peroneal nerve branches and peroneii
- ATFL and CFL ligaments remnants are identified
- The ATFL is divided mid substance
- The anterior cortex of the distal fibula is freshened with a burr, suture anchors are deployed into the anterior fibula and into the junction between the talus neck and body and then the ATFL halves are double-breasted over one another
- The Gould component of the repair involves gathering the free edge of the extensor retinaculum and, by means of the suture anchors, suturing it to the freshened surface of the fibula and the adjacent periosteum

Non-anatomical repairs

Non-anatomical repair uses tenodesis to restrict joint motion and restore stability without repair of the native ligaments. Naturally such repairs do not accurately restore the joint kinematics and have a tendency to stiffen both ankle and subtalar joint.

The Evans repair harvests the entire peroneus brevis tendon, suturing its muscle belly onto peroneus longus, then routes the tendon up through the fibula from the tip of the fibula emerging posteriorly, 3 cm proximally. The tendon is sutured under tension to the periosteum, to create a ligament.

The Chrisman–Snook repair routes the anterior half of peroneus brevis tendon through the fibula, while maintaining its distal attachment to the base of fifth metatarsal and attempts to recreate the ATFL and the CFL by routing the tendon graft from anterior to posterior through the fibula, securing the free proximal end in the body of the os calcis.

The non-anatomical repairs have been proven to provide reproducible, effective and longlasting stabilisation of the ankle, accepting that they do restrict subtalar joint motion, produce symptomatic stiffness and do not reproduce normal ankle kinematics.

Complications of surgery

- Ankle and subtalar stiffness
- Recurrence
- Scar tenderness
- Superficial peroneal/sural nerve injury

Flowchart for ankle sprains/instability (Figure 18.2)

Oral question

- Describe a surgical technique for chronic ankle instability

Examination corner

AP radiograph of the ankle demonstrating talar tilt (Figure 18.3)

This topic always begins with a clinical radiograph of the ankle showing talar tilt. There may also be a lateral radiograph demonstrating anterior talar shift.

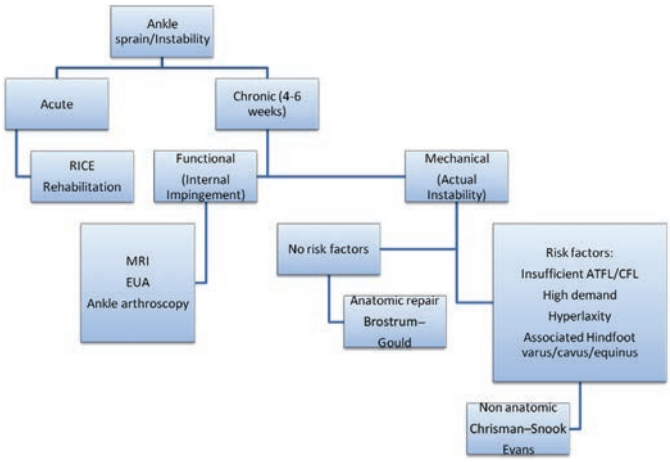

Figure 18.2 Flowchart for ankle instability

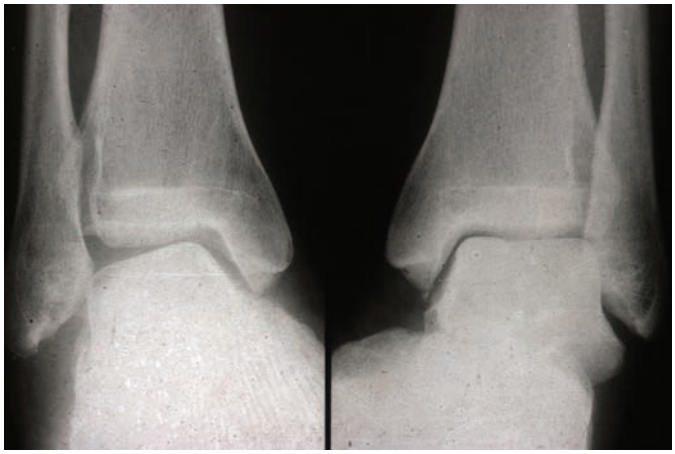

Figure 18.3 Radiograph of the ankle demonstrating talar tilt

Adult and pathology oral 1

EXAMINER: This is a radiograph of an ankle. What does it demonstrate?

CANDIDATE: This is a lateral stress radiograph of the ankle, which shows tibiotalar shift indicating injury to the lateral ankle ligament complex.

EXAMINER: How are you going to manage this patient?

CANDIDATE: I would initially manage this patient with physiotherapy.

EXAMINER: Come on, is physiotherapy going to help in this situation with this amount of talar tilt?

CANDIDATE: A patient may have significant talar tilt on stress views but not require surgery. The literature suggests that up to 80% of grade 3 injuries can be treated conservatively with good results.

EXAMINER: This patient is being seen in your fracture clinic 6 months following a lateral ligament injury which is not getting any better. She has severe symptoms of pain and instability and these are her stress views. Are you really going to manage her with physiotherapy, which she has had on two previous occasions?!

CANDIDATE: In this case I would consider surgical reconstruction.

EXAMINER: Can you name any surgical procedures performed for chronic ankle instability?

CANDIDATE: Brostrom–Gould lateral ligament reconstruction.

EXAMINER: Why a Brostrom–Gould?

CANDIDATE: I'm not sure.

EXAMINER: . . .

(Fail)

The first point would be to say that just mentioning physiotherapy as an initial management for the condition without qualifying the answer further is inviting trouble. A much better reply would have gone along the lines of '*The stress views show significant tibiotalar tilt and, if taken in conjunction with an appropriate history and clinical examination, are suggestive of chronic ankle instability, this may be an indication to consider surgical management.*' Even better to continue on with '*Surgical options can be either an anatomical repair such as a modified Brostrom repair or a non-anatomical repair such as Chrisman–Snook repair.*'

This examiner seemed to give the impression that a positive stress view equated with a surgical reconstruction. It is important to be quite clear that a positive stress view in itself is not an indication for surgery. Stress radiographs may show significant talar tilt and anterior subluxation but the patient may have minimal symptoms of ankle instability and may not require a reconstructive procedure. This point should have been mentioned in the general discussion in retort to the examiner's comments.

Trauma oral 1: clinical radiograph showing tibiotalar tilt

CANDIDATE: This is a radiograph, which is a stress view demonstrating significant tibiotalar tilt suggestive of chronic ankle instability.

EXAMINER: You are seeing this patient in the fracture clinic 6 months after an ankle sprain with this x-ray. What are you going to do?

CANDIDATE: If he has symptomatic, disabling ankle instability then I would offer him surgery. It is important to emphasize the need to identify hindfoot varus, forefoot pronation and subtle cavovarus deformity as a correctable underlying cause of instability.

EXAMINER: Is he likely to have anything else but symptomatic instability with this radiograph? *(Not a question but a somewhat sarcastic comment.)* What types of repair are you familiar with?

CANDIDATE: A repair of the lateral ligament can be either augmented or non-augmented. *(A slightly mixed up answer, not entirely accurate.)*

EXAMINER: *(Not entirely happy with answer.)* What is the recommended type of repair to perform?

CANDIDATE: It would probably be a modified Brostrom repair.

EXAMINER: An anatomical repair. What would you tell the patient the outcome is likely to be?

CANDIDATE: In fact, most ankle reconstructions do very well, with a 90% success rate. It's one of these operations that tends to do well.

EXAMINER: Yes, you are quite right about this. When would you allow a patient to return to playing football?

CANDIDATE: Three months. *(Complete guess)*

EXAMINER: Would you allow them to play unsupported or would you make them wear a support brace?

CANDIDATE: I would allow them to play without any support.

COMMENT: Complete guess and wrong answer. Most athletes should continue to use a tape or brace indefinitely during sports activities, but a brace is not routine after 3 months for most work-related activities or activities of daily living.

Adult and pathology oral 2: clinical radiograph showing tibiotalar tilt

CANDIDATE: This is a radiograph, a stress view demonstrating significant tibiotalar tilt suggestive of chronic ankle instability.

EXAMINER: How do you manage this patient? *(Saved by bell.)* We will leave it at that.

Adult and pathology oral 3: clinical radiographs, AP stress view showing tibiotalar tilt and lateral stress view showing anterior subluxation talus on the tibia

CANDIDATE: This is a radiograph, a stress view demonstrating significant tibiotalar tilt suggestive of chronic ankle instability.

EXAMINER: How much of a tilt is significant?

CANDIDATE: A tibiotalar tilt of >15°.

EXAMINER: You haven't commented on the lateral x-ray. What do you think about the talus?

CANDIDATE: It's moved forward.

EXAMINER: Yes, the radiograph demonstrates an anterior subluxation of the talus. What value of subluxation is significant?

CANDIDATE: 3 mm.

EXAMINER: 3 mm compared to what?

CANDIDATE: Compared to the normal contralateral side.

EXAMINER: What surgical operations do you know that can be used to manage this condition?

CANDIDATE: You can either perform an anatomical repair or a non-anatomical repair. An anatomical repair is usually preferred and this can be either augmented or non-augmented. The classic operation was a Brostrom repair in which there was a midsubstance repair of the ligament but this tended to stretch out in time. Gould modified the Brostrom repair, by reinforcing with suturing of the lateral extensor retinaculum. This modification is important in patients who have excessive inversion and laxity of the subtalar joint. However, there is very little to choose between the last two methods. There was a randomised controlled trial performed by Karlsson, which showed that the Gould repair had more complications and slightly lower functional scores than the modified Brostrom repair.

Diabetic foot

Introduction

Approximately 10% of diabetic admissions to hospital are with foot problems. The severity of diabetic foot disease is directly related to the adequacy of blood sugar control.

Pathology

- Diabetic ulcers : one-third neuropathic; one-third ischaemic; one-third mixed

Neuropathy

Symmetrical distal polyneuropathy involving motor, sensory and autonomic nerves.

Autonomic dysfunction

- Reduced sweating causes dry plantar skin. Prone to fissure
- Alters nail growth
- Reduced local vascular response to injury

Sensory disturbance

Painless sensory neuropathy causes a stocking-distribution sensory loss

Reduced pinprick sensation, light touch and vibration.

Semmes–Weinstein monofilament test

This simple test can identify persons at an increased risk for foot ulceration:

A 10-g (5.07) nylon monofilament is applied to the sole of the foot

If the patient perceives the touch of the monofilament at the point it buckles, they have protective sensation; if they fail to perceive it, they do not

Loss of vibration sense with a 128-Hz tuning fork is a sensitive predictor of early neuropathy

Motor involvement

Intrinsic muscles weakness and imbalance between the long flexors and extensors leads to cavus foot and claw toes.

Tibialis anterior weakness results in equinus contractures. The metatarsal heads are pulled forwards, decreasing cushioning and increasing vertical and shear forces.

Peripheral vascular disease

Is common, has bilateral involvement.

Clinical assessment

A. **Assess diabetic control** (type, duration, severity, medications, HbA1C level)

HbA1C level indicates blood glucose level control over the preceding 3 months

Normal: 6.5%

B. **Assess ulcers ± infection:** neuropathic/vascular/mechanical, duration, previous ulcers and treatments, bloods, x-rays, MRI, WBC scan, bone scan or labelled white cell scan. Wagner stage documentation

Risk factors for ulceration

- Dry skin leading to fissures
- Loss of protective sensation causing micro-trauma
- Autonomous vascular ischaemia
- Deformities causing high pressure areas especially MT heads and plantar midfoot area

Neuropathic ulcers

- Location: typically under metatarsal heads
- Painless
- Punched out margins
- Healthy granulation tissue at base which bleeds on touch
- Surrounding thick hyperkeratosis
- Palpable pulses and distended veins
- Evidence of loss of protective sensation

Ischaemic ulcers

- Location: anywhere in foot or leg
- Painful
- Dull fibrotic base, poor granulation tissue, doesn't bleed easily
- No hyperkeratosis
- Surrounding hair loss
- Pulses: less palpable, ABPI <0.8

Classification: Wagner (Table 18.1)

Assess neurovascular status (ABPI Index, monofilament testing, vascular team)

For nerve function

- Biothesiometer: Measures vibration perception threshold. The calculated standard deviation score evaluates the risk of ulceration
- Semmes–Weinstein hairs
- Nerve conduction studies

For vascular status

- Doppler ultrasound
- ABPI: Normal value is 1 (0.8–1.2); a value <1 indicates peripheral vascular disease: >0.45 required for any surgical intervention
- Transcutaneous oxygen saturation: Correlates highly with risk of wound breakdown. Normal: 40; <25 – high risk of wound dehiscence
- Angiography

Assess deformities (stable/unstable, pain, functional loss)

Deformities

- Clawed toes
- Plantar callosities: metatarsalgia with increased pressure under the metatarsal heads
- The claw toes expose the metatarsal heads to further mechanical insult with plantar ulceration
- Equinus contractures: increased risk of plantar ulcerations
- Other deformities: due to chronic Charcot foot – Cavus, hallux valgus, rocker bottom

Management

Mainstay of treatment is effective control of blood glucose level

Prevention is better than cure
Patient education
Accomodative footwear with padding in high-risk areas

Ulcer management –– **Superficial, non-infected ulcers and neuropathic ulcers:**

Total contact cast with areas cut to offload the ulcers
Correct mechanical deformities or equines contractures

Ischaemic ulcers:

Optimize vascularity: vascular team involvement for angioplasty or bypass surgery
Total contact casting (TCC)

Ulcers with infection:

Stepladder approach from antibiotics to amputation

Risk factors for failure of TCC

- Large ulcers (>2 cm)
- Long duration (>2 months)
- Wagner grade III or above
- Associated deformities (clawing/equinus/midfoot–hindfoot instability/rocker bottom)

Table 18.1 Wagner classification foot ulceration

Grade	Description	Management
0	No ulcer, but foot deformity present, at risk foot for ulcers	Educate the patient and advise them to modify their footwear
1	Clean, uninfected, superficial ulcer	Broad-spectrum antibiotics
2	Deep ulcer with necrotic tissue	Debridement
3	Deep ulcer with abscess/osteomyelitis If bone palpable/probed: 67% risk of osteomyelitis	Debridement + local antibiotic/sequestrectomy
4	Forefoot gangrene	Partial amputation
5	Gangrene of the entire foot	Below knee amputation

Deformities management (see Charcot foot) –– Pathophysiology:

- Neurotraumatic: loss of protective sensation – Microtrauma – Deformity
- Neurovascular: Autonomous hyperaemia causes increased bone resorption
- Inflammatory: injury – inflammation – Osteoclast stimulation – Bone resorption and remodelling

Classification: Eichenholz:

Stage 0 (prefragmentation)

Acute inflammation
Regional demineralisation
Difficult to differentiate from infection

Stage I (fragmentation)

Painful
Periarticular demineralisation and fragmentation, leading to dislocations/fractures

Stage II (coalescence)

Painless/dull ache
Sclerosis

Stage III (remodelling)

Painless
Deformities (mal-united ankylosed joints)

Goals of treatment

- Reduce deformities to prevent subsequent complications of ulceration, infection and amputation
- Provide stability mainly to hindfoot and ankle
- Prevent ulcerations through protective braces/footwear

Principles

- Mainstay is diabetic control and patient education
- Accomodative footwear
- Prevent ulcerations; when present: Aggressive treatment with TCC
- Avoid operating in acute (fragmentation/demineralisation) stage as difficult fixation
- Arthrodesis preferred over ORIF
- Treat equinus contractures
- Long-term non-weight-bearing: 2–4 months followed by protective weight-bearing in cast/brace for 6 months to 2 years
- Acute deformities/dislocations (esp. midfoot) need surgery for correction even during fragmentation stage
- Chronic deformities and dislocations should be managed with modified footwears/orthotics/braces. Indication for surgery in chronic stable arthropathies is uncontrolled progression of deformity not responding to conservative measures

Two acute deformities that frequently develop in the midfoot

- Medial dislocation of the navicular and cuneiforms with forefoot in abduction causing medial skin necrosis (apex medial): usually treated with osteoectomy
- Dorsal dislocation of navicular and cuneiforms with medial column shortening causing lateral rocker bottom deformity (apex lateral): Usually needs arthrodesis

Three main stays of surgical treatments

1. **Osteoectomy:** to offload the non-healing ulcers. Avoid incisions on weight-bearing surfaces. Limited bone resection not to jeopardize the midfoot stability as will lead to recurrence
2. **Hindfoot stability:** Commonly through tibiotalocalcaneal arthrodesis through hindfoot nail
3. **Amputations:** for recurrent, intractable deformities or ulcerations/COM

Oral questions

- Discuss the role of amputation in the diabetic foot
- Describe how to salvage 'the foot at risk'

Interdigital neuroma

Definition

A neuroma consists of degeneration and fibrotic changes in the common digital nerve near its bifurcation.

Aetiology

Unknown. There may be similar changes in unaffected nerves. Several causative factors have been suggested, although none is universally accepted.

- Anastomosis between the medial and lateral plantar nerves in third webspace
- High heeled shoes with narrow toebox: Forced toe dorsiflexion
- Compression by the transverse intermetatarsal ligament
- Bursal hypertrophy

Symptoms

Can be non specific

- Women > men
- Neuralgic sharp pain in a toe and/or interdigital space
- Burning, tingling pain over the involved toes
- Numbness
- Pain worse on walking, sometimes at night
- Most common third webspace, followed by second and fourth webspaces

Diagnosis

Examination

- Local plantar pain and tenderness of the involved nerve in the intermetatarsal space
- Mulder's click on metatarsal compression: Reproduces the patient's symptoms
- A local anaesthetic injection into the affected space that relieves symptoms

Special investigations

- Standing AP and lateral weight-bearing films to exclude other forefoot pathology
- MRI (preferred over ultrasound scan but has false positive rate)
- Ultrasound scan: Can be combined with steroid injection

Differential diagnosis

- Synovitis
- Bursitis
- Metatarsalgia
- Tarsal tunnel syndrome suggested by discomfort around the ankle
- Peripheral neuropathy, diabetes
- Spinal disorder: HNP, history of nerve root entrapment

Management

Conservative

- Advice and education of the patient
- Sensible accomodative shoewears. Wider toe box. Avoid high heels
- Metatarsal bar: Offload forefoot
- Corticosteroid injection can be successful, especially if the history is short and size is small

Surgery

- Dorsal/plantar incision: Revision mainly through plantar approach
- Nerve plantar to deep transverse ligament and, hence, easier exposure with plantar exposure
- Recurrence mainly due to left over plantar branches
- Planar incision may give painful scar
- Divide transverse inter meta-tarsal ligament
- Excise nerve 2–3 cm proximal to the bifurcation
- Can bury the nerve in interosseous muscle

Consent

- Warn patients of the 80% success rate
- Patients may develop a painful scar
- Recurrence
- Warn about the area of numbness in the web space

Nerve supply to the foot and ankle

This is a predictable question about foot anatomy. There are many ways for the examiner to lead into the subject.

Posterior tibial nerve

- A branch of the sciatic nerve
- Enters the deep posterior compartment of the leg between two heads of the gastrocnemeii
- Travels deep to soleus, between it and tibialis posterior
- Branches into medial and lateral plantar nerves, and calcaneal sensory branches
- It supplies the plantar aspect of the foot

Medial plantar nerve

Motor to
- Abductor hallucis
- FHB and FDB
- First lumbrical

Sensory to
- 3½ digits, like the median nerve in the hand

Lateral plantar nerve

Motor to
- Adductor hallucis
- Dorsal and plantar interossei
- Second–fifth lumbricals
- Abductor digiti minimi

Sensory to
- 1½ digits

Calcaneal sensory branches provide sensation to the heel pad

Saphenous nerve

- A terminal branch of the femoral nerve
- Supplies the anteromedial side of the leg down to the dorsomedial ankle and midfoot

Deep peroneal nerve

- A branch of the common peroneal nerve
- Supplies only a small area of skin on the first web space
- Passes beneath the extensor retinaculum at the front of the ankle joint between EHL and EDL

Superficial peroneal nerve

- A branch of the common peroneal nerve
- Exits the deep fascia anterolaterally about 8–12 cm above the tip of the fibula
- Supplies the dorsum of the foot except the first web space (deep peroneal nerve) and the lateral part of the foot supplied by the sural nerve
- It descends in peroneus longus until it reaches the peroneus brevis, and passes over the anterior border of peroneus brevis and descends between it and EDL

Sural nerve

- A branch of the tibial nerve
- Pierces the superficial aponeurosis halfway down the leg between the two bellies of the gastrocnemius and is joined by the peroneal communicating nerve
- Supplies the lateral aspect of the heel, the fifth metatarsal and small toe

Tarsal tunnel syndrome

Entrapment neuropathy of posterior tibial nerve and/or its branches due to compression in the tarsal tunnel. Patients can have both proximal (whole nerve) and distal (terminal branches, especially the lateral plantar nerve 1½ digits) syndromes.

Boundaries

- Medial: medial malleolus proximally, medial aspect talus and calcaneus distally
- Flexor retinaculum
- Abductor hallucis

Aetiology

Above medial mallelous

Space occupying lesions (SOLs)

- Ganglion in the tendon sheaths
- Varicosities
- Neurilemma tibial nerve
- Lipomas
- Diabetics
- Rheumatoid synovitis/tenosynovitis

At/below the level of medial mallelous

- Calcaneal fractures: susttentaculum tali/medial process of calcaneus
- Heel valgus: Pes planus, tib post insufficiency, talocalcaneal tarsal coalitions
- Accessory FDL muscle

History

- Intractable, diffuse and poorly localized nerve pain
- Burning, tingling, numbness and cramping sensation that radiates onto the plantar and medial aspect of the foot
- Hard- or soft-tissue mass

Examination

- Heel valgus with hyperpronation of foot
- SOL
- Pain/paraesthesia in typical distribution
- Positive Tinel's sign behind the medial malleolus
- Positive provocation test: direct manual compression or dorsiflexion and eversion

Investigations

- X-rays: exostosis/fractures
- Nerve conduction studies: 90% accurate; sensory changes more pronounced
- MRI: SOL

Management

- Steroid injection
- Insoles
- Surgical decompression

Surgery

- 80% successful
- Curved incision posterior to medial malleolus, extending distally upto abductor hallucis muscle
- Divide flexor retinaculum
- Divide both the superficial and deep fascia over abductor hallucis
- Release medial and lateral plantar nerve completely
- Release plantar fascia if necessary

Complications

- Recurrence: incomplete release, revision surgery; poor results
- Bowstringing

Anterior tarsal tunnel syndrome

Entrapment neuropathy deep peroneal nerve beneath inferior part of extensor retinaculum.

Aetiology

- Tight-laced shoes
- Anterior ankle osteophytes
- TMT joint osteophytes
- Tibialis anterior/EHL/EDL tenosynovitis
- SOL

Examination

- Paraesthesia first webspace
- Positive Tinnel's sign
- Positive provocation test: ankle forced plantarflexion

Management

- Non-operative
- Surgical: decompression of nerve with release of extensor retinaculum
- Cheilectomy ankle and TMT joints

Hallux rigidus

Hallux rigidus is a degenerative arthritis of the first metatarsophalangeal (MTP) joint of the big toe. There is painful

limitation of first MTP joint movement, particularly dorsiflexion. Later on, osteophytes appear on the dorsal and/or lateral articular margin and block extension.

Epidemiology

- Twice more common in females
- Usually bilateral

Aetiology

- Idiopathic
- Post-traumatic
- First-ray hypermobility
- Metatarsus elevatus (definite correlation; however, the MT elevation may be secondary due to intrinsic contracture and planar plate retraction)
- Osteochondritis dissecans
- Inflammatory: gout/pseudogout/rheumatoid

History

- Activity-related pain with dorsiflexion
- Stiffness, block to dorsiflexion (mechanical)
- Painful plantar flexion due to capsular stretch (functional)
- Difficult shoe wear: Especially high heels
- Difficulty in the push-off phase of running
- Dorsal prominence, swelling with ulceration

Examination

- Assess the presence of marginal osteophytes, which are typically dorsally and laterally
- Painful first MTP ROM
- Pain during tiptoeing
- Check motion at IP joint (it should be mobile)
- Dorsal medial cutaneous nerve is often sensitive
- Assess for presence of other foot pathology, e.g. lesser toe deformities, metatarsalgia

Investigations

Weight-bearing AP and lateral radiographs.

Stage I: mild osteophytosis, joint space preserved
Stage II: moderate osteophytosis, narrow joint space
Stage III: severe osteophytosis, joint space obliteration

Assess IP joint status, lesser toes deformities

Management

Conservative treatment

Reassurance: The radiological stage not always related to clinical symptoms, which may progress slowly
NSAIDs
Footwear modification: Rigid, moulded and stiff insoles or rocker bottom insoles

Limited surgical procedure

MUA and intra-articular steroid injection may provide relief of symptoms in mild/moderate cases. Not proven to be effective if severe changes are present.

Joint preserving

1. **Cheilectomy:** removal of the dorsal osteophyte first MT
   - Active patient, stage I disease,
   - Motion preserving, relieves pain while maintaining stability and allowing for a secondary procedure in the future
   - Oblique resection of 30–50% of joint (up to one-third of the dorsal MT head) and resection PP osteophyte
   - Simultaneous proximal phalanx or MT osteotomy
   - If degenerative changes present, then increased ROM can lead to more symptoms
   - Preop mid range of motion pain or crepitus is a warning sign for a poor result after cheilectomy
   - Cheilectomy des not work when hallux rigidus is severe, coexistant sesamoid disease or long first MT: Shorten MT1 to offload sesamoid joint
2. **Closing wedge osteotomy of the proximal phalanx (Moberg)**
   - Dorsiflexion osteotomy (~10°)
   - Changes the arc of motion from flexion to extension: End result is increased dorsiflexion (useful in runners/athletes)
   - Reduced pressure on dorsum of first MTP joint
   - Useful for adolescent with early degenerative change, can be combined with cheilectomy
3. **Metatarsal osteotomy**
   - Plantar flexion osteotomy
   - Reduces dorsal impingement
   - No better than cheilectomy

Joint sacrificing

1. **Keller's procedure: excision arthroplasty**
   - Old patients with limited activities
   - Low demand, home ambulators
   - Complications: transfer metatarsalgia, cock up deformity (persistent MTP joint dorsiflexion), weak flexor strength, hallux varus/valgus, hallux instability
   - Revision to fusion can be difficult and require bone block
2. **Interposition arthroplasty**
   - Proximal phalanx base resection and interposition material either dorsal capsule, gracilis tendon, extensor hood with extensor hallucis brevis
   - Weakens plantar flexion, transfer metatarsalgia

- Maintains MTP joint motion, whereas arthrodesis doesn't, good short term results reported
- Contraindications: Short MT1/adjacent metatarsalgia

3. **Silastic (Swanson) implant arthroplasty**
   - Earlier used for low-demand, elderly patients but now also used for younger patients
   - Many modifications of implants and cuts (primus-tornier) available
   - Complications: Silicon synovitis, implant wear, implant breakage, dislocation, bone lysis and loss, cock-up deformity
   - Limited evidence for use
4. **MTP joint replacement (hemi/total)**
   - Hemi (Hemicap™)
   - Total (Moje™)
   - Ceramic on ceramic/metal on polyethylene
   - Complications: Implant loosening, implant fracture, osteolysis, bone overproduction, cyst formation and transfer metatarsalgia
   - Limited long-term evidence to support use
5. **Arthrodesis**
   - Gold standard
   - Stage II and III
   - Optimum position of fusion: 10–15° valgus and 25° dorsiflexion
   - The IP joint should be mobile (accelerates IP joint arthritis)
   - Motion sacrificing: Loss of pivoting movement during sports; inability to wear high heels
   - Osteotomy saw cuts/cone reamers
   - Fixation with screws, plates or pins
   - Complications: delayed union/non-union, under/overcorrection of deformity, screw loosing/breakage, transfer metatarsalgia, sesmoiditis, degenerative arthritis IP joint
   - Non-union: inadequate joint surface preparation, instability of the fusion site, thermal necrosis bone, poor patient compliance postoperatively, smokers

Hallux valgus

Introduction

Common short case or adult and pathology oral topic.

High-yield orthopaedics, go through a viva dry run to polish your answers.

Definition

Hallux valgus is a lateral deviation of the great toe with medial deviation of the first metatarsal.

Aetiology

Multifactorial, more common in women (8 : 1), less common in non-shoe wearing population.

1. Intrinsic
   - Idiopathic
   - Familial
   - Hyperlaxity: Generalized hypermobility, tarso-metatarsal joint instability
   - Inflammatory: RA, gout
   - Neuromuscular: CP, Down's syndrome, Marfan's, stroke
2. Extrinsic (acquired)
   - Related to footwear(tight-fitting shoes, pointed toe shoes, high-heeled shoes)

Pathogenesis

Multiple theories

- Increased GRF: GRF acts on medial aspect of first MTPJ, pushes PP into valgus. This is currently most favoured
- Primary metatarsus varus: this is the primary deformity with hallux valgus as secondary deformity
- First TMTJ hypermobility

Pain may be extrinsic or intrinsic

Extrinsic pain

- Due to deformity

Extrinsic pain may be managed non-operative management by

- Shoes with a wider deeper toe box
- Padding the bunion
- Pressure from the next adjacent toe can be managed with a silastic toe spacer

Intrinsic pain relates to

- Joint incongruence
- Degeneration
- Synovitis: MTP joint/sesamoid joint

Intrinsic pain is more readily treated by surgical restoration of joint congruence although orthotics (sole stiffner, Morton's extension, forefoot rocker) may have a role.

Important assessments

- Painless/painful: must identify the source of pain (bunion/arthritis/inflammatory synovitis/transfer metatarsalgia). Inflammatory and degenerative conditions better treated with first MTPJ arthrodesis
- Age: extreme age preference for non-operative management
- Is deformity passively correctable? Need for Akin osteotomy or with severe deformity more proximal procedures
- Unstable first TMT/hyperlaxity/recurrence from previous surgeries: Role of Lapidus procedure to stabilise first TMT joint

- Tendo Achilles tightness
- Associated lesser toe deformities
- Previous interventions
- Radiological assessment: joint congruency (DMAA), joint degeneration, sesamoid stations, lesser toe deformities, first TMTJ hypermobility with plantar gapping/subluxation (Table 18.2)

Surgical principles

- Remove the bunion
- Correct the hallux valgus angle
- Correct the intermetatarsal angle
- Correct hallux interphalangeus
- Correct or maintain the distal metatarsal articular angle (DMAA)
- Restore joint congruence: Most deformities are incongruent and, hence, do not need DMAA correction. (Congruent HV: no joint subluxation and DMAA >10°; incongruent HV: Joint subluxation but DMAA is normal.)
- Avoid first MT shortening (defunctions first ray)
- Avoid first MT elevation (defunctions first ray)
- Stabilise and debulk the medial MTP joint capsule with capsulorhaphy
- Avoid plantar dissection to prevent AVN first MT head
- Relocate sesamoids under first MT head : Normally located under MT1 head over cristae, within FHB tendons. Sesamoids remain in their position but with first MT going into varus, lateral and upward rotation of sesamoid takes place. This leads to adductor and FHB contractures leading to pronation and progression of deformity
- The more proximal the osteotomy, the better the correction
- Double osteotomies for severe deformities with altered DMAA

Pressure studies demonstrate that hallux valgus deformity effectively defunctions the first ray; that is to say, the patient does not load the first ray optimally. The consequence of this is load transfer to the lesser rays, resulting in transfer metatarsalgia, plantar plate rupture and clawing of the lesser toes. The radiological correlation would be hyperostosis of the second ray and all the features of lesser toe clawing.

Hallux valgus surgery should be designed to 'refunction' the first ray, and in particular the maintenance of length. The limitations of some of the osteotomies performed in orthopaedics today can be easily identified when checking down this list. Thus, for example, the Mitchell's osteotomy inevitably shortens and defunctions the metatarsal; the distal chevron does not correct DMAA and is, therefore, not applicable where this is a goal of surgery (also associated with a risk of metatarsal head instability and AVN).

None of the metatarsal osteotomies address the problem of tarso-metatarsal joint instability, albeit a none-too-common phenomenon.

A lapidus fusion, first tarso-metarsal joint arthrodesis, may be applicable, though attention must be paid to MTP joint congruence, and the need for an attendant distal metatarsal osteotomy to correct the DMAA must be recognised. Lapidus fusion has some notoriety in being prone to delayed and non-union.

Problems of great toe deformity and posture are addressed with lateral release and, where necessary, Akin osteotomy to correct hallux interphalangeus and great toe pronation.

The lateral release primarily allows correction of the sesamoid station, and includes release of the metatarso–sesamoid suspensory ligament and the phalangeal insertion band.

For severe hallux valgus, with widely divergent intermetatarsal angle, many authors recommend proximal metatarsal osteotomy with distal soft-tissue balancing, such as the proximal chevron osteotomy, proximal crescentic osteotomy or the Ludloff osteotomy (critics of which cite instability, loss of fixation and dorsal mal-union, shortening and metatarsal head elevation as significant risks).

As with the lapidus fusion, the proximal osteotomy may render the MTP joint incongruent, by dramatically altering the DMAA. In these circumstances the need for a double osteotomy must be identified.

Where the MTP joint is mildly arthritic, the patient might be counselled with respect to corrective osteotomy, accepting the possible need for an arthrodesis at a later date. Where the MTP joint is frankly arthritic, the patient may be counselled with respect to the need for an arthrodesis.

Other indicators of a potential need for MTP joint fusion in managing hallux valgus include: Severe deformity with

Table 18.2 Angle values in hallux valgus

Angles	Description	Normal	Mild	Moderate	Severe
Hallux interphalangeus	Between the proximal and distal articular surfaces of PP1	<10			
Hallux valgus	Between MT1 and PP1 axes	<15	20–30	30–40	>40
Intermetatarsal	Between MT1 and MT2 axes	<9	11–15	15–20	>20
Distal MT articular angle (DMAA)	Between the articular surface of MT head and axis of MT1	<10			

osteoporosis, severe MTP joint incongruence or frank dislocation associated with severe hallux valgus, salvage after failed bunion surgery or failed arthroplasty, hallux valgus associated with neuromuscular disease/spasticity, inflammatory arthritis and salvage after severe infection.

For the purposes of the exam, make your mind up as to which osteotomy you prefer, and justify it in the context of the deformity correction goals described above.

If, the night before the exam, you still can't make up your mind, go for a Scarf and Akin osteotomy with lateral release, accepting the main criticism that the surgical exposure is overlong and that 'troughing' is a potential problem. The Scarf and Akin osteotomy with lateral release can be manipulated to achieve every surgical goal, and is applicable to the most severe deformity. Where tarso–metatarsal joint instability may be considered a potential problem, the Scarf osteotomy does not preclude a later lapidus fusion.

Radiographic parameters

Standing AP and lateral radiographs forefoot:

- Hallux valgus angle (HVA), the angle between the diaphyseal axis of the first metatarsal and that of the proximal phalanx of the great toe: Upper limit of normal 15°
- Intermetatarsal angle (IMA), the angle between the diaphyseal axis of M1 and M2: Upper limit of normal 9°
- Interphalangeal angle, the determinant of hallux valgus interphalangeus, reflects the angle of joint line convergence between the proximal and distal articular surfaces of the proximal phalanx, and is measured as the angle between the long axis of the phalanx, and the axis of the distal metaphyseal/joint segment of the phalanx, normally <10°
- The distal metatarsal articular angle (DMAA), describes the angular relationship between the articular surface of the head and the axis of the diaphysis of the first metatarsal: Normally <10°

Assess also for:

- Congruity of first MTP joint
- Degenerative changes
- Sesamoid position
- Overall foot shape

Degree of severity (Figures 18.4 and 18.5)

Mann and Coughlin classified deformities by HVA:

- **Mild,** <20°
- **Moderate,** 20–40°
- **Severe,** >40°

By IMA (some overlap and inconsistency in the literature):

- **Mild,** <11°
- **Moderate,** between 14° and 20°
- **Severe,** > 20°

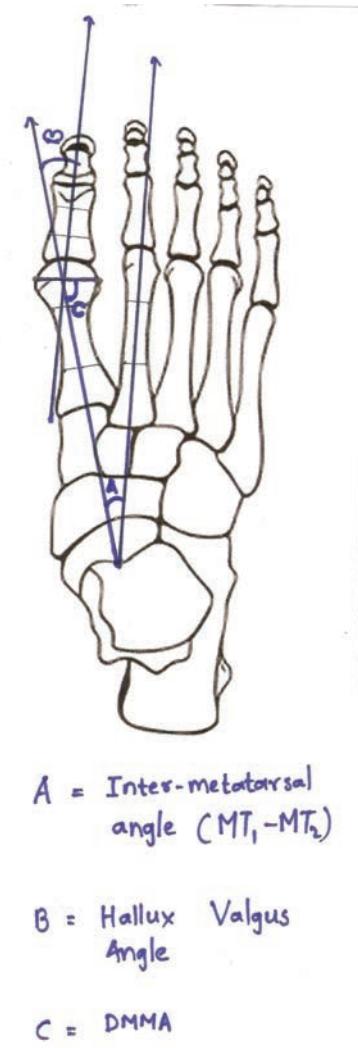

Figure 18.4 Hallux valgus angles. A, Intermetatarsal angle (MT1–MT2); B, hallux valgus angle; C, DMMA.

Management

Main clinical concerns are pain, difficulty with footwear and cosmesis.

Hallux valgus correction – The goals of treatment

- Alleviate pain
- Correct the deformity
- Refunction the first ray
- Reduce transfer metatarsalgia

Conservative management

- Activity modification
- Footwear modification (wide toe box, padding to the bunion, Silastic spacer)
- Orthotics (sole stiffener and a forefoot rocker to offload the forefoot)

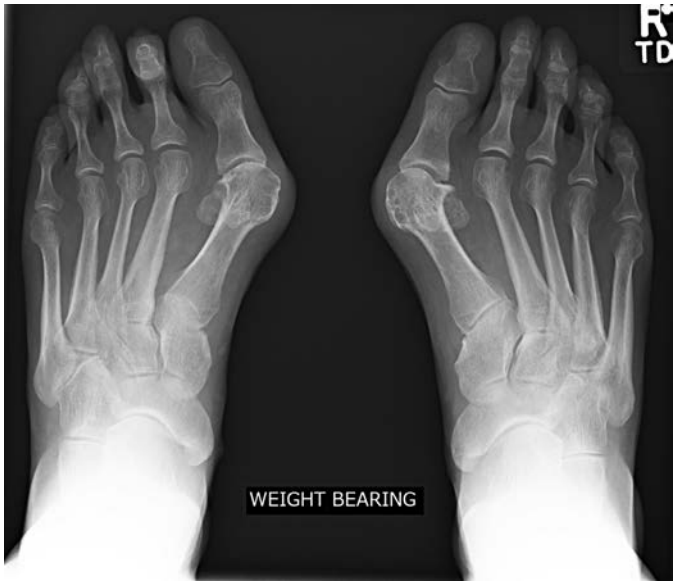

Figure 18.5 AP radiograph of left hallux valgus. There is an incongruent first MTP joint, lateral sesamoid dislocation and bunion exostosis. The metarsophalangeal angle is 44° and intermetatarsal angle is 17°

Operative management

- Mild deformities: lateral release and distal osteotomy
- Moderate to severe deformity: lateral release and Scarf/proximal osteotomy
- Severe deformity with altered DMAA: lateral release and Scarf/double osteotomy
- HV with first TMTJ hypermobility: Lapidus procedure
- Hallux interphalangeus: add Akin procedure

Mild disease

Lateral release

- Allows correction of the sesamoid station
- Includes release of the adductor hallucis, the metatarso–sesamoid suspensory ligament and the phalangeal insertion band

Silver

- Simple bunionectomy and capsular reefing

Complications

- Poor satisfaction rates
- High recurrence

McBride

- For HV with no contracture
- Simple bunionectomy
- Transfer of released adductor tendon from PP to lateral MT1 neck
- Lateral release, plus medial capsulorhaphy

Complications

- Poor satisfaction
- High recurrence (70%)
- Hallux varus
- Medial cutaneous nerve injury or entrapment, hypoaesthesia, neuroma

Mitchell's osteotomy

- Excision medial eminence, step cut osteotomy at the metaphyseal-diaphyseal junction and medial capsulorrhaphy
- For mild deformity with HVA >30° and IMA >13°
- Defunctions the first ray as shortens the first MT (transfer metatarsalgia, plantar plate rupture, clawing lesser toes, reoccurrence)

Chevron osteotomy

- V-shaped, extracapsular, distal metatarsal osteotomy
- For mild to moderate deformity but IMT angle <12°
- 70° cuts with more longer and horizontal plantar limb (perpendicular to GRF)
- Head displaced up to 50% width of metaphysis (>50% – Destabilisation)
- Can correct DMMA if medial wedge resection combined with osteotomy
- Intrinsically stable but fixation with single screw is preferred to avoid mal-union

Complications

- Loss of position
- Osteonecrosis (damage to plantar and lateral blood supply: 2–20%)

Moderate to severe disease

Wilson

- Oblique osteotomy made through the distal diaphysis from distal medial to proximal lateral
- The distal fragment is slid laterally to correct the intermetatarsal angle
- Redundant medial capsule plicated
- The geometry of the osteotomy requires significant shortening of the 1MT to correct the intermetatarsal angle
- Concern with transfer metatarsalgia means it is out of favour at the moment and not one to volunteer in the exam

Scarf osteotomy

- Most versatile Z-osteotomy allowing multiplanar correction
- Has advantage of both proximal and distal osteotomy due to the Z-cut
- Has larger surface area and, hence, high union rate

- Longitudinal cut of Z is parallel to ground ,and not MT shaft axis, perpendicular to GRF, and therefore stable
- Transverse cuts of Z perpendicular to MT2 shaft but divergent to avoid 'troughing'
- Lateral transfer of dorsal fragment corrects IMT angle
- Rotation of distal fragment can correct the DMAA
- Plantar orientation of cut prevents MT elevation
- Controlled shortening possible to reduce the MTP joint without tension or decompress an arthritic joint but avoid defunctioning the first ray with excessive shortening
- Long-term results very good although no RCT comparing Scarf osteotomy to any other technique
- Minimal risks of transfer metatarsalgia

Complications

- Troughing: Impaction of the two osteotomy fragments, resulting in loss of metatarsal height, fracture and malrotation, resulting in loss of correction
- Dorsal mal-union
- Stress fracture related to the proximal end of the cut
- Steep learning curve, can be difficult to salvage if it goes wrong

Proximal osteotomies

- Allows larger correction with same degree of displacement because osteotomy closer to CORA (centre of rotational angular deformity which is first TMT joint)
- This exerts a longer lever arm allowing correct of very severe deformities that may be beyond distal or diaphyseal procedures

Complications

- Osteotomy is away from the first MTP joint through which the GRF passes this may lead to dorsal mal-union due to the vertical cut in lateral views
- Often causes first MT shortening and, hence, risk of defunctioning first ray and transfer metatarsalgia
- Reoccurrence, hallux varus,first MTP joint stiffness, mal-union and non-union and stiffness

Mann crescentic osteotomy

- Crescentic nature of proximal osteotomy avoids shortening and, hence, the Gold Standard
- Combine with distal osteotomy if DMAA needs correction
- The stability of primary fixation (screw/plate) is pivotal
- Dorsiflexion mal-union common complication
- Good clinical results (level 2 evidence) with improved AOFAS scores

Proximal Chevron osteotomy

- Less correction than Mann
- Osteotomy cuts similar to distal chevron; however, joint penetration is a risk
- Needs stable fixation(screw and K-wire, two screws or a plate)

Ludloff

- Long oblique osteotomy
- Cut: dorsal proximal to plantar distal
- Osteotomy rotates around a screw to get desired correction and then locked with another screw
- Less risk of shortening and dorsal mal-union

Modified Lapidus procedure

- First TMT joint arhrodesis (original lapidus: MT1–MT2 base arthrodesis)
- For young patients with hypermobile joint
- For HV with first tarso–metatarsal joint instability or arthritis
- Severe deformities with large Inter metatarsal angle (>25°)
- Recurrent hallux valgus

Complications

- Non-union 10–20% (inadequate joint preparation, apposition or fixation, premature weight-bearing): Requires bone grafting as otherwise leads to too much shortening
- Doesn't correct DMAA
- May lead to MT elevation (dorsal mal-union) and shortening

Akin osteotomy

- PP base osteotomy: Adjunct to MT corrective osteotomy
- For residual HV or fixed pronation deformity after MT osteotomy
- Hallux valgus interphalangeus
- Crossover deformity with second toe
- Shortens PP1 and, hence, releases soft-tissue tension on flexors and extensors

Keller's arthroplasty

- Resection of the base (one-third) of the proximal phalanx of the great toe
- Low-demand patients

Complications

- Transfer metatarsalgia
- Cock-up deformity (hallux extensus due to PP shortening and EHL contracture)

First MTP joint arthrodesis in HV

- Severe HV with first MTPJ arthritis, joint incongruence or frank dislocation
- Severe deformity with osteoporosis
- Salvage after failed previous surgery
- HV associated with neuromuscular disease/spasticity, inflammatory arthritis
- Salvage after severe infection

Other complications

- Recurrence: 5–20%
- Hallux varus: 1–5%
- AVN: 1–2%

Hallux varus

Aetiology

- Congenital: (1) primary (uncommon), varus-only deformity; (2) related to metatarsus adductus, clubfoot, equinovarus
- Acquired – Neuromuscular, trauma, rheumatoid arthritis
- Over-correction of hallux valgus (common): typically valgus mal-union of MT1
- Described as varus deformity but triplanar involving supination first MT joint, hyperextension first MT joint and hyperflexion hallux IP joint
- Hallux deviated or subluxed medially with a non-purchasing digit in varus rotation and usually negative inter metatarsal angle (MT1–MT2)
- Excessive lateral release or tight medial capsulorrhaphy
- Abductor hallucis tightness
- Excessive resection of medial eminence
- Historically, fibular sesamoidectomy as part of McBride procedure thought to be primary aetiology. However, a sesamoidectomy done as an isolated procedure will not produce the varus. Though due to a combination surgical errors

Clinical features

Usually deformity well tolerated
Treatment if painful
Assess for ligamentous laxity, neuromuscular disorders

Important assessments (to be done as patient weight bears)

- Flexibility IP1 joint (deformity correctable or not)(static or dynamic)
- Flexibility MTP1 joint
- Associated contractures/ joint degeneration (arthritis/ rigidity will contraindicate tendon transfer/tenodesis)
- Associated MT1 deformities (Contraindicates tendon transfer/tenodesis unless deformity is corrected first)
- Gait
- Radiographs

  All hallux valgus angles are used, hallux abductus IP angle, degree of spray between first and second MT, any elevates

Management

- Reassurance if asymptomatic
- Strapping in valgus: if seen in early postoperative period; for 3 months
- Shoe wear modifications

Surgical indications

- Failure of conservative strapping
- Painful MTPJ
- Shoe wear problems
- Obvious cause of deformity: valgus mal-union

Surgical options

1. **Soft-tissue corrections**
   - Medial release and lateral stabilisation
   - Abductor hallucis lengthening/release/transfer from medial PP1 base attachment to lateral PP1 base under MT1 head
   - EHL split transfer/EHB tendon transfer/tenodesis: The harvested tendon is passed under the deep transverse MT ligament from distal to proximal. For dynamic transfer, tendon is stitched back to itself. For static transfer (tenodesis), tendon is passed through MT head
2. **Bony correction**

   MT1 osteotomy: If mal-union present
   PP1 osteotomy
3. **Joint procedures**
   - Arthrodesis IP joint: Especially if there is fixed varus and flexion deformity of IP joint
   - MTPJ arthrodesis: For rigid deformity with fixed varus and extension. Most idiopathic hallux varus would require MTPJ arthrodesis
   - Resection MTPJ arthroplasty: For coexisting IP joint arthritis Combine with IPJ arthrodesis

Bunionette deformity

- Idiopathic
- RA
- Hallux valgus and primus metatarsus varus – 'splayfoot'
- Women

Classification: Coughlin

- Type I: Large/bowed fifth MT head
- Type II: Bowed fifth MT shaft
- Type III: increased intermetatarsal angle MT4–MT5 >8°.

Important assessments

- Cosmetic/symptomatic
- Painful: Plantar condyles
- Painful callosities
- MTP joint stability
- Shoe-wear problems

Management

Non-operative: advice, shoe wear modification, padding, callus management

Surgical: failure of conservative treatment

- Type I: Osteotectomy – Bunnionette excision
- Type II: Chevron or diaphyseal osteotomy (reverse Scarf)
- Type III: Proximal oblique MT5 osteotomy
- Fixation with twist-off screws/ K-wire

Risks

- MTP joint instability
- Worsening of deformity if excessive bony resection
- Delayed-union

Examination corner

Adult and pathology oral 1

Discuss the conservative management and appropriate surgery indicated for a given clinical example of hallux valgus. A number of different severities could be shown in a viva and candidates ideally should prepare set answers for different degrees of HV. Typically a very severe deformity ± other associated deformities of the foot requiring surgery will be shown but any thing is game and several photographs may be shown. Make sure you know how to confidently talk around the radiology.

Adult and pathology oral 2: clinical photograph of a woman with a severe hallux valgus deformity

EXAMINER: This 48-year-old lady presents to your clinic complaining about this foot deformity.

CANDIDATE: This is a clinical photograph which demonstrates a severe left hallux valgus deformity. There is no obvious ulceration of her bunion but the skin over it appears atrophic, shiny and red.

EXAMINER: How are you going to manage this patient?

CANDIDATE: I would take a full history and perform a clinical examination of the patient. I would want to know if she has any pain in the big toe . . . *(at this point the candidate was cut short by the examiner)*

EXAMINER: She has pain and she cannot wear normal shoes.

CANDIDATE: Although she has a severe deformity I would still like to try conservative management. *(See Discussion, below.)*

EXAMINER: Come on now, is conservative management likely to be successful in this lady?

CANDIDATE: Not really, no.

EXAMINER: These are her radiographs.

CANDIDATE: I would need to calculate the hallux valgus angle and intermetatarsal angle.

EXAMINER: What are the normal values for these angles?

CANDIDATE: The normal hallux valgus angle is 9° and the normal intermetatarsal angle is 15°.

EXAMINER: I think you are a bit confused and have got them mixed up. Never mind, let's move on to another topic you may know more about.

(Candidate failed oral)

Discussion

The oral topic was lost at this stage because the candidate's reply was not particularly well thought out. The candidate first failed to mention the need for standing weight-bearing AP and lateral radiographs and what he/she would look for with them. The examiner alluded to this later on.

The second point concerns mentioning conservative management for a severe hallux valgus deformity. Standard examination protocol dictates that when discussing management options we are always told to mention conservative treatment first. Generally speaking, to jump in and start discussing surgery without first referring to it can be an invitation for trouble. Equally, you can dig a hole for yourself if you mention conservative management for a severe hallux valgus deformity and do not quantify your answer, as with this candidate.

A better reply would be, *'This lady has a severe hallux valgus deformity and I would offer her a basal metatarsal osteotomy for the condition if she is willing to accept the risks of surgery.'*

Or, covering all bases, *'This lady has a severe hallux valgus deformity. Conservative management is unlikely to be successful in this case and I would offer her surgery. Conservative management certainly has a place in a less severe deformity and is a perfectly acceptable form of management for mild deformities.'*

Adult and pathology oral 2: clinical photograph of a middle-aged woman with severe hallux valgus deformity

CANDIDATE: This is a clinical photograph, which demonstrates a severe hallux valgus deformity of her left big toe.

EXAMINER: She is complaining of severe pain in her bunion. How are you going to manage her?

CANDIDATE: Even though it is a severe hallux valgus deformity and conservative management is unlikely to be helpful I still think we should initially consider it[e].

EXAMINER: It is not likely to be successful though, surely.

CANDIDATE: No, it is not, but before I would consider surgery I would like to find out a little bit more about her pain, whether the MTP joint has arthrosis, how old she was . . . [f] *(Candidate was interrupted)*

EXAMINER: She is 52; she has pain only in the bunion.

CANDIDATE: I would like to examine the foot, paying particular attention to the neurovascular status because if it is compromised I will not be performing any surgery on her.

EXAMINER: Good.

[e] This candidate has an unfortunate turn of phrase with this answer. Be careful when mentioning conservative management for a severe hallux valgus deformity. The candidate has not explained clearly enough the role of conservative management, either generally or in this patient's specific case.

[f] The candidate's answer jumps about too much. The candidate mentioned conservative management and then backtracked and he then started to discuss history and examination findings. No mention at all about the role of radiographs in the management of the procedure although they were right in front of him on the table.

CANDIDATE: She's 52, so a Keller's procedure would not be a good choice for her as she is too young. If there is arthritis at the MTP joint then my preference would be to perform a distal osteotomy such as a Wilson's procedure or Mitchell's osteotomy. I realize that a basal osteotomy is used by some surgeons to a treat severe hallux valgus, but I am not familiar with this procedure. I have read about it but I have never seen one performed[g].

EXAMINER: How do you do a Mitchell's osteotomy? Why don't you draw it out for us. Here is a pen and paper.

CANDIDATE: This is the metatarsal and I would perform a double osteotomy here.

EXAMINER: Where?

CANDIDATE: At the neck.

EXAMINER: That's fine; it is not quite clear on your diagram.

CANDIDATE: I would then displace the metatarsal laterally and secure the displacement with a screw through the two fragments[h].

(Candidate failed)

Adult orthopaedics and pathology oral 3: hallux valgus in a young patient

- Diagnosis, including radiographs
- Various angles to consider
- Management

Adult and pathology oral 4: hallux valgus

- Detailed questioning
- Things to look for in history and examination
- Angles
- Scenarios for treatment
- Metatarsal cuneiform fusion

Adult and pathology oral 4: clinical picture of mild hallux valgus deformity

- Full discussion on hallux valgus
- Pathogenesis
- Angles

[g] You should be aware of basal metatarsal osteotomies and be able to describe how to perform one if asked. I presume the examiners were wanting to discuss this for the management of a severe hallux valgus deformity. The candidate's answer was more of an excuse than anything else and certainly not good enough for the examiners. Most foot and ankle surgeons prefer to perform either a basal osteotomy or a Scarf procedure for a severe deformity.

[h] The candidate was not detailed enough in his description of how to perform a Mitchell's osteotomy. A few extra details were needed to safely satisfy the examiners. The examiners wanted the candidate to discuss more fully the various surgical procedures available to manage a severe hallux valgus and how to perform them. They were not particularly interested in the history or examination findings and wanted to hurry the candidate along so that they could discuss the technical details of the operation. It is much safer to go through the history, examination and investigations regime if they allow you to do so if only to avoid getting caught out in the more difficult technical details of an operation.

EXAMINERS: The candidate was not confident in the management of hallux valgus.

EXAMINER: What would you do for this patient?

CANDIDATE: A distal osteotomy.

Discussion about consent for the procedure then took place and the candidate was asked to draw a diagram showing the distal osteotomy.

Adult and pathology oral 5: surgical options for a severe hallux valgus deformity

Scarfe and Akin oral

EXAMINER: Describe how you would perform a lateral release and a Scarf and Akin osteotomy.

CANDIDATE: A lateral release is performed to enable the sesamoid bones which are often subluxed to relocate to their normal station beneath the metatarsal head. It is performed through a dorsal incision in the first web space at the level of the first MTP joint. The release varies from surgeon to surgeon, but the elements released can include:

- The metatarso–sesamoid suspensory ligament which in reality is the condensation of the capsule of the MTP joint where it descends to the sesamoids. This incison is longitudinal rather than vertical, as a vertical incision will divide the collateral ligament and predispose to hallux varus. This allows the head to move laterally over the sesamoids
- The phalangeal insertion band, a condensation between the fibular sesamoid and the lateral base of the proximal phalanx, is released
- The tendons of the adductor hallucis muscle may be released in severe cases
- Finally, if necessary, the deep transverse metatarsal ligament may be released

EXAMINER: OK. How do you perform a Scarf and Akin osteotomy?

CANDIDATE: A medial longitudinal inscison is made along the midline of the medial border of the foot. Caution is exercised to avoid the medial dorsal cutaneous branch of the saphenous nerve. The capsule is divided in the midline and reflected dorsally but not plantarly as this is the point of entry of the vascular pedicle to the metatarsal head. The shaft is exposed proximally, identifying but not exposing the tarso–metatarsal joint. The capsular reflection is continued onto the base of the proximal phalanx, but protecting the insertion of EHL and the insertion of the plantar plate at the base of the proximal phalanx.

The bunion is excised, protecting and preserving the sesamoid ridge, again to avoid a hallux varus, and then the Scarf cut is performed.

EXAMINER: What are the principles?

CANDIDATE: A Z-cut osteotomy, with a short distal limb perpendicular to the second ray, sloping back at 60° and commencing at the shoulder of the metatarsal. A short proximal limb, again sloping back at 60°, but this limb may be perpendicular to the second metatarsal or sloping slightly proximally to facilitate rotation.

The third longitudinal cut joins the apices of the first two cuts, and the saw blade is directed in the plane of the sole of the foot, or, if necessary, slightly plantarward, to depress an elevated metatarsal.

EXAMINER: What are the concerns?

CANDIDATE: In displacing the osteotomy the metatarsal halves may trough, elevating the head. The displaced osteotomy preserves or even restores length and can, if necessary, be rotated to correct the DMAA.

EXAMINER: How is it fixed?

CANDIDATE: Two variable pitch screws are used, usually Barouk screws.

EXAMINER: Are there any concerns with this fixation?

CANDIDATE: The metatarsal is fragile and may split, so the surgeon must introduce the screw slowly, to allow stress relaxation to prevent a split.

EXAMINER: What are the principles of an Akin osteotomy?

CANDIDATE: The Akin is a medial closing wedge osteotomy of the proximal phalanx of the great toe, used to correct hallux interphalangeus.

EXAMINER: What are the concerns?

CANDIDATE: The plane of the cut must be perpendicular to the axis of the phalanx, to avoid a 'cock-up' deformity, and the great toe tendons must be protected as division of these in performing the osteotomy is reported.

EXAMINER: How would you fix the osteotomy?

CANDIDATE: With either a screw or a staple; my own preference is for a screw.

Bell goes. (Pass)

Examination corner

Radiograph shown of a failed Keller procedure done 15 years previously in a 62-year-old female (Figure 18.6). Discuss your treatment options.

'I went down the route of getting a full history and performing a detailed clinical examination of the foot to define the current problems. There are numerous complications associated with Keller's excisional arthroplasty including recurrent deformity. Although the Keller's procedure has been used for decades for thetreatment of advanced symptomatic hallux valgus, it fails to maintain the proper alignment and biomechanical functionality of the hallux and so has fallen out of favour in recent years. Occasionally it may be used in elderly low-demand patients with painful hallux valgus associated with arthritis of the MTP joint.

Complications include recurrence of the deformity, transfer metatarsalgia, excessive shortening of the toe and 'cock up' deformity. Salvage of the procedure can be difficult with techniques including arthrodesis, re-excision and reconstruction of soft tissue, and placement of hemi-implants or total implants.'

EXAMINER: What are 'YOU' going to offer the patient?

CANDIDATE : I would need to get much more information from the patient before offering surgery. I would want to know how much pain the patient was experiencing and whether it was globally around the foot or more localized from transfer metatarsalgia, beneath the sesaoids, along the cutaneous nerves or from any lesser toe deformity. I would additionally enquire about any difficulty wearing certain shoes, limitation of activities of daily living and the cosmetic appearance of the big toe.

I would also enquire about the previous surgery, any general conditions such as rheumatoid arthritis and any medical problems such as diabetes or peripheral vascular disease. I would additional want to know her occupation and any sporting activities she does. It is also very important to explore her expectations from surgery as any revision surgery is likely to interfere with high performance sports such as running or pivot type sports, plus the toe may still be sore after spending a long time on her feet during the day or going up and down stairs and shoe wear may still be an issue.

Operation-wise I would consider arthrodesis of the MTP joint with interpositional bone graft as it has reasonably good results reported in the literature and is recognised as a good procedure in the difficult situation of excessive shortening of the hallux.

EXAMINER: What would you warn the patient of postoperatively?

CANDIDATE: I would warn them it would be a complicated surgical procedure with no guarantee of success, and in particular there would be the increased risks of infection, delayed union, non-union, implant breakage, ankylosis of the hallux IP joint breakage of metalwork, reoccurrence of deformity and general dissatisfaction with the procedure.

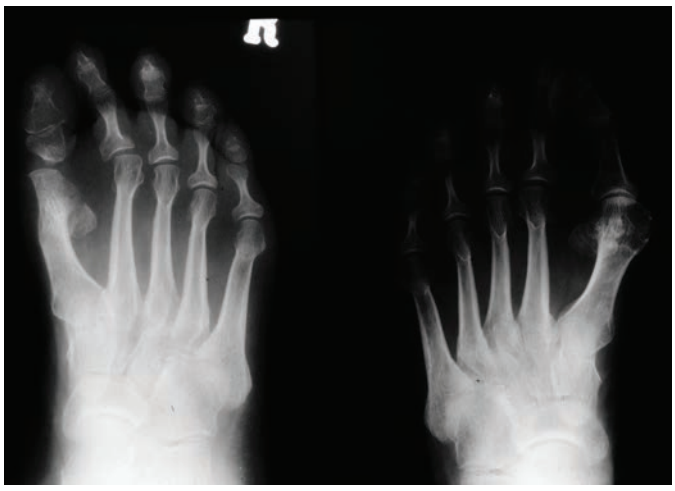

Figure 18.6 AP radiograph demonstrating excessive bone removal following Keller's excision arthroplasty

Pes planus

Two common presentations of flat foot in the exam are tarsal coalitions (covered in the paediatric section) and adult acquired flat foot.

Arches of foot

Longitudinal and transverse arches which are maintained by:

- Static stabilisers: The shape of the foot bones

- Dynamic stabilisers; the activity of muscles
- A wide variety of intrinsic muscles and ligaments

Longitudinal arches: medial and lateral (Figure 18.7)

Medial longitudinal arch (MLA)

- Tall, concave arch
- Resilient due to many bony components
- Consists of apex (trochlea surface of the talus) and 2 pillars (MT 1–3 heads, medial tubercle of the calcaneus)
- Strong dynamic component: tibialis posterior

Lateral longitudinal arch (LLA)

- Arch is flat and contains few bones
- The pillars are MT 4–5 heads and lateral tubercle of calcaneum
- Difficult to define the apex of the LLA because although body weight is transmitted into it via the talus, the talus is not part of the arch
- Dynamic component: peroneus brevis

The longitudinal arches are supported and stabilised by:

1. Bony support: The shape of the bones which allows them to interlock
2. Tendons which attach at the apex of the arches and increase the arch height (tibialis anterior)

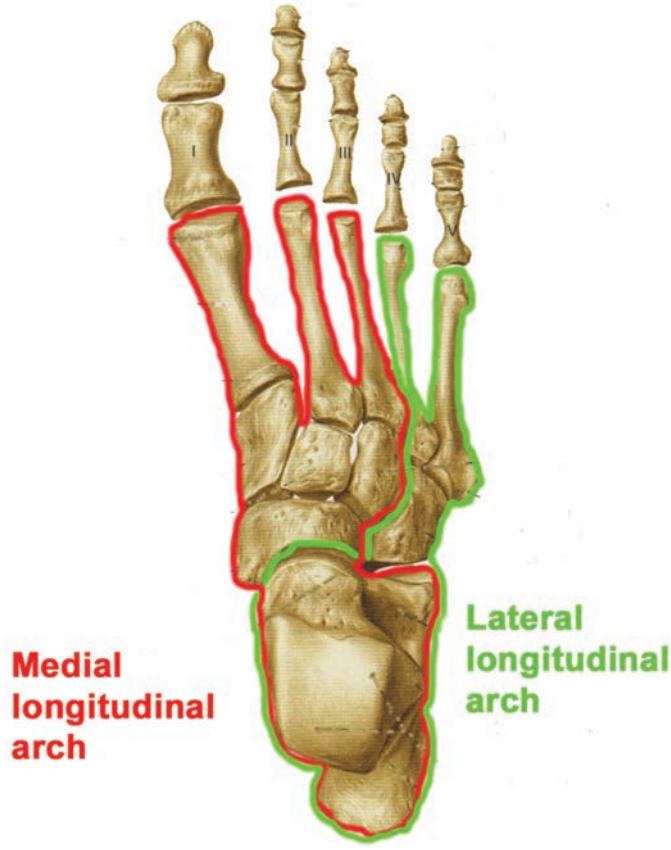

Figure 18.7 Medial and lateral longitudinal arches of the foot

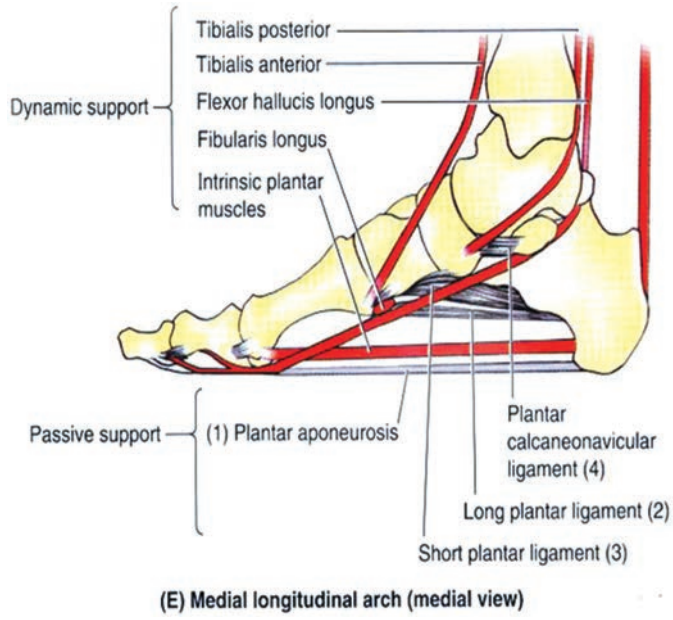

Figure 18.8 Medial longitudinal arch of the foot

3. Tendons which run a longitudinal course in the foot. They prevent the extremities separating (peroneus longus and small foot intrinsics)
4. A variety of longitudinally arranged ligaments that prevent the extremities separating. (The long and short plantar ligaments, plantar calcaneonavicular ('spring') ligament.)
5. The plantar aponeurosis links the extremities of the arches, and acts as the equivalent of a tie beam in an architectural arch

Transverse arch

- Not a true arch
- Provides mainly bony support: cuneiforms intrlinked with each other, supported by ligaments

Tibialis posterior insufficiency

More likely a clinical case rather than viva topic but would not discount either (Figure 18.9).

- Hypovascular zone: Within 14 mm of its insertion on the navicular tuberosity
- Female gender
- Inflammatory arthritis

The decision to operate is based on

- Mobility of the hindfoot (fixed/correctable heel valgus and ST joint)
- Mobility of the forefoot (can the heel valgus be corrected without forefoot supination?)
- Tibilalis posterior muscle(tear/defunctioned)
- Functionality of ligaments (spring and deltoid ligaments)
- Presence of arthritis

Surgery

- Conservative

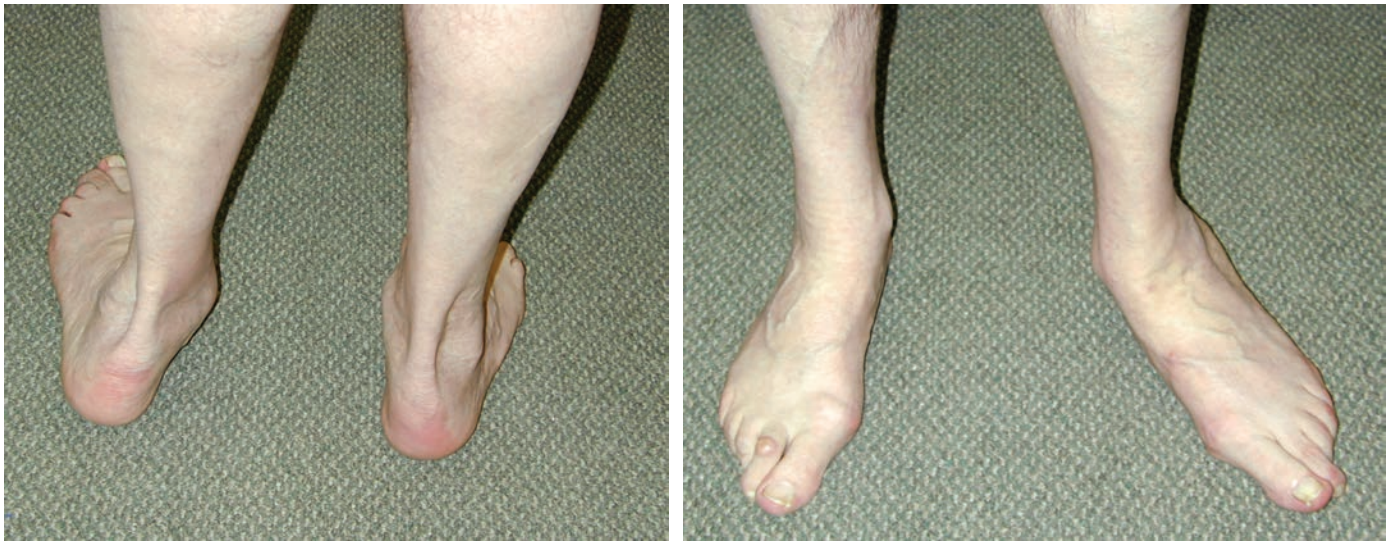

Figure 18.9 Clinical photograph of tibialis posterior insufficiency.Too many toes sign with heel valgus

- Joint preservation(soft-tissue reconstruction of tendons and ligaments, bony osteotomies
- Joint sacrificing(arthrodesis)

Classification: Johnson and Hattrup

Three clinical stages of posterior tibial tendon dysfunction. Myerson added a Stage IV, where there is a fixed foot deformity and tilting of the talus in the ankle mortice. The natural history is believed to be a progression from tendonopathy without deformity, through a mobile deformity to a fixed deformity.

Stage I: Tenosynovitis tibialis posterior, no deformity

Tib post is functional and intact but inflamed.

Patient can do a single heel raise test but with mild weakness.

Symptoms are mild to moderate with posteromedial pain, swelling but minimal deformity.

Absent "too many toes sign".

Treatment: Tenosynovitis: Tenosynovectomy of the tibialis posterior tendon.

Partial tear but normal hindfoot: Conservative treatment with medial heel wedge-brace.

If deformity appears: Consider tenosynovectomy and medial translational CO.

Stage II: Ruptured or dysfunctional tib post tendon with flexible deformity

Tib post is elongated or torn and, therefore, weak and dysfunctional.

Inability to maintain resisted inversion.

Inability to perform single heel raise test.

Heel valgus present but flexible; flatfoot present.

Subtalar joint still mobile.

Deformity is still passively correctable.

Treatment: FDL transfer to tib post and medial calcaneal translational osteotomy.

- Associated deformities
- Forefoot fixed supination: add medial cuneiform dorsal open wedge osteotomy
- Heel equinus: add Gastroc release /TA lengthening
- Forefoot abduction: add lateral column lengthening

FDL transfer

- Harvested at Master knot of Henry where it crosses over FHL
- Tenodesis of distal stump with FHL unnecessary
- Always excise diseased tibialis posterior tendon
- Can perform tenodesis with proximal tib post tendon if muscle healthy

Cobb procedure

- Split transfer tibialis anterior to tibialis posterior proximal stump. Transfer part of tibialis anterior through a tunnel in the medial cuneiform and back through the sheath of tibialis posterior, attaching it proximally
- Distal tibialis posterior excised or used to reconstruct spring ligament
- Tibialis posterior muscle has to be healthy and functional for transfer to work
- Combined with medial translational or Rose calcaneal osteotomy
- Not synergistic as FDL

Medial displacement calcaneal osteotomy

- Medial translational (~1 cm) or step cut Rose osteotomy
- Shifts Achilles pull axis medially
- Re-aligns the hindfoot biomechanics reducing ankle and subtalar joint reaction forces
- Brings axis of ST joint to neutral

Lateral column lengthening: Modified Evans

- Calcaneal lengthening when flexible flatfoot abduction present with first TMT joint instability
- 1-cm opening wedge with iliac crest autograft
- Osteotomy performed 1.5 cm proximal to Calcaneo cuboid joint

Stage III: Fixed hindfoot valgus

Hindfoot valgus rigid.
Forefoot supination and abduction rigid.
Represents advanced disease.
Treatment: Triple arthrodesis.

Occasionally, triple arthrodesis alone may not fully correct the deformity; adjunctive procedures may be necessary to correct residual forefoot varus, forefoot abduction, or hindfoot valgus deformities[10]. Adjunctive procedures include medial displacement calcaneal osteotomy to address residual hindfoot valgus; medial column procedures such as a plantar flexion osteotomy of the medial cuneiform, fusion of the first tarsometatarsal joint or naviculocuneiform fusion to address residual forefoot varus deformity; and lateral column lengthening to address forefoot abduction.

Stage IV: Ankle valgus

- Stage added later
- Chronic disease
- Medial strain at ankle causes deltoid insufficiency and rupture
- Could be flexible or rigid (more common)
- Treatment: Ankle arthrodesis/tibiotalocalcaneal arthrodesis/ankle replacement

Pes cavus

Epidemiology

- CMT is most common inherited motor neuropathy
- Can be neurological in nature
- **Often no underlying neurological cause found**
- Usually symmetrical
- Weakness mainly affects: Peroneus brevis, tibialis anterior, intrinsics
- Men > women but women more severe
- Patients present in second or third decade
- If unilateral it is due to a spinal cord tumour until proven otherwise

Inheritance pattern

- Autosomal dominant (AD) is most common – Defect on chromosome 17
- Affects peripheral nerve myelination (peripheral myelin protein 22 – PMP22)
- Sex-linked recessive and autosomal recessive (AR) forms present earlier
- Are more severe

Predictors of severity

- Early presentation (under 10 years)
- Autosomal or sex-linked recessive
- Females
- Associated sensory deficit (causes ulcers as in diabetes)

Hereditary motor sensory neuropathies

- AD
- Distal motor and sensory deficits
- Family history

Table 18.3 Main hereditary motor sensory neuropathies

Types	HSMN I (CMT hypertrophic)	HSMN II (CMT neuropathic)	HSMN III Dejerine–Stotta disease
Incidence	More common (AD)	Less common (AD)	AR
Onset	10–20 years	20–30 years	Infancy
Pathology	Demyelination	Wallerian degeneration	
Reflexes	Absent	Present	
NC/EMG	Prolonged latency Reduced velocity	EMG normal	
Features	Motor > sensory Peroneii Tib Ant most affected Cavus/cavovarus Hammer toes Hip dislocation rare Scoliosis rare Intrinsic hand muscles weakness	Motor > sensory Peroneii/Tib Ant most affected Cavus/cavovarus Hammer toes Hip dislocation rare Scoliosis rare Intrinsic hand muscles weakness	Foot drop Scoliosis Difficult mbulation

Types

There are seven in total; the most common are shown in Table 18.3.

Cavus

- Plantar flexion of the first ray: Peroneus longus overpowers tibialis anterior
- Exacerbated by intact plantar fascia windlass mechanism

Varus

- Plantar flexion of first ray also causes forefoot pronation. The compensatory tripod effect causes hindfoot varus
- Weak peroneus brevis overpowered by TP

Equinus

- Weak TA overpowered by gastrosoleus complex

Claw toes

- Tib ant weakness causes EHL/EDL recruitment for ankle dorsiflexion
- Intrinsic minus foot
- Both causes MTPJ hyperextension and PIP/DIPJ flexion – Clawing

Clinical features

History

- Deformity and stiffness are the chief complaints – Not pain or parasthesia
- Difficulty with uneven ground and sports
- Family history common
- Plantar pain from metatarsalgia
- Repeated ankle sprains and painful callosities(secondary to clawing)

Examination

- Typical foot deformities
- Are deformities correctable?
- Toe clawing
- Hindfoot varus with a high medial arch or pes cavus deformity?
  - Is the hindfoot deformity correctable with the Coleman block test. If so this shows that the subtalar joint is mobile and the varus hindfoot is driven by excessive plantar flexion of the first ray
- Silverskold test: Improved ankle dorsiflexion with knee flexed = gastrocnemius tightness. Equivalent ankle dorsiflexion with knee flexion and extension = Achilles tightness
- Are joints painful?
  - Indicates arthrosis
- Thighs normal girth, calves thin – ***Stork legs***
  - Sensory changes
  - Less pronounced
    - Proprioception, two-point discrimination and vibration may be affected
  - Muscle power
  - Test appropriate muscles for power (PL, PB, TA, TP, etc)
  - Screen for other neurological problems – Spine, skin, gait (broad-based ataxic gait)
  - In advanced severe cases patients develop upper limb signs
  - Intrinsic minus hand

Investigations

Radiographs

- Standing AP and lateral
- Lateral shows high arch with cuboid visible in profile
- Increased calcaneal pitch angle (>30° = abnormal)
- Positive Meary's angle (0–5° = normal)
- AP shows fibula more posterior due to external rotation of tibia

MRIs

- Be selective
- Asymmetric cases
- Other neurological features in foot
- Systemic signs of neurological disease

Genetic testing

- Screening for affected families

Nerve conduction studies and EMGs

- EMG more useful as motor nerves primarily affected
- Demyelination means conduction velocities are slowed

Management

Non-operative

- Footwear modifications and moulded AFOs, stretching physiotherapy
- Non-operative treatment does not alter course of disease
- These patients are often young. Treatment of choice is operative
- Can be used to delay surgery until deformities become symptomatic

Surgical treatment

- Aims of treatment are to achieve a plantargrade, stable foot that moves and is pain free
- Key to treatment is deciding how to correct the deformities depending upon their flexibility and degree of arthrosis
- Soft tissue or bony or both

First ray plantarflexion

- Dorsal closing wedge osteotomy

- Peroneus longus to peroneus brevis transfer
- Also corrects inverted forefoot

Cavus

- Plantar fascia release
- TP transfer to dorsum of foot through the IOM

Hindfoot varus

- Peroneus longus to Brevis transfer
- Lateral closing wedge calcaneal osteotomy (Dwyer)
- Lateral calcaneal slide osteotomy

Equinus

Usually corrects after other deformities corrected

TA lengthening left till all other corrections done to prevent over lengthening

Clawing

- Toes treated with Girdlestone Taylor split FDL to EDL transfer
- Combine with Weil's osteotomy if incomplete correction
- PIPJ fusions and Weil' osteotomies
- MTPJ release and PIPJ excision arthroplasty
- Big toe clawing
- Jones' procedure
- Big toe IPJ fusion and EHL transfer to first MT dorsum

Arthrodesis

- Triple arthrodesis is required to correct all deformities if fixed and painful
- Try to avoid as long as possible as patients are young
- Last resort

In reality

- Not all deformities are fully flexible or stiff
- A combination of corrective osteotomies and tendon transfers are used
- After all corrections patients often still need an orthosis to combat a weak tibialis anterior
- TP IOM transfer does help but may not be enough to combat drop foot deformity

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Chapter 19

Spine oral core topics

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Introduction

Spinal surgery is a diverse and rapidly evolving subspecialty within orthopaedic surgery, making it an exciting and rewarding career choice, yet a challenging area to review for the FRCS (Tr & Orth). It is a mandatory topic for assessment making it very difficult for exam candidates to be successful without a solid understanding of the core topics in this area. However, most examiners are not full-time spine surgeons and accept that most candidates are not going to become full-time spine surgeons either. The questions asked tend to be of one of two types: Either they are 'core topics' that any consultant orthopaedic surgeon should know about (and be able to manage appropriately or know when to refer on) or they are general orthopaedic questions that are being applied to the spine (e.g. the principles of bone grafting). In order to cover the breadth of material required, this chapter will aim to be succinct, cover core topics in sufficient depth to ensure a pass and provide the candidate with a framework with which to tackle spine questions. Areas that will be covered include:

- General knowledge
- Degenerative conditions (including the prolapsed intervertebral disc)
- Spinal trauma
- Tumours affecting the spine
- Infection and inflammation
- The paediatric spine
- Surgical approaches
- Other miscellaneous conditions
- Current areas of debate

General knowledge

Structure of the intervertebral disc

There are two main components of the intervertebral disc, the annulus fibrosus and nucleus pulposus (Figure 19.1). The annulus fibrosus consists of concentric rings of type I collagen. Sequential layers of oblique fibres resist hoop stresses and prevent excessive movement. The nucleus pulposus forms the gelatinous core, which allows elastic deformation. Type II collagen predominates in the nucleus pulpous. In adults, nutrition is via diffusion though the vertebral end plate. In children, vessels cross the endplate to the disc.

Age-related changes in the spine

The degenerative process that affects the spine is complex, it begins at an early age and is almost ubiquitous. It is often stated that the spine is the first part of the skeleton to age, with early degenerative change visible on cervical radiographs as early as the second decade of life. Men are affected more frequently than women and at an earlier age. The progression of the age-related change is relentless and affects all anatomical structures. The process begins within the intervertebral disc.

Increasing age is associated with dehydration of the intervertebral disc. This dehydration is associated with progressive histological, biochemical and biomechanical change. Histologically the boundary between nucleus pulposus and annulus fibrosus becomes less distinct and the organisation of collagen fibres less ordered. Biochemical changes include the loss of aggrecan and water from the extracellular matrix and an increase in the proteases responsible for enzymatic degradation. There is an increased keratin sulphate to chondroitin sulphate ratio. Fissuring and tears appear in the annulus fibrosis with degeneration and loss of orientation of collagen fibres.

These histological and biochemical changes lead to mechanical changes in the function of the intervertebral disc. Loss of disc height leads to instability within the motion segment, which in turn leads to compensatory calcification, osteophyte formation, hypertrophy and buckling of the ligamentum flavum. Facet joint arthrosis develops. Annular tears and disc herniation may occur. Progressive loss of disc height in combination with these changes leads to narrowing of the neural exit foramen, increased loading of the arthritic facet joints and narrowing of the spinal canal. This narrowing (stenosis) can be either central, involve the lateral recesses, or the neural exit foramen.

Within the spinal column altered biomechanics affect different regions in different ways. The support and relative stability provided by the thoracic rib cage cause the thoracic region to be relatively spared. In the more mobile cervical and lumbar regions degenerative change is more common. In the cervical spine the lower levels (C5/C6 and C6/C7) are most frequently affected. Likewise the lower lumbar motion

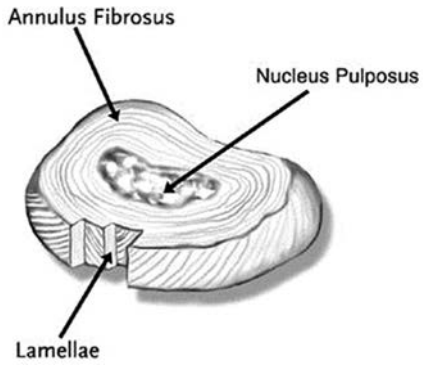

Figure 19.1 Structure of the intervertebral disc

segments are more frequently affected. Instability can progress and degenerative spondylolisthesis may occur. Degenerative spondylolisthesis is most common between the fourth and fifth lumbar vertebra.

The pathogenesis of these age-related changes is unclear, but a decrease in the nutrient supply to the disc is likely to be a key factor. The main pathway for nutrition to reach the intervertebral disc is via the vertebral body endplate. Permeability of the endplate to nutrients decreases with increasing age, thus, decreasing nutrient supply to the disc.

Biomechanics

The primary motion segment of the spinal column consists of two vertebral bodies and the intervening intervertebral disc. The combinations of flexion, extension and rotation in three planes produce six degrees of freedom. The most common biomechanical model used compares the spine to a crane with the vertebral bodies acting like the upright of the crane, the posterior spinal muscles acting as a posterior tension-band and the upper limbs acting like the arm of the crane suspending a load at a distance.

Bone graft

Any arthrodesis (fusion) procedure will require bone graft (e.g. posterior spinal fusion in scoliosis or spondylolisthesis). Autograft (the patient's own bone) and allograft (donated bone) are the two main types used, possibly supplemented by synthetic materials such as tri-calcium phosphate. Iliac crest autograft is considered the gold standard and has the best fusion rates but is associated with donor site morbidity. Allograft available as demineralized bone matrix avoids donor site morbidity, but is theoretically only osteoconductive rather than osteoinductive. Bone morphogenic protein is being increasingly used and may stimulate bone healing; however, it may be associated with increased rates of wound inflammation and its cost is prohibitive in some centres. Hydroxyapatite and demineralized bone matrix may also be used.

Spinal cord monitoring

Spinal cord monitoring is used in complex spinal surgery, such as deformity correction to detect early signs of neurological injury and to prevent devastating sequelae. Previously the Stagnara wake-up test was used to check spinal cord function. Intraoperative neurophysiological studies such as somatosensory evoked potentials (SSEPs) or motor-evoked potentials (MEPs) are used to monitor the spinal cord and detect early changes in spinal cord function that might be correctible such as early positional vascular or traction injuries. Stimulating a peripheral nerve using an overlying electrode produces SSEPs which test the posterior (dorsal) sensory columns by creating an afferent impulse that can be detected by recording electrodes over the sensory cortex. Motor tract status may be inferred from intact sensory tracts, but MEPs provide a better gauge of motor function. These are created by transcranial electrical stimulation of the motor cortex and recorded from a peripheral muscle.

Degenerative conditions

The lumbar spine – neurogenic claudication and radiculopathy

Neurogenic claudication and radiculopathy are the most common indications for spine surgery in older people.

Neurogenic claudication is a symptom complex that is caused by central lumbar spinal stenosis. It typically includes a reduction in walking distance due to bilateral leg pain (claudication), a feeling of heaviness, fatigue, aching, numbness and loss of function affecting the lower limbs. Symptoms are typically reduced by rest and bending forward. Bending forward flexes the lumbar spine reducing the lumbar lordosis increasing the space available for the cauda equina within the spinal canal. Activities that involve flexion of the lumbar spine (e.g. walking uphill, upstairs, pushing a shopping trolley and cycling) are frequently found to be easier than less arduous tasks that extend the lumbar spine (walking downhill, standing) which increase the lumbar lordosis.

One of the challenges for the clinician is to effectively distinguish a neurological cause for the claudication from a vascular one (Table 19.1). The characteristics of the symptoms described above will help, and clinical examination with palpation of peripheral pulses as well as ankle-brachial pressure recording is also useful. Standing relieves vascular claudication whereas neurogenic claudication may be exacerbated.

Neurogenic claudication is caused by central spinal canal stenosis. Facet joint hypertrophy, posterior bulging of the dehydrated intervertebral disc and thickening of the ligamentum flavum combine to reduce the space available for the nerves of the cauda equina within the canal. This degenerative process may or may not be associated with a degenerative spondylolisthesis that further narrows the central canal.

Degenerative spondylolisthesis, most commonly occurs at the L4/5 level. It may cause the symptoms described above as a result of central stenosis, it may also cause

Table 19.1 Neurogenic vs vascular claudication

	Type of claudication	
Symptom	**Neurogenic**	**Vascular**
Pain	Worse on standing	Relieved by standing
Numbness	Present	Absent
Site of pain	Leg/buttock/thigh	Calf (rarely anterior)
Relieving factors	Bending forward	Standing
Walking distance	Variable	Fixed
Worse going	Downstairs	Upstairs

symptoms by compressing nerve roots within the neural exit foramen or within the lateral recess. Exit foraminal stenosis will cause unilateral or bilateral *radiculopathy*. Different degrees of loss of disc height, osteophytosis, exit foramenal dimensions and central stenosis will determine the neurological structure compressed and, therefore, the symptoms experienced.

Radiculopathy is characterized by pain, paraesthesia, numbness and muscle weakness caused by injury to a specific spinal nerve or nerve root. Causes include lumbar disc herniations, lateral recess stenosis, foraminal stenosis, spondylolisthesis and other compressive pathology such as facet joint cysts. More rarely the pathology may originate within the nerve root itself (e.g. neurofibromas). Symptoms affect the dermatome and myotome of the injured nerve root. Thus, the pain felt on the outer aspect of the leg (L5 dermatome) originating from an L4/5 disc prolapse ('*referred*' pain) can be described as an L5 radiculopathy. The term 'sciatica' is frequently misused by patients and clinicians and it is perhaps best thought of as more general term. Sciatica describes pain in the distribution of the sciatic nerve and, therefore, encompasses the various different lumbar radiculopathies below the knee (L3, L4, L5 and S1). This is imprecise as the medial malleolus is innervated by the saphenous nerve which is a sensory branch of the femoral nerve (L4)[1]. The even older term 'lumbago' describes a severe low back pain that *radiates* round via the buttocks to the hips to the top of the thighs.

Investigations

MRI has become the gold standard for investigating these symptoms. Detailed images of the spinal canal in sagittal and transverse section can be used to assess the space available for neural structures. Its main disadvantage is that the investigation is performed supine which may cause a spondylolisthesis or other deformities to reduce or appear less severe. Standing x-rays may be helpful in this regard. Alternatives include CT myelography.

Lumbar disc herniation. The most common cause of lumbar radiculopathy is a herniation of the nucleous pulposus of an intervertebral disc beyond the normal limits of the annulous fibrosus. This typically affects those of working age (between 20 and 50 years). Disc herniation is the result of the degeneration of an intervertebral disc but its occurrence may be precipitated by a bending, lifting or jarring movement (coughing/sneezing). The natural history of most lumbar disc herniations is that of spontaneous resolution over time. A herniated disc is a localized displacement of nucleus pulposus beyond the normal limits of the disc. The clinical features and treatment options for disc prolapse vary depending on age and the location of the prolapsed disc.

Nomenclature

A disc prolapse can be broad based (involving between 25% and 50% of the disc circumference), focal (involves <25%) or symmetrical (involves 50–100% of the circumference of the disc). A focal disc herniation may be described as a protrusion or extrusion. An extruded disc has a narrow 'neck' at its base. Extruded disc material is 'sequestrated' if it is no longer in continuity with the disc[2]. A 'central' disc herniation is one that is located posteriorly, in the midline. Other locations include para-central, foraminal and far-lateral disc herniations.

In children the symptoms and signs of disc prolapse are less well defined and back pain is a more prominent feature. Nerve root tension signs are also less likely to be positive and spontaneous resolution is less likely.

Radiculopathy usually predominates over back pain and is often described as burning or electrical in nature, and is associated with paraesthesia and numbness. It is important to exclude non-spinal causes for back pain and exclude 'red flags' indicative of serious spinal pathology:

- History of trauma
- Age <20 or >55 years old
- Thoracic or abdominal pain
- Nocturnal pain
- Pain that is constant, progressive or non-mechanical in nature
- Constitutional symptoms (fever, night sweats, weight loss)
- History of malignancy, steroid use, drug abuse or HIV
- Persisting severe restriction of lumbar spine flexion
- Structural spinal deformity
- Widespread neurological abnormality

Investigation is with MRI but is not normally required in the acute setting unless there are features of cauda equina syndrome (bilateral lower limb radiculopathy, saddle anaesthesia, urinary and bowel symptoms). Initial treatment is non-operative with the provision of analgesia, including NSAIDs if not contraindicated. Consideration should be given to the short-term use of antispasmodics (a benzodiazepine). Neuropathic analgesics may be used. If conservative treatment has not been successful microdiscectomy may be considered and produces significant improvements in leg pain in >80% of patients.

Cauda equina syndrome

Cauda equina syndrome caused by compression of the cauda equina (usually by a large central low lumbar disc herniation) and is characterized by urinary retention, faecal incontinence, saddle area numbness and loss of anal tone. Bilateral radiculopathy, which is pathognomonic of cauda equina syndrome occurs when a central disc herniation is sufficiently large to compress nerve roots on both sides of the spinal canal.

The importance of detecting cauda equina syndrome early is that early intervention (<48 h) has been suggested to improve outcome. More recently the extent of the compression rather than the timing of surgery has also been linked to outcome.

Cauda equina syndrome can progress at a variable rate (hours, days or weeks), and patients present at various points along that process. A distinction may be made between cauda equina syndrome with symptoms of urinary disturbance (altered sensation or incontinence) but not retention (CES-I), and CES with retention (CES-R). CES-Incomplete patients may have subtle urinary difficulties including altered sensation, loss of the desire to void, poor urinary stream, stress incontinence or difficulty in initiating micturition. Saddle area sensory change may be subtle, unilateral or partial. Trigone (catheter-tug) sensation is be present. CES-Retention is characterized by complete loss of bladder sensation. Painless urinary retention results with overflow incontinence. There is extensive loss of sensation in the saddle area with absent trigone sensation.

The outcome of surgery for patients with CES-I is generally more favourable. Surgery aims to prevent progression to CES-R.

Outcomes of surgery for spinal stenosis and spondylolisthesis

There are many different surgical techniques that have been described for treating compressive lesions including interlamina decompression (most common), laminectomy, undercutting facetectomy, laminotomy, selective nerve root decompression and many others. All are aimed at relieving symptoms by relieving neural compression. In addition, where there is instability (spondylolisthesis), decompression may be supplemented with fusion. Whichever technique is undertaken treatment, it is aimed at removing the mechanical compression and the subsequent recovery of symptoms relies on recovery of the compressed neural structure. The outcome of surgery of this type is relatively predictable and significant improvements in leg pain, walking distance (and associated disability), and quality of life can be expected following surgery. Recent high quality data from prospective studies in a large number of patients have shown the clinically significant benefits of spine surgery over conservative treatment and the maintenance of these improvements in the medium term[3]. Patients undergoing surgery have shown significant improvements in pain, physical function and disability as assessed using validated patient reported outcome measures (SF-36 and Oswestry Disability Index).

Controversies and novel treatments

One of the main dilemmas that the spine surgeon faces when treating a degenerative spondylolisthesis is whether to fuse the spine in order to stabilise and prevent progression of the spondylolisthesis. Following the Cochrane review[4] demonstrating higher fusion rates with instrumentation this is usually undertaken using posterior screw and rod instrumentation with or without an anterior cage. In younger patients with higher demands fusion surgery may help prevent progression of the spondylolisthesis and the need for further surgery. However, in an older age group the spondylolisthesis rarely progresses and decompression without instrumentation avoids the complications associated with instrumented or non-instrumented fusion surgery.

Novel technologies including interspinous spacer devices are currently being evaluated but have not found wide acceptance. Typically they attempt to treat symptoms by flexing the spine at the symptomatic level, relieving symptoms in a similar but exaggerated way to bending forward. Although there have been some encouraging early results, the exact indications for such surgery have yet to be established.

Degenerative conditions affecting the cervical spine – Radiculopathy and myelopathy

Cervical spondylosis is a widely used but non-specific term referring to the generalized degeneration of the cervical spine frequently seen in older people although it can begin at an early age. Characteristic x-ray changes can be identified. Patients may present complaining of neck pain, radiculopathy or myelopathy. Neck pain caused by cervical spondylosis typically presents as episodic bouts of pain that resolve over days or weeks. Symptoms may be exacerbated by increased activity and may be associated with occipital headache. Treatment is largely conservative with rest and analgesia. Assessment is aimed at identifying and excluding surgically treatable conditions.

Cervical radiculopathy is typically caused by a compressive lesion affecting a single nerve root in the cervical spine. Compression can be caused by osteophytes, herniated disc fragments, facet joint hypertrophy and loss of disc height. It presents with unilateral neck pain, and pain radiating into the upper limbs in the distribution of the affected nerve root. These symptoms are associated with decreased or altered sensation and lower motor neurone signs in a similar distribution. The most commonly affected levels are the C6 and C7 nerve roots. The natural history of radiculopathy is benign with a significant proportion of cases resolving spontaneously or with conservative treatment. Its peak incidence is in middle age and this decreases with increasing age.

Radiculopathy affecting the C6 nerve root (exiting above the sixth cervical vertebra) will produce pain radiating to the radial side of the forearm and hand affecting the thumb and index fingers, decreased or altered sensation in a similar distribution with weakness of biceps (supination) and wrist extension. The brachioradialis reflex may be decreased.

Radiculopathy affecting the C7 nerve root will produce pain radiating to the long (middle) finger, decreased sensation in the same area, weakness of wrist flexion, elbow extension and a decreased triceps reflex.

Cervical myelopathy can be defined as spinal cord dysfunction due to compression of the cervical cord within the cervical spine. It is the most common type of spinal cord lesion in older people. The compression is commonly caused by osteophytes, ligamentum flavum hypertrophy and bulging or prolapse of the intervertebral disc.

Cervical myelopathy presents with upper motor neuron signs and symptoms in both upper and lower limbs. Initial presentations may be subtle but detection and treatment is essential before irreparable cord damage occurs. Typically cervical myelopathy follows a progressive stepwise deterioration with relatively stable periods and periods of rapid deterioration. It rarely resolves spontaneously. Symptoms include decreased coordination, loss of fine dexterity (e.g. buttoning a shirt, handwriting, manipulating small objects), balance and gait problems, and problems with bowel and bladder function. Balance and walking problems may lead to frequent trips, falls or bumping into things. There may be diffuse altered sensation and occasionally the sensation of shooting pains or 'electric shocks' radiating down the arms when moving the head in particular position.

Associated (upper motor neuron) signs include: A wide based unsteady gait, upper and lower limb weakness, hyperreflexia, intrinsic muscle waiting in the hand, positive Babinski and Hoffman signs and an inverted radial reflex.

Anterior cervical discectomy and fusion (ACDF)

ACDF is a key procedure in cervical spine surgery, providing excellent outcomes with a low complication rate. The anterior approach allows access to the cervical disc that can be removed along with osteophytes at the posterior aspect of the vertebral body. It allows removal of most lesions causing myelopathy or radiculopathy. Placement of anterior bone graft between the vertebral bodies in the excised disc space indirectly assists decompression of the exit foramen indirectly and facilitates bony fusion.

Complications that may occur include pseudarthrosis (increased in smokers), hoarseness and dysphagia as a result of retraction or injury to the recurrent laryngeal nerve (2–5%). This may also be caused by placement of the ET tube (more common). Graft complications also include the graft loosening and migration. Progression of the underlying disease process may cause degeneration of adjacent levels. Alternatively, fusion alters the mechanics of the cervical spine increasing the lever arms of forces acting at adjacent levels which may cause adjacent level degeneration.

Cervical disc replacement

Cervical disc replacement is an emerging surgical technique offering an alternative to ACDF. It is aimed at treating the same pathologies through the same anterior approach but attempts to preserve segmental motion potentially reducing rates of adjacent segment degeneration. Initial results are encouraging and appear to be at least comparable to ACDF[5]. However, this surgery does not prevent movement of the posterior facet joints as ACDF does. Posterior facet joint arthropathy is commonly associated with anterior degeneration. Although cervical disc replacement may improve pain from a cervical radiculopathy in the same what that an ACDF does it will not address pain from facet joint arthropathy which and ACDF might by preventing motion.

Posterior surgery – foraminotomy/laminoplasty

Posterior surgery may also be used in the cervical spine either to decompress a single nerve root (foraminotomy) or to decompress multiple levels. It has the advantage of being motion preserving and when surgery is required at more than two levels it is the treatment of choice. However, its disadvantage is that the surgery can potentially destabilise the spine posteriorly leading to a tendency to drift into kyphosis. Furthermore, as cervical spondylosis advances progressive loss of disc height anteriorly leads to a progressive loss of the normal cervical lordosis and may itself produce cervical kyphosis. To avoid this combination of risk factors kyphosis is a relative contraindication to posterior surgery in spondylotic patients.

Spinal trauma

Spinal fractures occur in 10% of all adult major trauma patients, although the incidence is probably higher as many minor or osteoporotic fractures presumably go unrecognised and untreated. Careful assessment and subsequent management of patients with potential spine injuries is essential to prevent spinal cord injury and maximise the potential for recovery. Patients suspected of having a spinal cord injury or unstable spinal fracture should arrive at their destination already immobilized on a spinal board, if this is not the case these measures should be put in place as soon as a spinal injury is suspected. Patients are kept immobile until a definitive opinion regarding spinal stability can be obtained. Standard spine trauma assessment should always proceed as per ATLS® principles.

Assessment

Airway (with cervical spine control), Breathing, Circulation, Disabilty (including neurological examination) and Exposure (including inspection of the back).

Life-threatening conditions take priority. An initial 5-second assessment for airway obstruction, massive haemorrhage or cardiac arrest is followed by a handover from prehospital personnel and then assessment of ABCD&E. The cervical spine needs to be controlled and protected until cleared clinically and radiologically. Sandbags, tape and hard collar immobilization on spinal board are typically used initially with the patient transferred off the spinal board as soon as is feasible due to the risk of development of pressure sores. A high index of suspicion is mandatory with high-energy injuries, head or facial injuries or an altered level of consciousness.

Hypovolaemic shock should be distinguished from neurogenic shock. Neurogenic shock is defined as vascular hypotension that occurs as the result of spinal cord injury. The cell bodies of the autonomic nervous system are located in the spinal cord grey matter. Injuries to the spinal cord above the mid thoracic level may affect the function of the autonomic nervous system, which results in loss of 'sympathetic tone'. This leads to peripheral vasodilation resulting in hypotension and if the lesion is above T2 there will also be bradycardia. Furthermore patients are unable to respond in a normal way to the hypovolaemia caused by other injuries. Neurogenic shock may be the only indication of spinal cord injury in an unconscious patient. Resuscitation should aim to restore cord perfusion without causing oedema. Neurological symptoms can be transient (fluctuating GCS) and if present suggest more serious underlying injury. Repeated neurological assessment is required to establish trends of improvement or deterioration. Bradycardia is suggestive of neurogenic shock and diaphragmatic breathing suggests cervical cord injury.

Spinal shock is defined as spinal cord dysfunction as a result of physiological rather than anatomical disruption. It is the result of swelling, oedema and inflammation and it usually settles in the first few days following spinal cord injury. The neurological dysfunction below the level of injury (e.g. absent reflexes) subsequently recovers with the resolution of spinal shock. The bulbocavernosus reflex recovers first. If a complete neurological deficit persists for >24 hours after spinal shock resolves the potential for recovery is limited. Incomplete deficits are more likely to recover.

Neurological examination

Assessment and onward communication can be greatly facilitated by the use of an ASIA chart. Structured scoring systems provide a reproducible template for serial evaluation. If a cranial nerve deficit is detected consider vertebral artery injury or high cervical fractures/dislocations. Neck or back tenderness is not always present. A palapable 'step' in the spinous processes may suggest a fracture or dislocation.

A 'complete cord' injury is defined as complete loss of motor and sensory function below the level of a spinal cord injury. An incomplete cord injury is defined as partial preservation of sensory or motor function below the level of spinal a spinal cord injury with sensory or motor function in the lowest sacral segment. Different patterns of incomplete injury have been identified. Sacral sensory sparing is important as it indicates an incomplete injury and the potential for improvement.

Brown–Séquard syndrome is an injury that affects one side of the spinal cord. It results in loss of ipsilateral motor function, conscious proprioception and contralateral pain and temperature sensation with decreased sensation to light touch. The prognosis is relatively good with 90% of people regaining bowel and bladder function and independent mobility.

Anterior cord syndrome has light touch and joint position sense proprioception preserved in dorsal columns with loss of motor function (movement), pain and temperature sensation from the anterior injury. The prognosis is more guarded with recovery only if it resolves within a short period (24 h).

Central cord syndrome is the most common incomplete spinal cord injury and frequently results from an extension injury in a spine with pre-existing degenerative change. The upper limbs are more affected than the lower limbs. Upper limb flaccid paralysis with hypertonic paralysis or preservation of function in the lower limbs is seen. Fifty-to-sixty per cent of patients regain lower limb function, but damage to the central synapses and cell bodies (in the grey matter) frequently result in poor hand function.

ASIA/Frankel grading system

A. Complete paralysis
B. Sensory incomplete – Sensory function but no motor function below the level of the injury
C. Motor incomplete – Motor function (MRC) grade 2 or less below level of injury
D. Motor incomplete – Motor function (MRC) grade 3 or above below the level of the injury
E. Normal function

MRC scale for muscle power

0. No muscle contraction is visible
1. Muscle contraction is visible but there is no movement of the joint
2. Joint movement is possible with gravity eliminated
3. Movement overcomes gravity but not resistance
4. The muscle can overcome gravity and move against resistance but is weak
5. Full and normal power against resistance

Clearing the spine

Local protocols will usually be in place. In order to clear the spine clinically (without radiological assessment) in a conscious patient the patient must be fully alert and orientated and not complaining of neck pain. There should be no associated head injury. There should be no involvement of sedative drugs or alcohol. There should be no other 'distracting' injury (long bone fracture). A complete neurological examination should be normal and be documented.

Under these circumstances the cervical collar can be removed and the neck examined carefully looking for signs of bruising, deformity, bony tenderness, reduced range of movement or pain on movement.

If none of these are present the cervical spine can be cleared and radiographic studies are unlikely to be required. If the patient complains of pain, radiographs in two planes of the C-spine from the skull to T1 are required. If radiographs fail to show a fracture and the patient continues to have moderate to severe cervical spine pain or tenderness, then the collar is left in place until voluntary flexion and extension radiographs or further imaging CT/MRI is undertaken.

Clearing the spine in unconscious patients remains controversial with recommendations ranging from removal of the cervical collar after 24 hours in patients with normal radiographs, to indefinite immobilization.

Radiological investigation

CT scanning is more sensitive than radiographs in two planes at detecting fractures and is becoming more frequently undertaken as part of the assessment of trauma patients. Head injuries and cervical spine injuries commonly occur together. Multiply injured patients will usually undergo a full trauma scan, but when thinking of ordering an isolated CT head in a patient with head injuries, a cervical spine CT scan should be requested as a routine. The remaining thoracic and lumbar spine may be adequately imaged either by AP and lateral plain radiographs or by sagittal and coronal reformatting of CT scans of the chest, abdomen and pelvis undertaken as part of a CT trauma series.

Oblique cervical spine radiographs (with the x-ray beam at 45°, patient supine, cassette on far side) show the pedicles and facet joints well. Dynamic imaging, including flexion/extension radiographs may be used to assess stability in the subacute setting but not initially. Other spinal x-rays are indicated when examination reveals a soft-tissue injury (e.g. a step in spinous processes, swelling or bruising), the patient complains of pain, or neurological deficit is detected. If one fracture is identified the whole spine should be imaged (10% have a second fracture). Other associated injuries (e.g. calcaneal fractures) may indicate a spine x-ray is required.

CT scans show laminar fractures, and retropulsed fragments better than x-rays. When viewing a CT canal size (and compromise) can be assessed by looking at the intact vertebral body. The canal is usually 0.8 times body diameter or more.

MRI also has its advantages. MRI is the urgent investigation of choice for spinal cord injury. Neural structures and soft tissues (including discs) are seen more clearly. Soft-tissue injuries can be identified form the high signal on T2 scans.

Classification of injury

After a severe trauma there are usually two aspects of the injury that require assessment the neurological and the osteoligamentous.

Neurological injury

This can be classified as 'complete' or 'incomplete' as described and graded above. Incomplete injuries include nerve root injuries or the syndromes described. Complete injuries have no function below a certain level. The 'level' of the injury is determined by the distal most level with intact sensory and motor function. Patients with spinal cord injuries are at risk of deterioration not only from progressive cord injury, but also from the dysfunction of other organ systems.

Positioning – Patients with spinal cord injuries should be nursed flat. The site of spinal cord injury is as a zone of critical ischemia. Loss of vasomotor tone means severe hypotension can result from sitting up a patient with spinal cord injury even after resuscitation. The hypotension that results from sitting a patient up can result in worsening ischemia to the injured spinal cord and brain and can adversely affect recovery.

Gastrointestinal system – Stress ulceration is common and consideration should be given to the prophylactic use of proton pump inhibitors. Paralytic ileus is common following all forms of major trauma. Bowel function is frequently affected by spinal cord injury. All patients should be kept nil by mouth initially. Once spinal shock has resolved, the use of regular suppositories and manual evacuation should be initiated early and monitored regularly.

Genitourinary system – Bladder function is commonly affected and prolonged retention with associated distension can lead to further injury to the bladder which is prevented by catheterisation. A catheter can also be used to assess trigone ('catheter-tug') sensation as part of neurological assessment.

Autonomic dysreflexia is a potentially life-threatening condition that can occur in patients with spinal cord injuries. The most common cause is an undetected blocked urinary catheter or bowel obstruction. Severe (paradoxical) hypertension and bradycardia result. Treatment is with removal of the initiating stimulus and pharmacological treatment with antihypertensives and vasopressors.

Skin – Pressure sores can develop rapidly owing to loss of protective sensation and the capacity for movement. Pressure area care should begin as soon as the patient arrives in hospital, with removal of the spinal board as soon as practicable. Regular turns from side to side (40°) with adequate support should be undertaken regularly and recorded.

Deep vein thrombosis – Thromboembolic disease is a substantial risk in immobile patients. This is now one of the most commonly reported causes of mortality in spinal cord injury.

Physiotherapy – Joint contracture as a result of loss of voluntary movement or spasticity can occur rapidly following injury. Regular passive range of movement exercises of all affected joints should be undertaken regularly. Resting splints should be provided overnight.

Osteoligamentous injury

The classification of the bony or osteoligamentous injury depends on the location of the fracture and will be discussed

below. The terms unstable and stable are often used. Denis classified the 'stable' fracture is one that will not deteriorate neurologically or structurally under normal physiological loads.

Management

The aim of management of a spinal injury is to minimize pain and maximise function. The risk of further neurological injury may be reduced by providing mechanical stability whilst at the same time facilitating other aspects of patient care.

The use of steroids has been widely debated, the theoretical concept being that a reduction in inflammation may reduce further injury to the spinal cord. Steroids are widely used in the context of brain and spinal cord tumours with good effect on symptoms and motor function. However, there is only limited evidence that they provide any benefit for fractures and treatment is not without morbidity. Several large studies (NASCIS 1, 2 and 3) have looked at the use of steroids in the early post-injury period. Unfortunately their methodology has been criticised, as has the interpretation of their results.

NASICS 1 – 1984. No difference in outcome was identified in the group that received steroids but subsequent animal studies suggested the dose of methylprednisolone was insufficient to see a benefit

NASICS 2 – 1990. An increased dose (30 mg/kg bolus then 5.4 mg/kg per hour for 23 h). No difference in initial analysis. A subgroup analysis showed a benefit in motor function at 6 months in those given the steroids within 8 hours of injury

NASICS 3 – 1997. Similarly required a subgroup analysis of those receiving the higher dose of prednisolone for a prolonged period (48 h) initiated between 3 and 8 hours to find a benefit

In a joint statement in 2013 The Congress of Neurological Surgeons (CNS) and The American Association of Neurological Surgeons (AANS) concluded *'Methylprednisolone should not be used for the treatment of acute spinal cord injury'*.

Specific injuries will be discussed below but in general terms decompressing, realigning and stabilising the spine will allow the best potential for early mobilization, prevention of complications and recovery of function. On the other hand, surgery is not without risk either and carries with it the risk of creating further injury including vascular injury. Management can be operative or non-operative and depends on the injury.

Upper cervical – occiput to C2

Occipital condyle fractures

These are rare injuries usually caused by axial compression on a laterally flexed neck or represent an avulsion injury. They are rarely detected in x-ray but are being identified more frequently with the increasing use of CT scanning for trauma. They are associated with potentially lethal trauma and are associated with other spinal fractures in 30% of patients. In the conscious patient occipital headache may be present although neurological examination may be normal. Dysfunction of cranial nerves IX–XII may be present but is hard to detect. This injury has been classified by Anderson and Montesano:

Type I comminuted undisplaced axial impact fracture

Type II continuous with base of skull fracture

Type III avulsion at the attachment of the alar ligament

Treatment for types I and II is conservative (cervical collar). Type III injuries are unstable and treated with halo vest or with occipito-cervical fusion.

Atlanto-occipital joint

Traumatic dislocations at this level are usually fatal. If a cord injury exists at this level most patients will die before reaching hospital as a result of respiratory compromise. More minor traumatic atlanto-occipital subluxations may reduce spontaneously. Rare survivors may have injuries to cranial nerves VII–X. Powers' ratio is the distance from the basion to the posterior arch of C1 divided by distance from the opisthion to the anterior arch of C1. Normal is 1, >1 suggests anterior dislocation, <1 posterior dislocation. Treatment is with Halo-vest immobilization. Traction is avoided as there is a risk of over distraction. Occiput-C1 fusion can prevent late displacement.

Atlas (C1) fractures

Injury to C1 is associated with other injuries in 50% of cases. Neurological injury is less common as there is a relatively large canal diameter at this level. Diagnosis is usually made on the trauma CT although it may also be detected on an 'open mouth' radiograph. Four types of C1 fracture are recognised:

Burst fractures involving the anterior and posterior arches (the 'Jefferson' fracture)

Isolated posterior arch fractures

Isolated anterior arch fractures

Unilateral lateral mass fractures

The lateral masses of C1 and C2 should be in line on imaging. If the combined displacement of the C1 lateral masses is 7 mm or more relative to C2 the transverse ligament is likely to be disrupted and the injury unstable. Stable fractures can be managed in a cervical collar for 12 weeks. Unstable fractures require an initial period of traction for 3 weeks or until reduction of the lateral masses is achieved and then a halo vest for a total time of 12 weeks. Occipito-cervical fusion may be required for unstable injuries.

Atlanto-axial instability

Atlanto-axial stability relies on the transverse ligament, which passes between the lateral masses of C1 behind the odontoid peg, and the alar ligaments, which connect the odontoid peg and the occipital condyles. Instability may be in AP translation or in rotation. Instability may result from a fracture of C1, an odontoid peg fracture, or rupture of the transverse and alar

ligaments. Radiological investigation will reveal an abnormally large atlanto-dens interval (5 mm or greater). MRI scan may reveal the ligamentous injury. Also, dynamic CT scans have been used to assess bony stability. Acquired instability may also arise in Down's syndrome or rheumatoid arthritis.

Rotational instability is more commonly seen in children and typically presents with neck pain, torticollis and a decreased range of movement. It has been classified by Fielding and Hawkins into 4 types based on the amount and direction of displacement. Type I–III C1 rotates anterior to C2 (<3 mm, 3–5 mm, >5 mm respectively) and type IV C1 rotates posteriorly.

Treatment is aimed at preventing further displacement in unstable injuries and symptom control in stable injuries. Stable injuries with intact transverse and alar ligaments are suitable for non-operative management in a rigid cervical collar or halo-vest for 6–12 weeks. In an adult with a transverse ligament injury that involves a large bony fragment, reduction may be achieved with traction and immobilization with a halo vest for 12 weeks. In the elderly, life-long conservative treatment may be an option for those in whom surgery is contraindicated.

In children with rotatory instability treatment is usually conservative. Many of these injuries in children reduce spontaneously and initial immobilization is relatively contraindicated as it may hold the subluxation in the displaced position. Rarely, if the injury does not reduce spontaneously gentle halter traction may be required.

A mid-substance tear of the stabilising ligaments will not usually heal and C1–2 fusion (with resultant loss in cervical rotation) is indicated.

When atlanto-axial instability exists care must be taken during intubation and the preoperative investigation of patients with rheumatoid arthritis with flexion and extension x-rays is routine. Advanced techniques such as fibre optic intubation are routinely employed.

Atlas (C2) fractures

Odontoid peg (Dens) fractures have been classified by Anderson and D'Alonzo into three types based on the location of the fracture:

Type I – Fracture of the tip caused by avulsion of alar ligaments (which connect the dens to occiput). These are treated symptomatically

Type II – Fracture of the base of the odontoid peg. This is the most common and most problematic type with a high rate of non-union. Typically these fractures displace with the C1 dens ligaments intact. Management is reduction and either posterior arthrodesis of C1–C2, or anterior (odontoid screw) stabilisation. Halo vest immobilization for 3 months may be used if displacement <5 mm and the patient is <50 years old

Type III – Fracture through vertebral body. Management is usually with a halo vest. A cervical collar may be used in elderly patients with stable fractures

Bilateral C2 pars fractures/C2 traumatic spondylolisthesis/Hangman's fracture

These are relatively common injuries usually caused by hyperextension in motor vehicle collisions. Hanging is now a rare cause! Thirty per cent are associated with other spinal fractures. Dysphagia and dyspnoea are recognised complications of a retropharyngeal haematoma. They have been classified by Effendi and modified by Levine and Edwards:

Type I – Minimally displaced (<3 mm no angulation). Caused by axial compression treatment is symptomatic with cervical collar

Type II – Significant angulation and >3 mm displacement. Caused by hyperextension with axial load treatment is traction or halo-vest immobilization

Type IIA – Angulation at the fracture sight without displacement. Resulting from a flexion–distraction injury these can displace further with traction which should be avoided. Treat with a halo vest

Type III – Anterior translation with uni or bi-lateral facet joint dislocations at C2/3. Attempted closed reduction or open reduction and stabilisation

'Subaxial' C3 to C7 injuries

These injuries have been classified by Allen and Ferguson and are based on the mechanism of injury. There are six mechanisms which are then subdivided into stages based the severity of the injury and the appearance of radiographs. The classification guides treatment. The three most common types are flexion–compression, flexion–distraction and compression in extension.

Flexion–compression injuries. This mechanism causes failure of the anterior column in flexion and the posterior column in distraction. There are five stages. Blunting of the anterior superior vertebral body (stage I), vertebral body beaking (stage II), beak fracture (stage III), retrolisthesis <3 mm (stage IV) and retrolisthesis >3 mm (stage V). Stages I and II may be managed conservatively in cervical collar or halo vest, other stages may require surgery and type V may require anterior and posterior surgery. Teardrop fractures of the anteroinferior endplate as it is driven into flexion by a compressive flexion injury should be distinguished from a benign osteophyte or avulsion fractures. Teardrop fractures are relatively unstable and may require stabilisation.

Flexion–distraction injuries (facet joint dislocations). This mechanism of injury causes failure of the posterior tension-band and progressive subluxation and dislocation of the facet joints. These are frequently missed injuries, oblique radiographs visualise the facet joints more clearly. Lateral radiographs may show anterior subluxation of the vertebra, soft-tissue swelling and the articular processes may overlap more. On an AP radiograph the spinous process may be deviated to affected side. The diagnosis is confirmed on CT. Stage 1 <25% subluxation of the facets, stage II unilateral facet joint

dislocation, stage III bilateral facet joint dislocation, stage IV is bilateral facet joint dislocation with displacement of the vertebral body the full vertebral width. All stages require reduction with traction (up to one-third body weight) and subsequent instrumented fusion. The general consensus is that awake reduction regardless of neurological deficit should be undertaken to monitor neurological function. With reduction there is a risk that an associated disc extrusion may cause cord compression and, therefore, a post-reduction MRI should be undertaken and a surgical team available on standby in case urgent ACDF is required. Anterior decompression and fusion is required when a discectomy has to be undertaken otherwise posterior fusion may be chosen. In type IV injuries both may be required (anterior to decompress and posterior to facilitate reduction with facetectomies and stabilisation).

Extension–compression injuries. These injuries cause failure of the posterior column in compression. There are five stages. Stage I is a unilateral vertebral arch fracture, stage II bilateral lamina fractures, stage III bilateral vertebral arch fractures with minor anterior displacement of the vertebral body, stage IV is further displacement of the vertebral body and stage V severely (complete). Types I–III can be treated with immobilization in a cervical collar or halo vest. Types IV and V are displaced fractures that are treated with posterior cervical fusion.

Vertical compression injuries. These injuries are divided into three stages. Compression of the vertebral endplate (stage I), compression and fracture of the vertebral endplate with minimal displacement (stage II) and displacement or fragmentation (stage III). Stage I and II are usually managed conservatively with a cervical collar or halo vest for 6–12 weeks. Stage III 'burst' fractures are caused by severe compressive load and are commonly associated with spinal cord injury and may require surgery. Surgical treatment may aim to decompress the canal and stabilise the spine. Decompression is most commonly achieved anteriorly as compression is frequently caused 'retropulsed fragments'. Stabilisation with fusion may be achieved anteriorly posteriorly or both.

Extension–distraction injuries. There are two stages. Stage I is failure of the anterior longitudinal ligament with vertebral body fracture treated with halo vest immobilization and stage II there is further displacement leading to injury of the posterior column which requires surgical stabilisation.

Lateral flexion injuries. There are two stages. Stage I is an undisplaced unilateral fracture, stage II is displaced with a contralateral ligamentous injury. Stage I can be treated with immobilization in a cervical collar, stage II required surgical stabilisation.

Soft-tissue injury (whiplash)

This area is controversial and not well understood. Patients may complain of a plethora of symptoms including pain and stiffness. Classically the patient presents following a low-speed motor vehicle collision, in which they have been the restrained occupant of a stationary car that has been stuck from behind by another car. There is considerable controversy regarding the pathophysiology and natural history of this condition. At the moment of collision the head is thrown back causing abnormal extension of the cervical spine. The recoil of the seat then throws the individual forward and as the torso is restrained by their seatbelt, and the cervical spine flexes. Many report persistent symptoms. Most patients who settle do so in first 3 months. Treat as for sprains elsewhere – Rest, NSAIDs and mobilization. Many cases result from RTAs and involve medico-legal claims.

Thoracolumbar spine trauma

This is the most common site of spinal injury (40–60% of all spinal fractures involve T12, L1 and L2). Amongst young patients, thoracolumbar fractures are usually the result of high-energy trauma, whilst fractures in the elderly may be caused by osteoporosis. Thoracolumbar fractures are often associated with abdominal trauma and 10–15% of thoracolumbar spinal injuries have significant visceral injuries.

Nomenclature

The title of this section points to a difficulty that currently exists with respect to nomenclature. Both the AO group and the Scoliosis Research Society define the term 'thoracolumbar' as referring to the region of the spine that is the junction between the thoracic and lumbar spine including T12, L1 and the intervening disc. However, most fracture classification systems use the term to refer to the whole thoracic and lumbar spine extending from T1 to L5 the majority of which involve T12, L1 and L2. The biomechanical environment from T1 to L5 changes considerably so the applicability of one classification system for all regions of the spine is perhaps doubtful.

Background

In 1931 shortly after the development of medically diagnostic x-ray, Watson Jones published the results of the system of treatment he had inherited from his mentor Sir Robert Jones in Oswestry. In the majority of cases flexion was thought to be the deforming force and extension (in a cast) was thought to reverse the force of injury, and reduce the fracture. Extension casting was proposed as the preferred method of treatment. Extension casting was used to try and correct the kyphosis and put tension on the anterior longitudinal ligament to help reduce anteriorly displaced fragments.

In 1948 Chance (a radiologist) described the appearances of a more unstable fracture in which the posterior bony elements were disrupted (separated/distracted).

In 1962 Holdsworth produced the first widely used classification system that incorporated the importance of the posterior ligamentous complex in the stability of fractures. Holdsworth's two-column theory of spinal stability divided the spine into the anterior column and the posterior

osteoligamentous complex. He suggested that the extension brace was not required for single-column anterior fractures, but also that it was ineffective at controlling posterior two-column injuries.

Classification systems

Three common classification systems are commonly in use. The Denis three-column theory of spinal stability is widely known and its terminology frequently used, but the usefulness of this classification system has been questioned. The AO/Magerl classification system is a detailed comprehensive classification following the usual AO ABC 1,2,3 system but its complexity limits its practicality. This classification has recently been updated. The Thoraco-Lumbar Injury Classification and Severity Score (TLICS) system is perhaps the most useful as it guides treatment.

In 1983, following the advent of CT, Denis produced a three-column theory of spinal stability. Denis defined the middle column as lying between the posterior ligamentous complex and the anterior longitudinal ligament, comprising of the posterior wall of the vertebral body, the posterior longitudinal ligament and posterior annulus fibrosus. He concluded that the mode of failure of the third column correlated with both the type of fracture and neurological injury. Burst fractures were reported as resulting from failure of the vertebral body under axial load. The key concept of the classification is that burst fractures in which the middle column is disrupted have failed in axial load and, therefore, cannot be stabilised with an external support (extension brace), and are, thus, thought of as being unstable under axial load. The radiological features of widened pedicles, loss of height and cortical integrity of the posterior vertebral body on the lateral x-ray, and retropulsed fragments seen on CT scan are described. The usefulness of this classification has been brought into question as many burst fractures are mechanically stable and are well-managed non-operatively.

In 1994 following the advent of MRI, Magerl et al. proposed a 'comprehensive' classification of thoracolumbar fractures. The authors proposed a classification system based on the familiar AO system of A, B and C types with 1, 2 and 3 subgroups further divided by 1, 2 and 3 sub-subgroups. The system is based on fracture morphology and the severity of the injury increases from A1 to C3. This AO classification has recently been simplified and updated by Vaccaro and adopted by AO[6] with the inclusion of a neurological score and slight changes to the classification of both A and C type injuries.

A – Compression injuries. In these injuries the anterior structures fail under compression. Minor non-structural (A0) fractures are fractures that are not associated with instability (e.g. transverse process). Wedge compression fractures (A1) are the most commonly encountered spinal fractures. The posterior elements are intact, but the anterior vertebral body fails in flexion and compression. Spinal cord injury is uncommon. These are usually stable injuries when there is a kyphosis of $<30°$ or loss of vertebral body height $<50\%$. Pincer fractures (A2) are fractures in which both end plates fail but both the posterior and anterior walls of the vertebral body remain intact. Incomplete burst fractures (A3) are fractures involving the posterior bony wall but only one endplate. Burst fractures (A4) occur when the entire vertebral body fails under compression. The radiological features of burst fractures include widened pedicles on the AP view and loss of vertebral height/cortical integrity of the posterior vertebral body on the lateral radiograph.

B – Distraction injuries. In these injuries the posterior or anterior tension-band fails in tension. As previously described Chance fractures (B1) are characterized by failure of the posterior bony elements in tension. Soft-tissue disruption of the posterior ligamentous complex (B2) is important because it results in instability that is not easily visible on plain radiographs. Hyperextension injuries may result in failure of the anterior tension-band.

C – Displacement or dislocation injuries. These severe injuries are rare and represent dissociation between the cranial and caudal sections of the spine. They are commonly associated with spinal cord injuries.

The score is completed by recording the patient's neurological status:

N0 – Neurologically intact
N1 – Transient neurological deficit, which is no longer present
N2 – Radicular symptoms
N3 – Incomplete spinal cord injury or any degree of cauda equina injury
N4 – Complete spinal cord injury
NX – Neurological status is unknown due to sedation or head injury

In 2005 Vaccaro et al. published the TLICS score emphasizing the importance of the integrity of the posterior ligamentous complex and neurological status (Table 19.2). This scoring system aids surgical decision making by indicating the likelihood of instability and the requirement for surgery. In the absence of strong outcome data it is based on the expect opinion of a panel of authors/contributors. The classification is based on injury morphology, the integrity of the posterior ligamentous complex and the neurological status of the patient producing a score out of 10. Injuries that score 5 or more are treated surgically and 3 or less conservatively.

As the fracture becomes more unstable and the potential benefit of neurological decompression increases the score increases.

Imaging

Radiographs – AP and lateral radiographs of the spine reveal most information pertinent for planning treatment and are mandatory. CT is best for assessing fracture patterns and spinal canal dimensions. MRI is excellent for assessing the posterior ligamentous complex, neural structures and cord injury.

Table 19.2 Thoraco-Lumbar Injury Classification and Severity Score (TLICS)

Morphology	Neurological status	Integrity of PLC
Compression – 1	Intact – 0	Intact – 0
Burst – 2	Nerve root – 2	Indeterminate – 2
Translation/rotation – 3	Complete cord injury – 2	Injured – 3
Distraction – 4	Incomplete cord injury – 3	
	Cauda equina injury – 3	

Treatment

AO type A1

These 'wedge compression' injuries affect the anterior vertebral body (anterior column). Most follow minor trauma and are associated with osteoporosis. These injuries are stable and rarely require surgical stabilisation or involve neurological injury. Traditional treatment is symptomatic with bed rest, analgesia and subsequent mobilization. More recently NICE has recommended vertebroplasty and kyphoplasty as possible treatment options for some people with spinal compression fractures caused by osteoporosis[7]. Kyphoplasty and vertebroplasty are both associated with a reduction in pain in the short term. The long-term outcome remains unclear and these procedures may be associated with an increased adjacent level fracture risk (possibly as a result of increased vertebral stiffness, possibly as a result of disease progression).

AO type A3 & A4 'burst fractures'

With burst fractures there is disruption of the anterior and middle columns and frequently retropulsion of fragments into the spinal canal. If the posterior elements are involved, 50% have neurological injury. No studies have found a direct correlation between the percentage of canal occlusion and neurological injury. Management depends on mechanical stability and neurological injury (as assessed using the TLICS score). Conservative management has been advocated in neurologically intact, mechanically stable patients. With conservative treatment long-term back pain is usually mild and there is unlikely to be any neurological deterioration. Retropulsed bone may reabsorb or remodel. Alternatively posterior fixation may improve mechanical stability, restore alignment and indirectly reduce retropulsed bone by distraction on the posterior longitudinal ligament. Complications with surgery include recurrence of kyphosis, a (low) risk of neurological injury with pedicle penetration or over distraction. Some authors advocate anterior decompression as a more complete clearance of the canal can be achieved at the same time as reconstructing the anterior column and avoiding further injury to the posterior tension-band.

Dislocations (AO type C)

These are unstable injuries with all three columns involved. They are often associated with severe neurological deficit, dural tears and other visceral injuries. Surgical treatment is to realign and stabilise the vertebral column. Careful attention to basic patient care (pressure area care, nutrition, etc) is required to prevent subsequent injury. Early mobilization (either independent or assisted) helps prevent complications of recumbency.

Timing of surgery

Emergency decompression is indicated for progressive neurological deficit or when a cauda equina syndrome is present. In the neurologically intact unstable injury operate once other conditions allow. Where there is a complete cord injury delay surgery until oedema settles (48 h) as early decompression does not improve results and may be associated with a risk of hypovolaemia causing further ischemia to the already injured cord.

Sacral fractures

These injuries are often overlooked but may represent severe injuries. Twenty-five per cent are associated with distressing neurological complications including incontinence or sexual dysfunction. Sacral fractures may be associated with pelvic fractures and spino-pelvic dissociation.

They have been classified vertical into three types (Denis) affecting three zones. Zone 1 injuries are lateral to the sacral foramina. These fractures may be associated with L5 nerve root injury. Zone 2 injuries run thought the sacral foramina and 15% have sacral nerve root injuries. And Zone 3 injuries are medial to the foramen and 30–50% are associated with nerve root injury. Transverse fracture lines produce H-type and U-type fracture patterns, which are highly unstable injuries spino-pelvic dissociations. The strong sacrospinous and sacrotuberous ligaments hold the distal part of the sacrum fixed to the pelvic ring, while the superior part of the sacrum and spine rotates out of the pelvic ring, resulting in the fracture. A subclassification (Roy Camille) divides these injuries further into type 1 angulated fractures, type 2 angulated and displaced, type 3 complete (100%) translational displacement and type 4 comminuted. Indications for surgical treatment include displacement of >1 cm or fractures associated with neurological injury (decompression and stabilisation). Surgical requires fixation of the lumbar spine to the pelvis with sacroiliac screws.

Tumours

Extradural tumours

Introduction

Metastatic disease is the most common form of skeletal tumour, and the spine is the most common site for skeletal metastasis with the most common primary tumours being breast, lung, prostate, renal, thyroid, the GI tract, and Myeloma. Primary spinal tumours occur but are much less common. The role of spine surgery is dependent on the aetiology, stage and grade of the lesion. Curative resection is possible in a few cases but is the least common indication for spinal surgery. Palliative resection for the prevention of (or prevention of progression of) neurological impairment is a common reason for surgical intervention. Pain from bony destruction and resultant mechanical instability may respond to surgical stabilisation. The most common presenting complaint is pain, which can either be bony (mechanical) or occur as the result of neural compression. Muscle weakness may occur as a result of neural compression and can be detected in up to 50% of cases of metastatic disease.

Assessment

The assessment of a patient with a spinal tumour follows the general pattern of ***staging, grading***, (multidisciplinary) assessment and treatment. Investigation will begin with history (paying particular attention to 'red flags'), examination and imaging studies. Examination should include breast, thyroid, respiratory, abdominal and rectal examinations and a test for faecal occult blood. Blood should be sent for inflammatory markers and tumour markers such as serum plasma electrophoresis and PSA.

Radiological investigations should include ***local and distant imaging***. Local imaging should include plain radiographs and a whole spine MRI (looking for neural compression and the extent of spinal involvement). A CT may be required for detailed bony anatomy if resection is being considered. Distant imaging depends on the likely pathology and may include a bone scan (looking for evidence of other skeletal metastases), a chest x-ray, or a CT scan of chest, abdomen and pelvis to search for a primary tumour or visceral metastasis.

Histological grading requires a biopsy. As tumours and infection are occasionally difficult to distinguish, ensure that samples are also sent for culture: *'Biopsy all infections and culture all tumours'.*

Some basic facts about the epidemiology of spine tumours help significantly when staging the tumour. With increasing age metastatic disease becomes more common. Vertebral body lesions are more likely to be malignant and posterior lesions benign. Under the age of 21 most spinal tumours are benign, over 21 most are malignant. Under the age of 3 metastatic malignant tumours become more common again. The thoracic spine is the most commonly affected region and the cervical spine the least.

The scoring system proposed by Tokuhashi[8] is useful in establishing indications for treatment and subsequent surgical approach. A poorer prognosis is correlated with a lower score. Six parameters are given a score (0–2). A score of <5 indicates a life expectancy under 1 year and a palliative approach is suggested. A score of over 9 indicated a longer life expectancy and suggests excision should be considered.

General condition (poor 0, moderate 1, good 2)

Number of extra-spinal metastases (3 or more scores 0, 1 or 2 scores 1, 0 scores 2)

Number of spinal bony metastases (3 or more scores 0, 2 scores 1, 1 scores 2)

Number of metastases to major internal organs (unremovable 0, removable 1, no mets 2)

Tissue of origin (lung, stomach 0, kidney liver uterus 1, other, breast, thyroid, prostate, rectum 2)

Spinal cord palsy (complete 0, incomplete 1, none 2)

If a malignant lesion is suspected the next step in assessment will be to biopsy the lesion to obtain a tissue diagnosis and histological grading. This biopsy should be done within the unit that will treat the tumour and also sent for culture.

Surgical treatment

Surgical resection of tumour may be aimed at improving survival (resection) or palliating symptoms by decompressing neural structures and stabilising the spine. This may be undertaken anteriorly or posteriorly or both depending on the size and location of the lesion and the goal of surgery. In general terms if a curative resection is hoped for or survival is likely to extend beyond 6 months, intervertebral bony fusion should be undertaken to avoid instrumentation failure. If life expectancy is short and a palliative procedure is being considered, fusion may not be required and posterior surgery is more commonly undertaken.

Following surgery patients can often expect functional improvement, pain relief, and in a few cases, cure. Surgery is increasing being performed. NICE have issued clinical guidelines on the treatment of metastatic cord compression[8]. Most large centres now have metastatic spinal cord compression services and multidisciplinary teams with surgeons, oncologists, haematologists and radiologists to decide the best treatment for each individual case. Decompression (with stabilisation) of compressed neural structures may lead to functional improvement even with prolonged paraplegia. If posterior surgery is being performed, a simple laminectomy to 'decompress' the tumour is contraindicated as progression of the tumour (most frequently in the vertebral body), will lead to mechanical instability and kyphosis. Decompression and stabilisation is frequently undertaken.

Radiotherapy

Although surgery has an increasing part to play in the treatment of spinal tumours, many malignant tumours respond to radiotherapy. Radiotherapy may be used to reduce tumour

bulk and treat pain in situations where there is neurological compromise without significant vertebral collapse. Tumour type, neurological status and radiosensitivity determine outcome. Prostate and lymphoreticular tumours respond best. Approximately 70% of breast tumours are radiosensitive. GI and renal tumours are often resistant to radiotherapy.

Minimally invasive surgery and vertebroplasty

Some patients with malignant disease and associated co-morbidities are too unwell or are unwilling to consider resection or major surgery. Surgery may also interrupt chemotherapy or radiotherapy regimens. When there is pain caused by instability minimally invasive surgery may allow the surgeon to stabilise the spine whilst minimizing soft-tissue trauma facilitating a faster postoperative recovery in patients with limited life expectancy. Fusion may not be required as the strength of the implants is likely to outlast the demands of the patient.

Vertebral body augmentation with high viscosity cement (PMMA) may be considered at the same time as skeletal biopsy and may improve pain in patients where decompression and instrumentation is not required.

Specific tumours

Benign lesions include:

Haemangioma are slow growing, commonly asymptomatic and often detected as an incidental finding on imaging. They appear lytic on x-ray and may cause pain from loss of mechanical support.

Osteoid osteoma/osteoblastoma are usually found in the posterior neural arch. Most present with the typical (NSAID sensitive) pain. Excision is curative but treatment with an NSAID may be all that is required.

Osteochondroma are most commonly found on the spinous process (related to apophysis). Excision is for symptomatic treatment. Sarcomatous change has been described and excision is indicated if a large (>10 mm) cartilage cap is seen on MRI.

Aneurismal bone cysts typically affecting the posterior elements and Giant cell tumours (affecting vertebral the body) are also seen.

Malignant lesions include

Most commonly malignant disease is metastatic.

Solitary plasmacytoma/multiple myeloma typically presents with pain and can be treated with radiotherapy (highly sensitive), surgical excision (solitary) or stabilisation.

Chordoma is locally aggressive and may present with compression of pelvic contents.

Lymphoma most commonly occurs in the elderly (mean age 85) and more frequently in men than women.

Chondrosarcoma typically presents with pain and x-rays may show typical matrix calcification.

Osteosarcoma presents in the young (<20) it is rare and survival is poor (median survival 6–10 months).

Intradural tumours

In contrast to extradural tumours most intradural tumours are not metastatic. It is useful to divide intra dural tumours into 'extramedullary' and 'intramedullary' groups.

Extramedullary tumours

Extramedullary tumours occur inside the dura but outside the spinal cord. They are usually benign. They cause symptoms by compressing neural structures, which can lead to pain or loss of motor function. Examples include neurofibromas, schwanoma (of dorsal sensory roots) and meningioma.

Intramedullary tumours

Intramedullary tumours occur within the spinal cord. Most are malignant. Examples include astrocytomas (affecting children), ependymomas (affecting adults) and, rarely, haemangiomas.

Infection and Inflammation

Infection

Spinal infections are relatively common and can affect the disc (discitis), the vertebrae (osteomyelitis), the spinal canal (epidural abscess), or affect the soft tissues surrounding the spine (psoas abscess). Symptoms can be severe and prolonged and there is the risk of severe neurological injury with epidural abscess or kyphosis. Diagnosis is often delayed. Pain may be vague and poorly localized and often other infections coexist. There is a change in the pattern of infection with age (50% occur in the over 50s). The intervertebral disc (like the meniscus of the knee) is vascular in younger children. In the neonate intraosseous, vertebral arteries anastomose with the adjoining disc through the vertebral end plate. With increasing age these arterioles within the end plate involute and the disc loses its vascularity. Discitis is, therefore, more common in younger children and vertebral osteomyelitis more common in adults. Risk factors include surgery, intravenous drug use, diabetes, steroid use, chronic infection and other immunocompromised states. Most infections are caused by *Staphylococcus aureus* or *Streptococcus*.

Tuberculosis (TB) varies in incidence geographically and with socio-economic factors and is increasing in incidence. TB mimics other infections and affects the disc late. Vertebral collapse and spinal deformity is more common than in other forms of infection. The anterior longitudinal ligament may be lifted off adjacent vertebrae. Extensive debridement and reconstruction is often required especially when there are neurological abnormalities. Antituberculous therapy should be given at the same time as surgery. Antituberculous therapy may be given alone when there is no deformity or neurological compromise. Neurological injury can recover as late as 9–12 months post-decompression.

Investigations

MRI with gadolinium contrast is the investigation of choice. It is non-invasive and both sensitive and specific (96% sensitive,

93% specific). Technetium bone scans are 90% sensitive. Plain radiographs do not usually show changes for several weeks. Discitis has a low signal on T1-weighted images and increased (high) signal within the disc is seen in T2-weighted images. There may be loss of endplate definition. Blood cultures may help obtain an organism (especially if pyrexial). Tissue for culture can be obtained with a needle biopsy (pathology specimens can be sent at the same time). Treatment can be monitored with ESR CRP and WBC.

Management

Treatment of osteomyelitis and discitis is initially conservative with rest and targeted intravenous antibiotics (high dose intravenous antibiotics for 6 weeks or until CRP normalizes and then oral antibiotics for 6 weeks or until there are no signs of infection). Bracing may provide pain relief. Surgical debridement may be required if there is no improvement on antibiotics, progressive vertebral collapse and deformity or the presence of neurological injury. Perhaps counter intuitively, spinal stabilisation can have a role to play as it has a beneficial effect on infection. Non-operative management does not usually improve neurology. Decompression is indicated for the treatment of an abscess. Radiological decompression may be an option for a soft-tissue abscess. Most epidural abscesses should be decompressed surgically, particularly if associated with abnormal neurology as neurology may improve dramatically after decompression.

Rheumatoid arthritis

The cervical spine is frequently affected in rheumatoid arthritis. However, since the advent of DMARDs and anti-TNFs, there has been a significant reduction in the amount of rheumatoid spinal disease encountered.

Disease progression causing erosion of bone and soft tissue with subsequently instability may lead to symptoms of neck pain, stiffness occipital headaches and the gradual onset of a cervical myelopathy. Neurological symptoms may be caused by bony compression of neural structures and compounded by pannus. Three main forms of instability are observed. Atlantoaxial instability is the most common and may lead to myelopathy. Later cranial settling (basilar invagination) may occur compressing the upper cord and brainstem. Subaxial instability is also observed. It has been classified by Ranwat:

I. Pain, without neurological deficit
II. Altered sensation, subjective weakness, hyperreflexia
IIIA. Objective weakness, early myelopathy
IIIB. Objective weakness, advanced myelopathy non-ambulatory

Assessment

Initial assessment is with plain radiographs including flexion/extension views to assess stability.

MRI is required to assess the soft tissues and spinal cord. Most patients have pannus visible around the odontoid peg.

Management

Management is initially conservative management with medical therapy and supportive bracing. Surgery is reserved for cases of intractable pain, progressive instability or neurological deterioration. The goal is to prevent further neurological deterioration. The atlanto-dens interval (ADI) and space available for the cord (SAC) are assessed on lateral flexion and extension lateral radiographs. Instability can be defined as a change of >3.5 mm in the ADI between flexion and extension views but in itself is not necessarily an indication for surgery. If the change is >10 mm difference in ADI there is an increased risk of neurological injury and, therefore, an indication for surgery.

The SAC measures the distance between the posterior neural arch and the posterior aspect of the dens. An SAC of <14 mm associated with increased risk of neurological injury and is an indication for surgery.

Usually a posterior approach is used. Subaxial disease is mainly treated with posterior instrumented fusion at the affected level(s). Subluxation should be stabilised before surgery with the patient awake. Posterior C1–C2 fusion is indicated is the change in ADI is >10 mm the SAC is <14 mm or there is progressive myelopathy. Adding transarticular screws may reduce the need for postop halo immobilization. Posterior occipitocervical fusion with resection of the C1 posterior arch is indicated if there is basilar invagination. Fixation may allow pannus to shrink. Complications include problems with wound healing and pseudarthrosis.

Ankylosing spondylitis

Ankylosing spondylitis is a chronic autoimmune seronegative spondyloarthropathy affecting the spine and sacroiliac joints. Orthopaedic manifestations include bilateral sacroiliitis, progressive spinal kyphotic deformity, cervical spine fractures, large-joint arthritis (hip and shoulder) and spinal deformity. It typically begins in the second decade of life. It affects men more severely than women and has a prevalence of approximately 1 in 1000. Serum HLA-B27 is positive in 80–90% of patients Rheumatoid factor is typically negative. A definite diagnosis of ankylosing spondylitis is made using the 'New York' Criteria. This requires the presence of sacroiliitis visible on imaging and two of three clinical criteria:

Low back pain and stiffness for >3 months that improves with exercise but is not relieved by rest
Limitation of motion of the lumbar spine in both the sagittal and frontal planes
Limitation of chest expansion

On radiographs the sacroiliac joints are affected first, followed by the progressive appearance of syndesmophytes (vertically orientated) and eventually fusion with the bamboo spine. Squaring of the vertebral bodies is seen. Other systemic manifestations include anterior uveitis & iritis, heart disease, pulmonary fibrosis, renal amyloidosis, aortitis, aortic stenosis, regurgitation as well as *Klebsilella* pneumonia. Classically the

whole spine may be affected with the 'question mark' posture. The cervical spine may be fixed in flexion (chin on chest deformity if severe). Flexion (kyphosis) is also seen in the thoracic and lumbar spine as well as the hips. If this is severe patients cannot see ahead whilst walking.

Treatment

Anti-inflammatory analgesics are the first choice of treatment combined with physiotherapy. The role of physiotherapy (in particular posture education) is essential as it helps prevent deformity as the disease progresses and fusion occurs reducing the requirement for osteotomy.

Spinal osteotomy may be considered using sagittal balance and in particular the 'chin-brow angle' to determine the amount of correction required. Address hip and lumbar deformities first. Cervico-thoracic osteotomy may also help but carries the highest risk.

Fractures of the ankylosed spine are common and may be missed on plain radiographs. Most fractures occur at the mid-cervical level or at the cervicothoracic junction. Frequently fractures are unstable involving 'all three columns'. Long lever arms mean the risks of subsequent (late) neurological deterioration are high. Likewise surgery is associated with increased risk of complications including instrumentation failure and deformity progression. Fusion should be undertaken in the pre-fracture position as the fracture may result in deformity correction (in a similar way to osteotomy) but is not controlled and is likely to result in neurological consequences. Strong constructs with multiple fixation points are required. Sudden onset new pain (especially if associated with correction of deformity) requires investigation to rule out a fracture. Long lever arms mean injuries are likely to be unstable and pseudarthrosis more likely. CT and MRI are very useful imaging modalities to detect a fracture if one is clinically suspected.

Large joint arthropathy can be treated with arthroplasty but if this is to be the case this should be undertaken after any spinal osteotomies as the sagittal realignment may change the relative orientation of stem and acetabular component.

The paediatric spine

The spectrum of spinal disease seen in the paediatric population differs from that seen in adults. Spinal deformity and back pain are the most common presenting complaints. The investigation of back pain in children requires a different approach from that in adults as the number and frequency of significant pathologies demands a higher index of suspicion and more detailed investigation.

Back pain

Common causes for back pain in children and adolescents include:

- Disc herniation
- Spondylolysis and spondylolisthesis
- Scheuermann's disease
- Infection
- Tumour

When investigating the cause of back pain in a paediatric population the age of the patient can help narrow the diagnosis. Children under the age of 10 are more likely to suffer from infections or tumours whilst in older children disc herniation, spondylolysis and Scheuermann's disease are more common. Also lower back pain becomes more common with increasing age.

Investigation depends on the suspected pathology. Inflammatory markers provide a serological assessment for infection. Standing plain radiographs may detect spondylolysis or spondylolisthesis. An MRI scan is likely to detect a disc herniation, infection, but may miss spondylolysis or early spondylolisthesis as it is performed supine. MRI may also detect intra-spinal anomalies such as spinal dysraphism or spinal tumours. CT scanning provides greater bony detail. A bone scan may be used when other investigations have failed to confirm a diagnosis.

Spondylolysis and spondylolisthesis

Spondylolysis is a defect in the pars interarticularis. Bilateral defects may allow an anterior slip of one vertebral body on the next most caudal level (spondylolisthesis). It most frequently affects the L5 vertebra at the L5/S1 level and is most frequently detected in adolescents and different aetiologies have been suggested including repetitive micro trauma. It is more common in gymnasts and other athletes in which spinal extension is repetitive (cricket, bowling). There are also racial differences in incidence (Inuits 25%, Whites 6%, African 2–3%). Patients typically present with back pain made worse by extension. A palpable step may be present. There may be leg pain and neurological signs if a spondylolisthesis is present. Hamstring tightness may also be present. In adolescents a spondylolytic spondylolisthesis is most commonly seen. The types of spondylolisthesis have been classified by Wiltse et al. (1976)[9]:

I. Dysplastic
II. Isthmic
    A. Spondylolytic (pars fracture)
    B. Pars elongated/attenuated
    C. Acute fracture (other than pars)
III. Traumatic
IV. Degenerative
V. Pathologic
VI. Iatrogenic

Treatment initially is with analgesia and activity modification. Bracing and physiotherapy (hamstring stretches) may also be considered. Follow up until skeletal maturity is required to ensure spondylolisthesis doesn't occur. Surgery may be considered after 6 months if pain persists or there is evidence of progression to spondylolisthesis. Different surgical treatments have been described including decompression and posterolateral fusion, repair with Scott wiring or Buck screw fixation.

If a spondylolisthesis is present management depends on pain and the grade of the slip. The Meyerding classification divides spondylolisthesis into grades I–IV with each representing a slip of 25% of vertebral body diameter and grade V spondyloptosis.

Grade I or II spondylolisthesis is treated initially with analgesia and activity modification in a similar way to a spondylolysis. Pain, slip progression and neurological symptoms are indications for surgery (decompression and in situ posterolateral fusion). An acute presentation of a high-grade slip (spondylolytic crisis) is an indication for urgent surgical stabilisation. The most common complication of surgery is L5 nerve root injury associated with attempted reduction.

Disc herniation

The presentation of disc herniation in children is somewhat different to that in adults. Back pain is typically the dominant symptom and radiating neurological symptoms are less common. A coronal imbalance or 'list' may be present on examination together with positive nerve root tension signs. Focal neurological signs may also be absent. An MRI scan is the investigation of choice. Treatment is initially non-surgical but symptoms may persist requiring surgical treatment.

Scheuermann's disease and kyphosis

Scheuermann's disease was first described using lateral radiographs and defined as anterior wedging of >5° in three consecutive vertebrae. The cause in unknown but deformity results from abnormality of the ring apophysis of the vertebral end plate. Patients typically present with pain and (if severe) deformity. The normal thoracic kyphosis is approximately 20–45°. Treatment depends on the severity of symptoms and degree of the deformity. Non-surgical treatment includes analgesia, physiotherapy and activity modification and pain may settle as growth slows towards skeletal maturity. With severe curves (>75°) surgery may be required to correct the deformity. Bracing may be effective at controlling progression of a kyphosis until skeletal maturity but compliance is poor.

Scoliosis

Scoliosis (derived from the Greek *Skolios* meaning crooked) is a term that describes a deformity of the spine in coronal plane. It is defined as a lateral curvature that measures >10° using the Cobb method. When present it usually forms part of a three-dimensional spinal deformity. The 'side' of a scoliosis is the side to which the spine deviates away from the midline, it is the side of the convexity of the curve. The Cobb angle defines the magnitude of the curve with minor (small) curves measuring between 10° and 25°, moderate curves between 25° and 50° and severe (large) curves measuring over 50°. Scoliosis is also described by the region of the spine that it affects. A 'thoracic' scoliosis has its apex between T2 and the T11–T12 disc, a 'thoracolumbar' curve has its apex between the T12 and L1 vertebrae and a lumbar scoliosis has its apex between the L1–2 disc space and L4. The apex of the curve is located by the most laterally deviated vertebra. Scoliosis occurs in different groups of patients and is classified according to pathogenesis. Scoliosis may be idiopathic (70%), congenital (15%), neuromuscular (10%) or due to miscellaneous other causes such as Marfan's syndrome or neurofibromatosis (5%). Congenital scoliosis is a scoliosis that arises as the result of the growth of abnormal vertebrae present at birth. Neuromuscular scoliosis is a scoliosis that arises as the result of a neuromuscular condition such as cerebral palsy or Duchenne muscular dystrophy. Scoliosis can also be classified according to its age of onset. Early-onset scoliosis has its onset before the age of 7. Late-onset scoliosis has its onset after the age of 7.

Late-onset (adolescent idiopathic) scoliosis

Late-onset (adolescent) idiopathic scoliosis is the most common form of scoliosis. The prevalence of curves over 10° in an at-risk population (children aged 10–16) is approximately 2% with the same number of males as females affected. As curve magnitude increases there is a decrease in incidence, and an increase in the proportion of girls affected. For curves over 20° the female to male ratio is 5.4 : 1 and for curves over 30% the female to male ratio is 10 : 1 and a prevalence of 0.2%. Aetiology is largely unknown and is probably multifactorial. There is a genetic tendency to develop scoliosis with 20% of affected individuals having at least one affected family member. Of curves measuring 20° or more, 20% will not progress (or progress very slowly) and do not require treatment. Others will progress significantly and cause major spinal deformity.

The development and progression of scoliosis is related to skeletal growth. Scoliosis typically deteriorates (progresses) during growth and once skeletal maturity is reached it stabilises. Scoliosis tends to progress most rapidly during periods of increased growth velocity, in particular the adolescent 'growth spurt'. Features of immaturity and increased growth potential indicate curves that are likely to progress.

The majority of individuals (80%) affected by scoliosis will go on to develop back pain but this is not disabling in the majority of cases. If the curve is particularly severe >90° it may affect cardio-respiratory function but the majority of patients go on to lead normal lives with minimal functional deficit. Indications for consideration of treatment in idiopathic scoliosis are unacceptable deformity and evidence of curve progression. Surgery is usually reserved for severe curves measuring over 50°.

The two main forms of treatment for scoliosis are surgery and brace treatment.

Bracing

Bracing is used to try and maximize growth by delaying the need for surgery and possibly reducing the number of patients that require surgery. Bracing is not a corrective treatment. Its

goal is to halt curve progression. The best that can be expected of brace treatment is that when the brace is removed, the curve is the same size as when it was applied. Bracing is not without morbidity. In order for a brace to be effective it must be worn almost all if not full time (23 h a day). Generally, bracing is poorly tolerated by patients (particularly in hot climates) and it may set up an adversarial relationship between parent and child. It can cause deformity of the thoracic cage and may have a deleterious effect on pain and outcome following surgery. Typically if bracing is to be used it is applied when there is evidence of curve progression and the scoliosis is moderate in size (25–45°) whilst there is still the significant potential for growth. It is then maintained until skeletal maturity or if there is evidence of progression despite the brace another mode of treatment is employed.

Surgery

The aim of surgery is to (partially) correct and stabilise the curve, reducing the deformity and the risk of further progression. Different techniques have been employed including posterior and anterior approaches. During surgery the different instrumentation constructs are inserted in order to apply a controlled force to the spine to correct the three dimensional deformity. The spine is then fused using bone graft that can be autogenous (typically local bone from spinous processes), or donated allograft bone (typically fresh frozen femoral heads). The bone graft forms a scaffold along which the remodelling process of osteoclasts and osteoblasts can take place. Recently bone substitutes have also been used to supplement bone graft and encourage bone healing. These can be calcium phosphate preparations, bone morphogenic protein, or demineralised bone matrix. Eventually a solid mass of bone is formed stabilising the spine and instrumentation. This process can take 6 months or even up to a year to complete and it is, therefore, usually recommended that patients avoid contact sports throughout this period.

Early-onset scoliosis

The term early-onset scoliosis applies to patients under the age of 7 with an idiopathic scoliosis. It is significant because at this young age the development of the lungs is not complete and cardiorespiratory compromise may result from a progressive curve and decreased life expectancy.

Patients that present with an idiopathic scoliosis below the age of 3 years (infantile scoliosis) have the most heterogeneous prognosis. A significant number (80–90% of curves) will resolve before the age of 2 years. However, those that do not resolve go on to develop extremely severe curves that cause major deformity and associated affects on cardiac and respiratory function resulting in limited life expectancy and death in early adult life. Treatment is problematic and prolonged. The most common forms of treatment are serial plaster jackets (localizer casts) and subsequently bracing and eventually growing rods.

Neuromuscular scoliosis

Neuromuscular scoliosis is a scoliosis that occurs in association with a neuromuscular condition. The pathogenesis of neuromuscular scoliosis is different from that of congenital and idiopathic scoliosis in that the scoliosis occurs as a result of muscular weakness which in turn leads to a lack of support for the spine and the resultant 'collapsing curve'. The shape of the scoliosis may be a long 'C' shaped curve, although other curve patterns have been described.

The Scoliosis Research Society (SRS) has classified neuromuscular scoliosis into upper motor neurone, lower motor neurone and myopathic types according to the anatomical location of the neuromuscular lesion. Upper motor neurone causes include cerebral palsy, Friedreich's ataxia, syringomyelia, tumour, trauma. Lower motor neurone causes include myelodysplasia, spinal muscular atrophy, poliomyelitis, tumour and trauma. Myopathic causes include Duchenne muscular dystrophy, arthrogryposis and congenital hypotonia.

The two most frequently encountered neuromuscular conditions causing scoliosis are cerebral palsy and Duchenne muscular dystrophy. In 90% of individuals with Duchenne muscular dystrophy a scoliosis will develop and this frequently develops 1–2 years after progressive muscular weakness has lead to loss of ambulatory function.

Cerebral palsy on the other hand is a condition in which there is a wide variety of function and the likelihood of developing a curvature is related to its severity. Overall 25–30% of patients with cerebral palsy develop a scoliosis but in 4-limb cerebral palsy the incidence of scoliosis increases to 75%. In cerebral palsy the average age of onset of a scoliosis is approximately 10 years.

In these patients scoliosis can lead to problems with sitting balance, causing patients to become hand-dependant sitters, which in turn limits upper limb function. Other complications include pressure sores, back pain, pain from costo-pelvic impingement and an overall poorer quality of life. Other problems such as reflux and the ability to swallow (together with associated chest complications) may be affected by patient positioning and the presence of a scoliosis.

Treatment options for neuromuscular scoliosis include conservative management with braces, wheelchair modifications, total contact orthoses or surgery in form of posterior spinal fusion. A brace will only be effective whilst it is worn and does not have a 'corrective' effect.

The goals of treatment in neuromuscular scoliosis are aimed at maximising quality of life, maintenance of function, maintenance of respiratory function and sitting balance. Surgery in the form of posterior spinal fusion corrects deformity and may help to preserve function and preserve quality of life. In cerebral palsy a high level of carer satisfaction following surgery has been reported. Similar benefits are seen in Duchenne muscular dystrophy and surgery may also allow patients to live for longer having an additive effect with nocturnal ventilation in delaying the deterioration of respiratory function.

Congenital scoliosis

Congenital scoliosis is a scoliosis that develops as a result of the growth of various congenital vertebral anomalies that are present at birth, as a result of either a failure of formation or segmentation or both. In congenital scoliosis multiple vertebral anomalies are often hereditary but isolated anomalies are mostly sporadic. No single genetic or environmental cause has been identified.

The bony structure of the spine is determined at the mesenchymal stage of embryonic development in the first 6 weeks of intrauterine life. Somites form and then undergo a process of segmentation and recombination to give rise to the cartilaginous mould that will subsequently ossify to form the bony spine. Errors in this process can lead to formation defects or segmentation defects or a combination. Commonly seen congenital vertebral anomalies include the unilateral unsegmented bar, the hemivertebra (either fully segmented, semi-segmented or incarcerated), wedge vertebra and block vertebra.

The prognosis and progression of congenital curves depends on their growth potential and whether that growth is balanced. Thus, a fully segmented hemivertebra in connection with a contralateral unsegmented bar has the least balanced growth and the worst prognosis. A block vertebra on the other hand has benign prognosis rarely leading to a curve beyond 20°.

Congenital scoliosis may be associated with other congenital malformations as part of the VATER (Vertebral, Anorectal, Tracheal, Oesophageal, Renal) or VACTERL (Vertebral, Anorectal, Cardiac, Tracheal, Oesophageal, Renal and Limb) associations as well as many others. Detection of a congenital scoliosis should, therefore, prompt further investigation, including an echocardiogram and renal ultrasound as a baseline.

Congenital vertebral anomalies may generate a congenital kyphosis or kyphoscoliosis. Congenital kyphosis and kyphoscoliosis are much less common than either idiopathic or congenital scoliosis. When present it is potentially a more serious diagnosis as progression can be rapid and may lead to spinal cord compression and paralysis.

Syndromic/miscellaneous causes of scoliosis

Many different syndromes with different aetiologies are associated with scoliosis. The following are just a few of the more common examples:

Marfan's syndrome is a connective tissue disorder with manifestations in many organ systems. Caused by a dominantly inherited defect in the chromosome 15 gene *FBN1*, which encodes the glycoprotein fibrillin-1. Patients appear tall with elongated limbs. Skeletal manifestations include: Hammar toes, pes planus, elongated limbs, ligamentous laxity, scoliosis, chest wall anomalies, arachnodactyly and a high-arched palate. Other problems include ocular (lens) problems and cardiac anomalies (dilated aortic root).

Osteogenesis imperfecta is a connective tissue disorder caused by a defect in type 1 collagen. Different types of osteogenesis imperfecta and inheritance patterns have been described, but it is now generally believed to be inherited in an autosomal dominant manner with variable penetrance. Features of the condition vary with type but include: Brittle bones, blue sclerae, absent or poor dentinogenesis, scoliosis, poor muscular tone, short stature, respiratory and hearing problems.

Neurofibromatosis is a condition that affects neural crest cells and is inherited in an autosomal dominant manner. There are two types. Type 1 neurofibromatosis is caused by a mutation in the neurofibromatin gene on chromosome 17 and presents with: neurofibromas, groin and axillary freckling, café-au-lait spots, tibial bowing, lisch nodules and optic nerve tumours. Type 2 is characterized by tumours affecting the eighth (vesibulocochlear) cranial nerve causing hearing loss, balance problems and headache.

Other syndromes associated with scoliosis include but are not limited to: Angleman syndrome, Di George syndrome, cleidocranial dysplasia, Goldenhar syndrome, Klippel Feil syndrome, Sprengel's shoulder, Noonan syndrome, Retts' syndrome and spondyloepiphyseal dysplasia.

Assessment

History

A history of pain should be investigated to rule out another cause (prolapsed disc, osteoid osteoma, spondylolisthesis intraspinal anomaly). An assessment of the patient's skeletal maturity (age, menarche, parental height), to estimate the risk of curve progression and an assessment of the general health of the individual (fitness for anaesthesia). In the neuromuscular group the assessment of general fitness is likely to require input from multiple different specialties and a multidisciplinary team approach.

Examination

Assessment of the curve includes: Site, size and location of the curvature. Common associated problems such as rib prominence, shoulder height and waist asymmetry. Features of underlying spinal dysraphism (hairy patches). Abnormal abdominal reflexes are most commonly associated with intraspinal anomalies. Neurological examination of the lower limbs. Features of recognisable syndromes present (café-au-lait spots, axillary freckling, blue sclerae, long arms, etc).

The Adam's forward bend test performed by examining the patient from the back and asking the patient to bend forward accentuates the deformity particularly rotational deformity and the rib prominence.

The most common type of curve seen in late-onset idiopathic scoliosis is a 'right thoracic' curve presenting in a girl just after menarche. Atypical features indicate possible underlying pathology. Atypical features include a left-sided curve, severe pain, rapid progression and short angular deformities.

Investigations

Full-length standing plane radiographs that show the iliac crests and hip joints.

Risser's sign which examines the progression of development and fusion of the iliac apophysis is visible on plain x-rays and is a useful indication of maturity.

MRI scanning of the spine can be used to detect underlying neuroaxial anomalies, such as diastomatomyelia, syringomyelia and Arnold–Chiari malformations.

Surface topography is also frequently used to assess scoliosis. This can produce an objective assessment of the results of surgery but in some circumstances can also be used instead of frequent x-ray when following up the progression of curves.

Treatment

Idiopathic – Curves under 20° or non-progressive curves do not require treatment. Curves between 20° and 50° can be treated in a brace or if not tolerated observed. For severe curves over 50°, surgery. For thoracic curves and double major curves, posterior spinal fusion. Anterior surgery for thoracolumbar curves. Anterior release for stiff curves >80°.

Neuromuscular – bracing or posterior spinal fusion

Surgical approaches

Cervical spine

Anterolateral approach (Smith–Robinson)

Commonly used for ACDF this is one of the most common approaches to the neck. It allows access to all levels.

Technique

Patient placed in supine position with their neck and head slightly extended (roll under shoulders). Head may be stabilised in a Mayfield clamp or head ring and the shoulders may be taped holding them inferiorly and posteriorly to facilitate intraoperative x-ray. Right-sided approaches may be more commonly used (easier for right-handed surgeons). Recent anatomical studies have demonstrated that in terms of the relative courses of the recurrent laryngeal nerve, there is no difference in the side of the approach[10]. Differences in the course of the nerves with the nerve on the right side looping round subclavian artery and the left the aorta only occur below the level of T1. The skin incision is made along a transverse skin crease and is guided by surface anatomy. The Hyoid bone corresponds to C3. The thyroid cartilage corresponds to the C4–C5 level. The cricoid cartilage corresponds to the C6–C7 level. Platysma is incised in line with the skin incision or split along the length of its fibres to allow extension of the approach superiorly and inferiorly. The anterior (medial) border of SCM is identified and the fascia is divided anteriorly. Blunt dissection continues in the avascular plane between the carotid sheath (palpated laterally) and the trachea (medially) to expose deep fascia. The deep (pretracheal) fascia is divided longitudinally in the midline. The anterior longitudinal ligament is divided in midline, retracted laterally with periosteum and retractors inserted deep to longus colli. The level is checked with a clip on the anterior portion of the cervical disc to prevent disc injury.

Complications include a hoarse voice, most commonly associated with endotracheal tube placement but also by injury to recurrent laryngeal nerve by retraction or dissection. Dysphagia due to swelling. Vascular or visceral injuries are possible. Neck swelling with airway compromise caused by haematoma requires urgent decompression closure is usually with clips to allow ward staff to decompress haematoma.

Thoracic spine

Anterior approach

This approach may be used for an anterior release in scoliosis surgery, the treatment of fractures, tumours or for thoracic disc excision.

Technique

The patient is positioned in lateral decubitus position with the table flexed. The side of approach depends on pathology. For scoliosis surgery approach the spine from the side of the convexity. The incision is made one to two levels above the affected level along the level of the rib, extending from paraspinal muscles to midclavicular line. Trapezius and latissimus dorsi dissected superficially. Rib dissected subperiosteally and removed preserving the intercostal bundles. Parietal pleura incised. Lung retracted (gently) to expose vertebra and discs. Segmental vessels are ligated and divided.

Thoracolumbar (Hodgson)

Typically used for thoracolumbar scoliosis correction or for access to thoracolumbar vertebrae for tumour or fracture.

Technique

Patient in lateral decubitus position with their limbs and trunk supported. Flex the table with apex at thoracolumbar junction. Usually left-sided approach to avoid the IVC and liver. Make a skin incision over the rib, curved distally and longitudinally. Dissect serratus anterior, external oblique and latissimus dorsi. Remove the rib. Incise the pleura, open the chest and site rib spreaders. Split the costal cartilage to enter the retroperitoneum. Sweep away Gerota's fascia and peritoneum with swabs. Divide the abdominal muscle layers. Divide the diaphragm 2 cm from its origin down to the vertebrae using marking stitches. Ligate segmental vessels.

Closure – Close the diaphragm using marker sutures to help. Suture the costal cartilage anteriorly ± retroperitoneal drain. Close abdominal muscles separately. Place a chest drain before rib approximation. Close pleura, periosteum and intercostal muscles. Suture serratus anterior and latissimus dorsi separately.

Lumbar spine

Anterior approach to the lower lumbar spine

This approach may be used for anterior discectomy and fusion or disc replacement, corpectomy and cages for tumour, trauma and infection.

Technique

Pararectal retroperitoneal approach. Patient supine with their hips/knees slightly flexed. Make a skin incision in the midline. Incise the linea alba, lift the left rectus and retract it laterally (preserve segmental nerve supply). Incise the posterior rectus sheath laterally above the arcuate line. Find the peritoneal edge. Extend the sheath incision proximally/distally. Avoid entering the peritoneum. Enter the retroperitoneal space and sweep away the peritoneum with a swab; expose psoas (nerves on surface), ureter and iliac vessels. For L5/S1 dissect the lumbosacral plexus with a pledget to avoid injury (risk of retrograde ejaculation in males). Ligate the median sacral vessels and expose the disc. For L4/5 retract the aorta/IVC to the right. Ligate and divide segmental vessels at L5 and the iliolumbar vein/ascending lumbar vein. Closure: Close the rectus sheath, being careful to prevent hernias.

Other miscellaneous conditions

Thoracic disc prolapse

Thoracic disc prolapses are rare and make up <1% of all disc surgery. Asymptomatic prolapse probably more common. Diagnosis is often delayed. An MRI scan is the investigation of choice. Patients present with pain and sign and symptoms of cord compression, lower limb weakness, ataxia, numbness and bowel/bladder dysfunction.

Treatment is surgical via anterior (thoracotomy) or posterior (costotransversectomy). Neurological recovery often very good.

Diffuse idiopathic skeletal hyperostosis (DISH – Forestier's disease)

First described by Forestier in 1950 as a 'senile ankylosing hyperostosis'. It is a systemic condition in which ligaments, tendons and joint capsules ossify. It can occur anywhere in the spine and may also affect other joints and ligaments. It is more common in men and uncommon under the age of 50 years. The cause is unknown. There are three types described. Type 1 ossification of the anterior longitudinal ligament (Forestier's), type 2 diffuse changes (DISH) and type 3 ossification of the posterior longitudinal ligament. It is often asymptomatic and diagnosed incidentally (ossification continuous along at least four adjacent vertebrae). Management is symptomatic.

Paget's disease affecting the spine

Paget's disease is a disorder of bone remodelling and one-third of patients affected will have spinal involvement. There is an exaggerated osteoclastic bone resorbtion followed by exaggerated bone formation. It typically follows three phases, lytic, active, and burnt-out phases. Bone formation may cause spinal stenosis or foraminal stenosis with neurological compression as well as facet joint arthropathy. Back pain, symptoms of spinal stenosis or radiculopathy may develop. Also increased blood supply to the bone may cause a relative ischemia of the neural tissue (steal syndrome). Plain radiographs reveal expanded bones with thickened cortices. Serum alkaline phosphatase is elevated with a normal calcium level. Urinary hydroxyproline levels are elevated. Bone scans show increased uptake. The mainstay of treatment is non-operative with NSAIDs and bisphosphonates. Symptoms from spinal stenosis and radiculopathy commonly resolve with medical treatment and surgery is rarely indicated. If surgery is contemplated for resistant cases, increased intraoperative bleeding should be expected.

Current controversies

Vertebroplasty

Vertebral body cement augmentation has been used to treat pain from the mechanical instability of tumours (NICE Guideline CG75[8]): 'Vertebroplasty or kyphoplasty should be considered for patients who have vertebral metastases and no evidence of MSCC or spinal instability if they have either, mechanical pain resistant to analgesia, or vertebral body collapse.'

Its role in treating osteoporotic spine fractures is becoming increasingly accepted. NICE technology appraisal (TA279[7]): 'NICE recommends vertebroplasty and kyphoplasty (without stenting) as possible treatment options for some people with spinal compression fractures caused by osteoporosis.' Prospective randomised trials show conflicting results[11,12].

Mild complications of the procedure include transient arterial hypotension; cement leakage into the intervertebral disc space (little or no clinical consequence), or paravertebral soft tissues. A substantial number of patients with osteoporosis develop new fractures after the procedure; two-thirds of these new fractures occur in a vertebra adjacent to those previously treated. Moderate complications include infection (discitis, osteomyelitis; or epidural infection-can be difficult to treat), pulmonary cement embolism and cement leak into the epidural/foraminal space. Severe complications include cement leakage into the paravertebral veins leading to pulmonary embolism, cardiac perforation, cerebral embolism and even death.

Disc replacement

Proposed for the treatment of degenerative disc disease not resolving with conservative measures. Benefits include maintenance of mobility delaying the onset of adjacent level degenerative change.

Procedure is performed via an anterior approach (retroperitoneal). A Cochrane review (2012)[13] on the use of TDR in LBP however could not find any evidence of better outcomes in terms of LBP and patient function between TDR and spinal fusion, and advised caution on adopting TDR.

Spinal spacer devices

An alternative to conventional decompressive surgery in managing symptomatic lumbar spinal pathology, especially in the older population. The aim of these devices is to unload the spine, restoring foraminal height, and stabilize the spine by distracting the spinous processes. Marketed as a safe, effective, and minimally invasive surgical alternative for relief of neurological symptoms in patients with low back degenerative diseases, recent studies suggest less impressive clinical results and higher rate of failure than initially reported. Current evidence is not sufficient to know of their real outcome and the evidence of their effectiveness is open to discussion.

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Chapter 20

Tumour oral core topics

Thomas Beckingsale and Craig H. Gerrand

Patients with suspected cancer MUST be referred without delay!

NICE guidelines and referral pathways[1]

In the UK, patients with suspected cancer must be offered an appointment with a specialist within 2 weeks of GP referral. Treatment must be started within 62 days from the date of referral, or within 31 days of a definitive diagnosis of cancer. Children must receive treatment within 31 days of referral for suspected cancer.

Examiners have recently been instructed by the Intercollegiate Board not to ask candidates about NICE guidelines. These are national UK guidelines and the exam is international therefore international candidates would not be familiar with them.

Suspected sarcoma

As per NICE guidelines, patients with a palpable lump that meets any of the following criteria:

- >5 cm
- Increasing in size
- Painful
- Deep to fascia
- Recurrent after a previous excision

Should be referred to a local sarcoma service under the 2-week rule.

Patients with x-rays suspicious of a malignant or aggressive process should also be referred under the 2-week rule.

Sarcoma treatment is delivered by specialist multidisciplinary teams (MDT), comprising diagnostic and treatment expertise to cover the range of diagnoses and presentations of sarcoma as well as complex benign tumours. In England and Wales, there are five specialist bone tumour units in Newcastle-upon-Tyne, Birmingham, London, Oxford and Oswestry. In addition, Liverpool, Leeds, Sheffield, Manchester, Nottingham, Bristol, Plymouth, and Exeter are home to soft-tissue sarcoma units, and referrals can also be made through the South Wales MDT, and the London Sarcoma Service.

Northern Ireland has its own Sarcoma MDT for bone and soft-tissue sarcomas centred in Belfast, while in Scotland, referrals are made through the Scottish Sarcoma Network with patients treated in Aberdeen, Glasgow, Edinburgh, Dundee and Inverness.

A high index of suspicion!

- Delays in diagnosis are common for sarcoma patients and may lead to poorer outcomes
- Failure to suspect a sarcoma can lead to mismanagement
- In the UK there are fewer than 600 bone sarcomas, and 3300 soft-tissue sarcomas diagnosed each year, <1% of all cancers. (Based on figures for 2013.)
- Those affected are often teenagers or young adults.
- Clinicians, therefore, need to be vigilant to ensure timely diagnosis and treatment, and avoid mismanagement
- Unexplained bone pain in children should never be dismissed as 'growing pains', especially if unilateral
- X-ray investigation is mandatory to exclude bony malignancy, avoid delay in diagnosis, and prevent incorrect treatment, such as arthroscopy or steroid injection when there is an underlying tumour
- Patients with suspicious lesions should be referred urgently to a specialist Sarcoma Service (see above) for biopsy and treatment

X-rays are mandatory!

- Patients with increasing, unexplained or persistent bone pain or tenderness, particularly pain at rest, an unexplained limp, or a suspected spontaneous fracture should be investigated urgently
- X-rays can look normal, even in the presence of a primary bone tumour, so consider safety-netting through early review if there is concern

Tumour biology/basic concepts

What is a sarcoma?

Sarcomas are a rare and diverse group of malignant tumours arising from connective tissue (the embryonic mesodermal layer) and also, by convention, nerve tissues.

A **tumour** is an abnormal growth which:

- enlarges by cellular proliferation more rapidly than surrounding normal tissue
- Continues to enlarge after the initiating stimulus ceases
- Usually lacks structural organisation and functional coordination with normal tissues

- Serves no useful purpose to the host organism

A **malignant tumour** is one with a predisposition to invasive and destructive local growth, and **also** to distant metastatic spread.

Benign tumours, while in some instances still locally aggressive, do NOT metastasise.

How are tumours classified?

The World Health Organisation (WHO) publishes classification systems for all types of cancer as part of the *International Classification of Diseases* (ICD). The *WHO Classification of Tumours of Soft Tissue and Bone*, covering mesenchymal tumours, was revised in 2013 (fourth edition).

- The WHO classification system provides a universal nomenclature
- It ensures comparability of translational research and international clinical trials
- Changes from the third edition have been driven by advances in molecular biology and include more cytogenetic data
  - Malignant fibrous histiocytoma (MFH), previously one of the most common diagnoses, has been removed
  - Advances in molecular/genetic diagnosis have shown that MFH can usually be more correctly categorized under other tumour categories
  - Tumours that, despite extensive testing, do no fall under other headings, are now termed **undifferentiated pleomorphic sarcomas**
  - There are over 100 specific soft-tissue tumour subtypes

See Table 20.1 for common genetic translocations seen in sarcomas.

Tumour biology

Concepts of tumour growth[2]

- **The compression zone/pseudocapsule** – Tumours grow in a centrifugal fashion leading to compression and then atrophy of the normal surrounding tissue
- **Reactive zone** – Surrounding the pseudocapsule is an area of oedema and neovascularity characterized by the presence of inflammatory cells and micronodules of tumour. Resection should, therefore, pass outside the reactive zone to ensure complete removal of the tumour and minimize local recurrence

Table 20.1

Diagnosis	Chromosomal abnormality	Genes involved
Alveolar rhabdomyosarcoma	t(2;13)(q35;q14) t(1;13)(p36;q14)	*PAX3-FKHR* *PAX7-FKHR*
Alveolar soft part sarcoma	t(X;17)(p11.2;q25)	*TFE3-ASPL*
Angiomatoid fibrous histiocytoma	t(12;16)(q13;p11)	*FUS-ATF1*
Clear cell sarcoma	t(12;22)(q13;q12)	*EWS-ATF1*
Congenital fibrosarcoma/ Congenital mesoblasticnephroma	t(12;15)(p13;q25)	*ETV6-NTRK3*
Dermatofibrosarcomaprotuberans	t(17;22)(q22;q13)	*PDFGB-COL1A1*
Desmoplastic small round cell tumor	t(11;22)(p13;q12)	*EWS-WT1*
Endometrial stromal sarcoma	t(7;17)(p15;q21)	*JAZF1-JJAZ1*
Ewing's sarcoma/ Peripheral primitive neuroectodermal tumor	t(11;22)(q24;q12) t(21;22)(q22;q12) t(7;22)(p22;q12) t(17;22)(q12;q12) t(2;22)(q33;q12) t(16;21)(p11;q22)	*EWS-FLI1* *EWS-ERG* *EWS-ETV1* *EWS-FEV* *EWS-E1AF* *FUS-ERG*
Inflammatory myofibroblastic tumor	t(1;2)(q22;p23) t(2;19)(p23;p13) t(2;17)(p23;q23)	*TPM3-ALK* *TPM4-ALK* *CLTC-ALK*
Low-grade fibromyxoid sarcoma	t(7;16)((q33;p11)	*FUS-CREB3l2*
Myxoidchondrosarcoma	t(9;22)(q22;q12) t(9;15)(q22;q21) t(9;17)q22;q11)	*EWS-CHN* *TFC12-CHN* *TAF2N-CHN*
Myxoidliposarcoma	t(12;16)(q13;p11) t(12;22)(q13;q12)	*TLS-CHOP* *EWS-CHOP*
Synovial sarcoma	t(X;18)(p11;q11)	*SSX1-SYT* *SSX2-SYT* *SSX4-SYT*

- In High-grade tumours, micronodules and extension of the tumour into and through the reactive zone can lead to satellite and skip lesions

Local behaviour of soft-tissue tumours

- Soft-tissue tumours tend to respect anatomical boundaries, i.e. fascia and bone resist invasion by tumour
- Thus, tumours tend to remain within the osteofascial compartment in which they arise. For example, the thigh has three compartments: Anterior, posterior and medial
- Anatomical compartments with less well-defined boundaries are termed extra-compartmental, e.g. popliteal fossa. Extra-compartmental tumours can extend further than those contained within compartments and are, thus, more difficult to remove with a wide margin (see below)
- Extension of tumour through the boundaries of a compartment does occur but tends to be late in the disease process, and can follow defects in the osteofascial envelope, for example where there are perforating vessels

Margin[3]

Based on this understanding of tumour growth and anatomical compartments, Enneking et al. described resection margins as intra-lesional, marginal, wide, or radical as follows:

- **Intra-lesional nargin** – The resection passes through the tumour and macroscopic tumour deposits are left in the wound
- **Marginal margin** (*sic*) – The tumour is excised with an intact pseudocapsule but the reactive zone is violated possibly leaving microscopic satellites within the wound
- **Wide margin** – The tumour is excised with a cuff of normal surrounding tissue. In high-grade lesions there is a risk that microscopic skip lesions will remain within the normal tissue
- **Radical margin** – The entire compartment in which the tumour resides is excised *en bloc* in theory removing the entire tumour

NB. The risk of local recurrence is directly related to the surgical margins.

How to approach a patient with a suspected tumour

Clinical history and examination

A careful and thorough history and examination can help point to a likely diagnosis. Key points include:

- Patient age. For example, Ewing's sarcoma is most common in adolescence but is exceptionally rare after 40, whereas giant cell tumour (GCT) of bone is almost never seen in children but increases in incidence in the fourth decade
- Duration of symptoms
- Rapid growth of a new lesion or change in a pre-existing lesion, which might signify malignant transformation; for example, growth of an osteochondroma after skeletal maturity
- Red flags for cancer including a history of lethargy or weight loss. This may be more common in patients with metastatic bone disease
- Pain history: site, character, severity, radiation, modifying factors, onset, periodicity. Night pain is classically associated with malignant disease. Pain from tumours may fluctuate
- If patient presents after a fracture, was there pain beforehand (prodromal) or only minimal trauma?
- A personal or family history of malignancy or a predisposing condition (e.g. Li–Fraumeni syndrome, multiple osteochondromas ollier disease, Maffucci syndrome, neurofibromatosis type 1 (Nf-1))
- Previous radiotherapy is a risk factor for the formation of secondary malignancies, commonly sarcomas such as angiosarcoma. It is also associated with pathological fractures of bones within the treated field, the treatment of which is complicated by frequent non-union

Key features on examination include:

- Swelling: site, size, depth (above or below fascia) shape, surfaces, edges, consistency, fluctuance, pulsatility, tethering, overlying skin, draining lymph nodes
- Involvement of adjacent joint and neurovascular structures
- Signs of previous treatment, e.g. scars, radiotherapy tattoos, mastectomy
- Abdominal examination for masses, organomegaly. Consider rectal examination
- If metastatic bone disease a possibility, consider examination of other sites, e.g. breast and thyroid

An abnormal x-ray: seven questions

1. **Where is the lesion? (Figure 20.8)**
   - The particular bone involved, the part thereof (epiphysis, metaphysis, diaphysis), and the relationship to the medullary cavity (central or eccentric) give valuable diagnostic information; e.g. unicameral bone cysts are central, metaphyseal lesions and 50% arise in the proximal humerus (Figure 20.1)
2. **How extensive is the lesion?**
   - How large is the lesion? Is it solitary or multiple? e.g. skip lesions of primary bone tumours or multiple metastatic deposits (Figure 20.2)
3. **What is the lesion doing to the bone?**
   - Look at the *zone of transition* between the lesion and the bone
   - A **narrow** *zone of transition* is well-defined and geographical (like the edge of a land mass on a map). It is usually possible to draw round the edge with an imaginary pencil. This is seen in lesions that grow slowly enough to be walled off at the margins. There may also be bony expansion and a neo-cortex. This usually indicates a benign lesion (Figure 20.1)
   - A **wide** *zone of transition* is one that is ill-defined and permeative, with a poorly demarcated border. This is

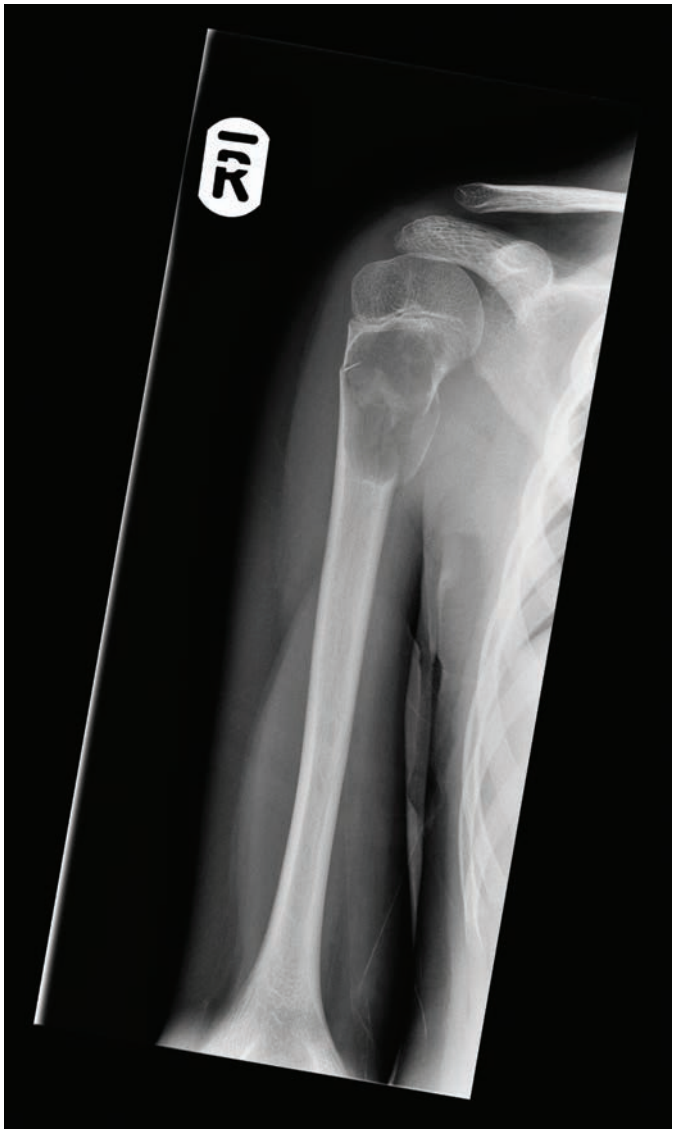

Figure 20.1 Pathalogical fracture through a unicameral bone cyst of the proximal humerus in a 9-year-old girl. 50% of unicameral bone cysts arise in the proximal humerus. The lesion has occurred in the metaphyseal region of a skeletally immature patient. It has a geographical border with a narrow zone of transition. There is no periosteal reaction and no obvious matrix production

seen in rapidly growing, aggressive lesions and usually indicates a malignant lesion (Figure 20.2)

4. **What is the bone doing in response?**
   - The bone can respond by 'walling off' a slowly growing lesion leading to a narrow *zone of transition*
   - The periosteum can react to the tumour, producing
     - A **Codman's triangle.** A triangle of reactive bone at the edge of the tumour where the periosteum is elevated (Figure 20.3)
     - **Sunray spiculation.** A spiculated periosteal reaction reflects rapid underlying growth preventing normal new bone formation under the raised periosteum (Figure 20.4)

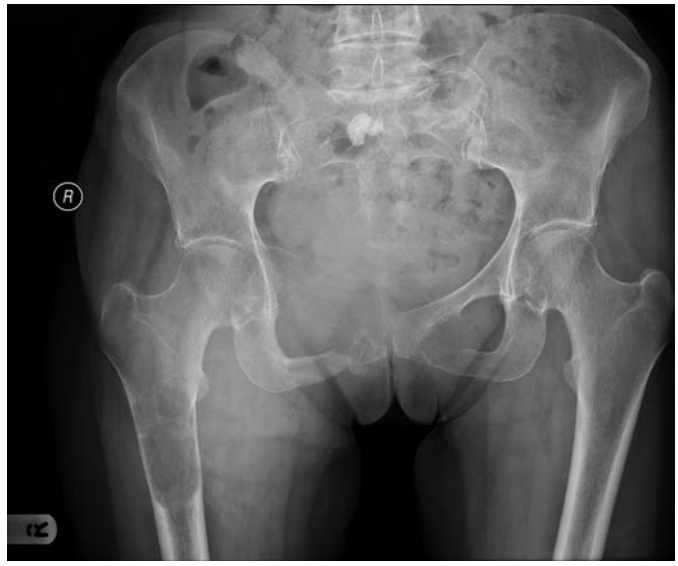

Figure 20.2 Metastatic disease affecting the pelvis and right proximal femur. There are multiple lesions affecting more than one bone. The lesions have a wide zone of transition, are producing no specific matrix, are eroding the cortex and are extending into the soft tissues (all comments especially true of the lesion in the right superior pubic ramus)

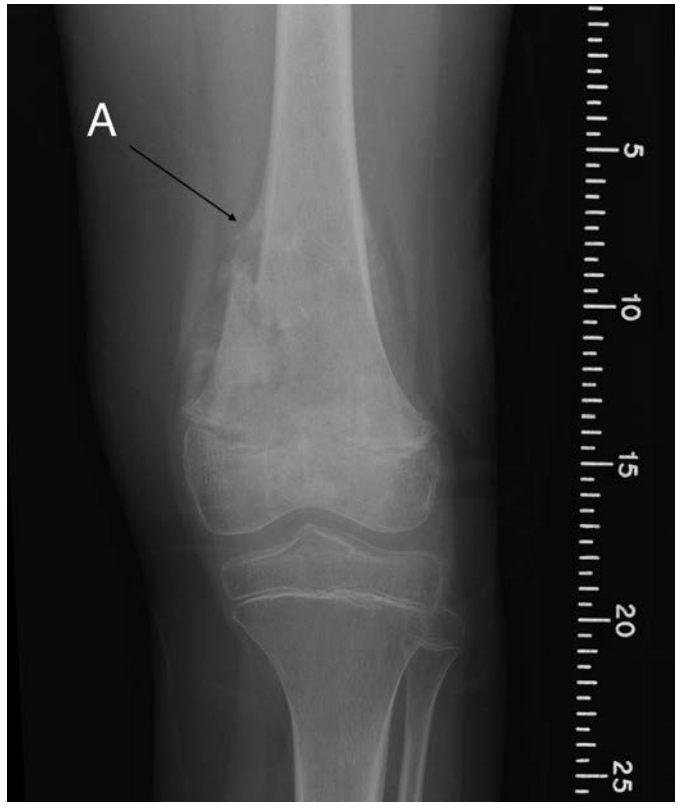

Figure 20.3 Osteosarcoma of the left distal femur. There is a metaphyseal lesion in a skeletally immature patient, with a wide zone of transition. A widespread periosteal reaction is seen with classic Codman's triangle (A). The lesion is producing an osseous matrix, which is extending through the cortices and into the surrounding soft tissues

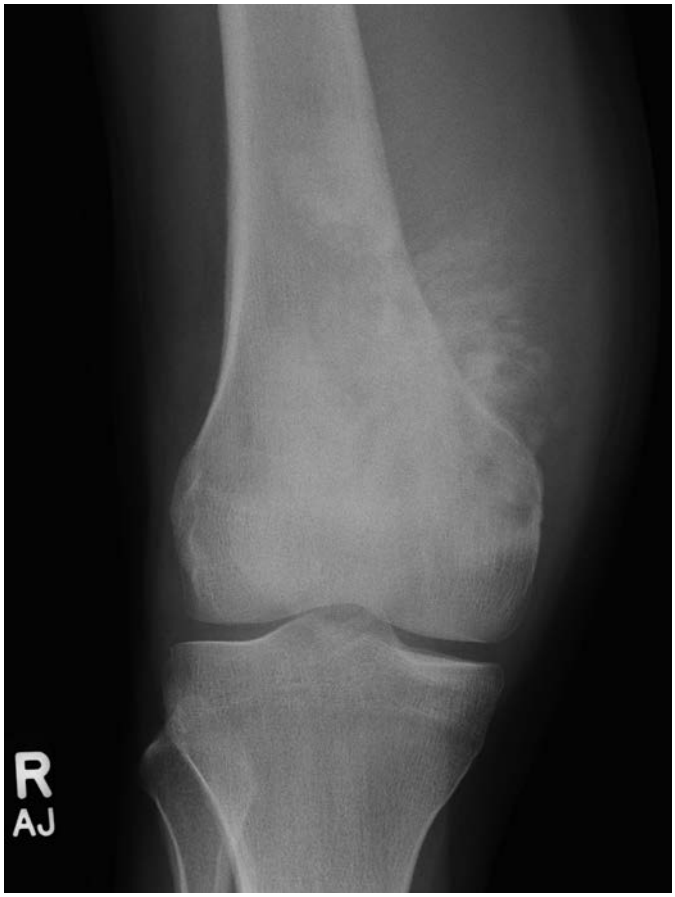

Figure 20.4 Osteosarcoma of the right distal femur. There is a metaphyseal lesion in a skeletally mature patient extending into the epiphyseal region. It has a wide zone of transition, there is a corresponding sunray periosteal reaction, and it is producing a bony matrix that is extending through the cortex into the soft tissues

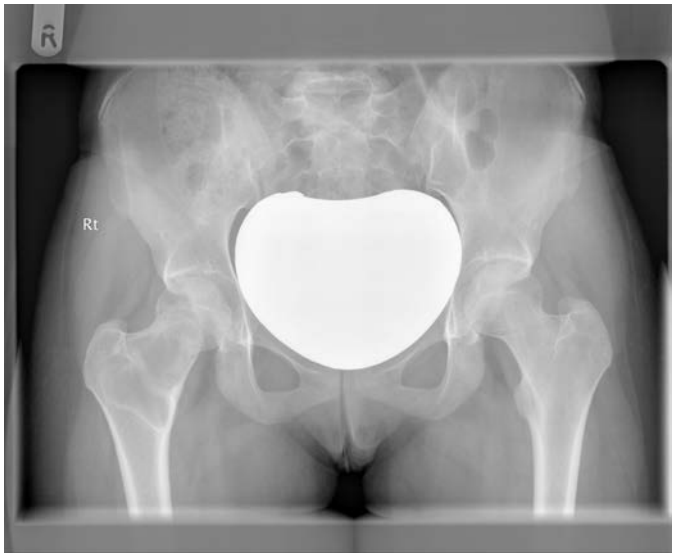

Figure 20.6 Fibrous dysplasia of the right hip. There is a metaphyseal lesion in a skeletally immature patient. It has a narrow zone of transition, which is walled off proximally, and there is no periosteal reaction. There is a ground glass matrix and no cortical destruction or extension into the soft tissues

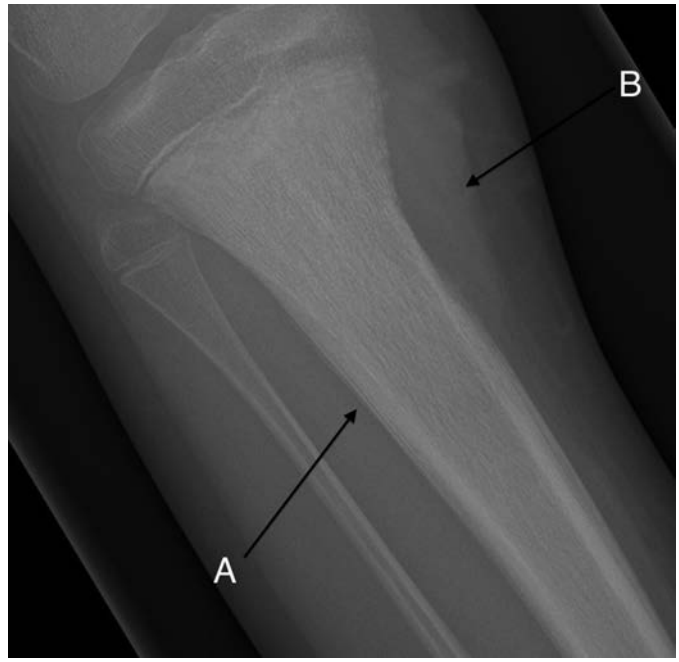

Figure 20.5 Ewing's sarcoma of the right proximal tibia. There is a lesion in the metaphyseal, diaphyseal region in a skeletally immature patient. It has a wide zone of transition and there is an onion-skin type periosteal reaction laterally (A). There is no particular matrix. Medially there is cortical destruction with a soft-tissue mass (B)

  - **Onion-skinning.** Multiple layers of new periosteal bone formation, possibly reflecting phases of growth of the tumour (classically seen in Ewing's sarcoma) (Figure 20.5)

5. **Is the lesion making matrix?**
   - **Ground glass.** This is classically used to describe fibrous lesions within bone, e.g. fibrous dysplasia, although the appearances of fibrous dysplasia can be diverse (It looks like the medullary trabeculae have been smudged out by the viewer's thumb) (Figure 20.6). Fluid-filled lesions (e.g. simple bone cyst) may also look like this
   - **Popcorn calcification.** Punctate, ring and arc calcifications are seen in cartilage forming tumours, e.g. enchondroma or chondrosarcoma, where calcification occurs at the margins of tumour lobules (Figure 20.7)
   - **Bone forming.** This is characteristic of osteogenic tumours (i.e. osteosarcomas). New bone can be mixed with lytic areas within the bone, and is often seen within the associated soft-tissue mass (Figures 20.3 or 20.4)
   - **No specific matrix.** This is seen in rapidly growing tumours that cause bony destruction and lytic lesions, e.g. metastatic/myelomatous deposits
6. **Is the cortex eroded?**
   - Medullary lesions may cause endosteal scalloping or cortical resorption, the presence of which is indicative of more aggressive behaviour

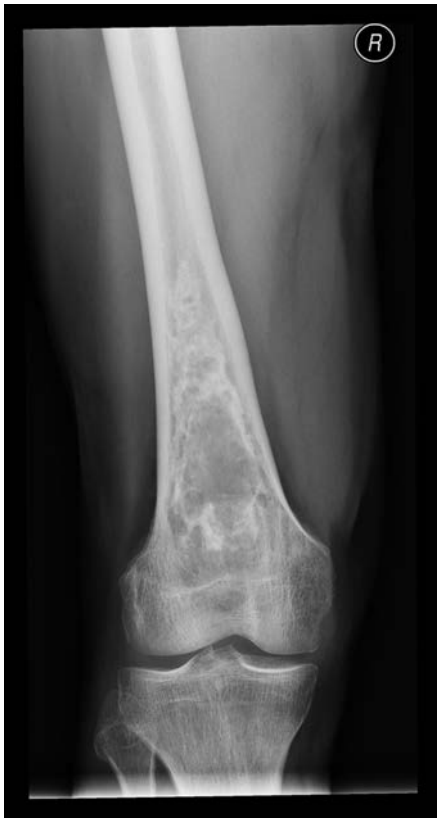

Figure 20.7
Enchondroma right distal femur. There is a metaphyseal lesion in this skeletally mature patient. It has a relatively narrow zone of transition and there is no periosteal reaction. It is producing rings and arcs of calcification, consistent with a cartilaginous matrix. There is no cortical erosion or soft-tissue mass

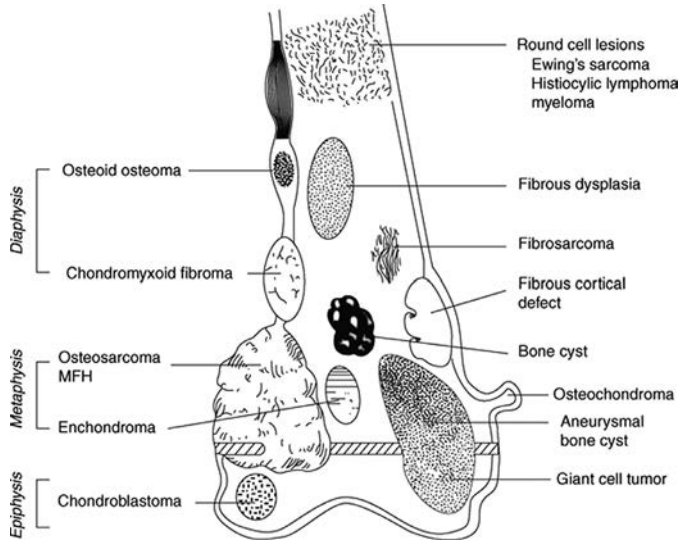

Figure 20.8 Characteristic sites of tumours within bone

7. **Is there a soft-tissue mass?**
   - Soft-tissue extension is also indicative of more aggressive behaviour, be that secondary to cortical destruction in metastatic disease or due to permeative growth through the cortex in bone sarcomas, e.g. osteosarcoma or Ewing's sarcoma. (Figures 20.3, 20.4 or 20.5)

How to investigate a patient with suspected metastatic bone disease

Patients with a clear diagnosis of metastatic bone disease (MBD) (i.e. multiple characteristic lesions and a clear history of cancer) should be discussed in a local cancer MDT. Every orthopaedic department should have a clinical lead for MBD. If there is dubiety or a lesion meets the referral guidelines then urgent referral to the local sarcoma service is needed. The BOA publishes guidelines on orthopaedic standards of care for MBD. Care should be taken when a patient appears to have a solitary metastasis that a sarcoma is not missed.

Where a radiograph has shown destructive, usually lytic, possibly multiple bony lesions, and metastatic disease without a known primary is suspected, then investigation should include the following:

1. **Get more information about the lesion itself. Local site imaging**
   (a) **Full length x-rays** (Figure 20.9) of the affected bone including the joint above and below the lesion may show multiple lesions
   (b) **MRI** and **CT** give better three-dimensional information about the extent of the tumour in the bone and possible soft-tissue involvement, and can help to plan any surgical intervention once the diagnosis is made
   (c) **Isotope bone scan** may show lesions in multiple bones
2. **Screen for potential primary tumours**
   (a) **History and examination**
   - **History** may give useful clues to guide further investigation, e.g. previously unreported haematuria
   - **Examination** may identify a suspicious lump, e.g. in breast or thyroid

   (b) **Blood tests**
   - **Serum electrophoresis** may diagnose myeloma
   - **Tumour markers** including prostate specific antigen (PSA) carcinoembryonic antigen (CEA) and carcinoma antigen 125 (CA125) may suggest potential primary tumours
   - **Blood biochemistry** including liver and thyroid function tests may help to identify other organs affected. Bone biochemistry for hypercalcaemia is mandatory. Patients with hypercalcaemia may appear moribund but may make a rapid recovery when this is treatment
   - **Full blood count.** May be helpful as part of the workup

   c. **CT chest/abdo/pelvis** is used as a screening tool to pick up tumours in the main body cavities (Figure 20.10)

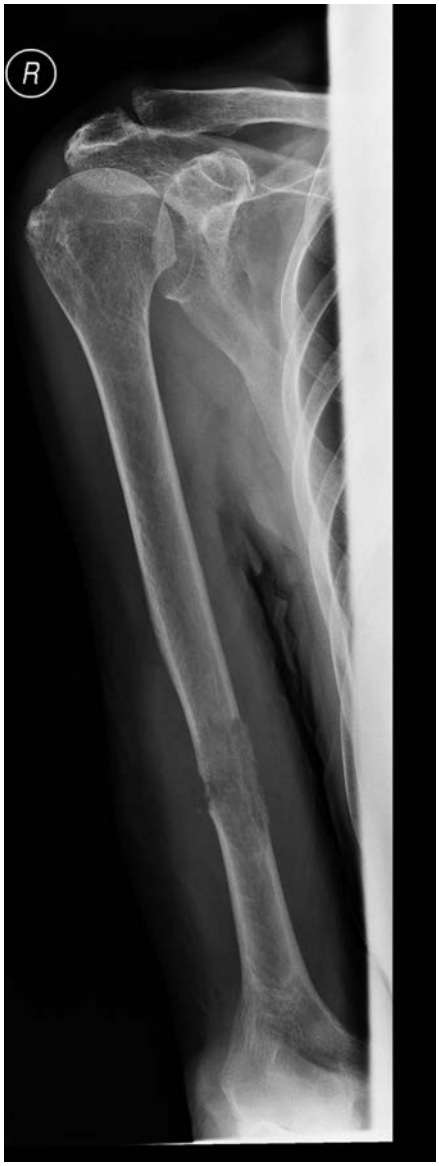

Figure 20.9 Metastatic lung cancer in the right humerus. There is a diaphyseal lesion which appears aggressive, particularly in its effect on the cortex, which appears hazy, and, therefore, has a wide zone of transition. There is no specific matrix, the cortex is eroded and it appears to extent out to the soft tissues. There is also a suspicion of a pathological fracture

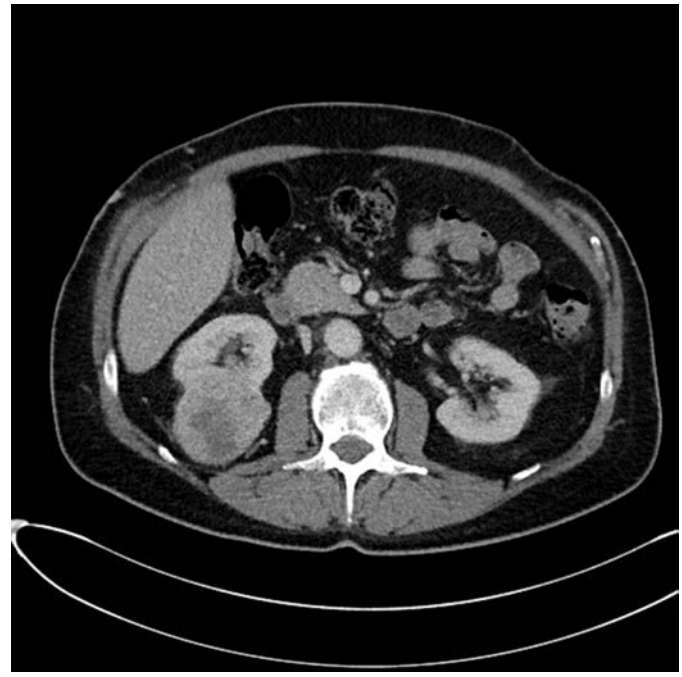

Figure 20.10 CT showing an incidental lesion on the right kidney, likely the source of the metastatic deposit

d. **Other investigations**

- These may be indicated according to the clinical picture and the results of other investigations, e.g. **mammography** for suspected breast primary; **thyroid ultrasound** for suspected thyroid cancer, etc
- If a patient has metastatic disease and a primary tumour cannot be identified, many hospitals now have a 'carcinoma of unknown primary' team with whom the patient can be discussed. They may advise further investigations such as a PET scan

3. **Biopsy if a primary source can not be identified**

(a) If doubt remains after investigation, or a lesion is solitary, discussion with a bone tumour unit is recommended

NB.

- **Never assume that a solitary lesion is a metastasis**
- **Do not rush to fix a pathological fracture, even if open, if the underlying diagnosis is unknown – Wound care with traction or splintage will suffice while investigations are performed**
- **If in doubt discuss the case with a bone tumour unit**

How to investigate a suspected primary bone or soft-tissue tumour in a sarcoma unit

Patients referred to a sarcoma service under suspected cancer guidelines may have a biopsy earlier in the care pathway. This prevents unnecessary staging investigations for initially suspicious lesions that turn out to be benign. After initial history and examination, investigation should include the following.

1. **Image the lesion itself – 'local staging'**

- Soft-tissue lesions can initially be investigated with an ultrasound scan. This can give useful information about the lesion, in particular size and depth, and is helpful for rapidly excluding benign soft-tissue masses such as ganglions and lipomas. Experienced musculoskeletal radiologists, working within a sarcoma MDT, may perform image guided biopsy during this initial investigation as part of 'one-stop clinics', thus, significantly shortening the time to diagnosis
- Further 'local staging' is best performed by **MRI** scan which shows the local extent of the tumour. **CT** can be

used to gain further information about primary bone tumours, and may also be helpful if there is calcification in the tumour or if the tumour is located in the pelvis

2. **Biopsy**
   - The 'principles of biopsy' are discussed later but this can be performed in clinic by Tru-cut needle, with or without ultrasound guidance as above, or in an open manner in theatre
3. **Systemic staging**
   - **CT chest/abdo/pelvis** is used after a diagnosis of sarcoma has been made to look for metastases in lung, and less often lymph nodes, liver and other bones,
   - Other investigations are guided by tumour type. Patients with primary bone tumours routinely have an **isotope bone scan** to rule out multifocal or metastatic disease in bones. Ewing's sarcoma patients should also have **bone marrow aspirate** as part of staging. PET scans are being used more frequently, particularly when the presence of otherwise undetected metastatic disease might influence treatment

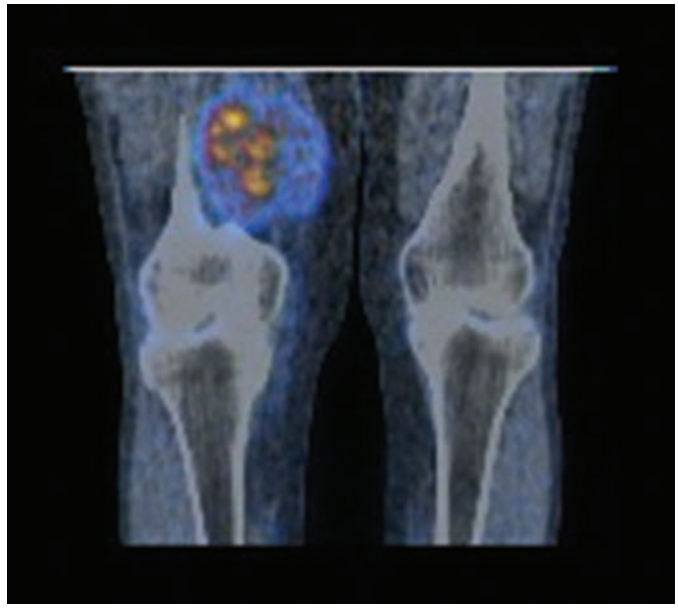

Figure 20.11 PET–CT scan showing increased uptake in a leiomyosarcoma of the right thigh

Imaging modalities – when to use which?

Magnetic resonance imaging (MRI)

- Shows the local extent and anatomical relationships of the tumour, including critical neurovascular structures, compartments and local joints
- Shows further pathological detail, the extent of local oedema, and the presence or absence of skip/satellite lesions and local lymphadenopathy
- Is critical for planning surgical procedures and margins
- Is excellent for viewing soft tissues but less good at delineating bony architecture

Computerized tomography (CT)

- Is better than MRI at imaging bone where its use can add further important information, e.g. showing the nidus of an osteoid osteoma, or endosteal scalloping and matrix formation in cartilaginous tumours

Positron emission tomography (PET)

- PET scanning gives metabolic information. It involves injecting a positron-emitting radionuclide tracer attached to biologically active molecule (usually fluorodeoxyglucose (FDG)). The biological molecule is taken up by active tissues, including the tumour, and the radionuclide then undergoes beta decay, emitting a positron. The positron travels a short distance through the tissues losing kinetic energy until it decelerates enough to interact with an electron, releasing a pair of gamma photons in opposite directions. These photons are then detected by the scanning device. The scan is usually performed in conjunction with a CT scan and the active areas on the PET scan are then correlated with the anatomy on the CT scan (Figure 20.11)
- PET scanning in orthopaedic oncology is unusual as a primary investigation but can be useful to detect recurrent or metastatic disease and is becoming more widely used

How to perform a biopsy

- A biopsy is performed to obtain a histological diagnosis and is critical to guide management
- Biopsy planning is important as all tissue contaminated by the biopsy and its track must be removed during the definitive resection. As such, it should be performed by, or after discussion with, the specialist MDT
- Biopsy can be excisional, incisional or, most often, percutaneous, but must adhere to the following principles
  1. Incisions should be placed longitudinally on limbs and positioned such that they can be removed *en bloc* during the definitive resection
  2. The biopsy track must only pass through a single muscle compartment
  3. The biopsy track must not contaminate critical neurovascular structures
  4. For open biopsy, close attention must be paid to haemostasis and tissue dissection should be kept to a minimum to reduce local tissue seeding
  5. A drain, if sited, must be placed in the line of the incision
  6. Biopsies for sarcoma should be discussed with the pathologist to ensure tissue is sent fresh if appropriate

- **Percutaneous biopsy**, e.g. by Tru-cut needle, is the most popular method of biopsy in the UK. It reliably provides enough tissue for diagnosis and has a low complication rate. Assessment of necrosis and mitotic rate is less reliable on core needle than incision biopsy, but this seldom influences management
- **Incision biopsy** is generally only performed if percutaneous biopsy is non-diagnostic, further information is required, or to obtain material for research
- **Excision biopsy**, where the entire lesion is removed, is reserved for benign lesions where imaging has been diagnostic or for small superficial tumours for which an excision biopsy would not compromise later re-excision. If there is any dubiety then an incision or percutaneous biopsy must be performed prior to excision

How to 'stage' a lesion

Staging of benign bone tumours[3]

- Benign bony tumours were described, by Enneking et al., as latent, active, or aggressive[3]
  - **Latent** lesions are asymptomatic and are often incidental findings. They are often treated with observation only, e.g. bone island, osteoma of skull, non-ossifying fibroma
  - **Active** lesions cause symptoms including pain and swelling, and are often treated by curettage and grafting, e.g. osteoblastoma or chondroblastoma
  - **Aggressive** lesions are symptomatic and are locally destructive. Treatment usually involves curettage and grafting, but may require *en bloc* resection and reconstruction, e.g. giant cell tumour of bone

Staging of sarcomas

- The simplest staging system is that described by Enneking et al.[3]
- The Enneking/Musculoskeletal Tumor Society (MSTS) system uses grade (low or high), site (intra-compartmental or extra-compartmental) and presence or absence of metastases (defined as any skip lesions, regional lymph nodes or distant metastasis)
  - **Low-grade** tumours are defined as **stage I**
  - **High-grade** tumours are defined as **stage II**
  - **Metastatic** tumours are defined as **stage III**
  - **Intracompartmental** tumours are further classified as **"A"**
  - **Extracompartmental** tumours are further classified as **"B"**
  - The MSTS staging system is equally applicable to bone and soft-tissue tumours (Table 20.2)
- The American Joint Committee on Cancer (AJCC, now version 7) staging systems are also widely used. They are more complicated and probably beyond the scope of the FRCS examination but are included here for completeness and to enhance understanding
  - **The AJCC system for bone tumours** classifies tumours as stage I–IV using the size, the presence or absence of regional lymph node involvement, the presence or absence of distant metastasis, and the histologic grade of the tumour[4] (Table 20.3)
    - The size of tumour is designated T_1 if it is ≤8 cm in maximum diameter, T_2 if it is >8 cm, and T_3 if there are multiple or discontinuous tumours
    - Lymph node involvement is designated N_0 if there is no nodal involvement, and N_1 if there is regional lymphatic spread
    - Metastasis is similarly designated M_0 if there is no evidence of metastasis, and M_1 if there is evidence of metastatic disease
    - Grade is designated G_1 for well-differentiated tumours, G_2 for moderately differentiated tumours, G_3 for poorly differentiated tumours, and G_4 for undifferentiated tumours
  - **The AJCC system for soft-tissue tumours** classifies tumours as stage I–IV using the size, site, the presence or absence of regional lymph node involvement, the presence or absence of distant

Table 20.2 MSTS staging system[3]

Stage	Description	Grade	Site	Metastases
IA	Low-grade, intracompartmental	G_1	T_1	M_0
IB	Low-grade, extracompartmental	G_1	T_2	M_0
IIA	High-grade, intracompartmental	G_2	T_1	M_0
IIB	High-grade, extracompartmental	G_2	T_2	M_0
III	Any grade, metastatic	G_{1-2}	T_{1-2}	M_1

Table 20.3 AJCC staging system for bone tumours[4]

Stage	Grade	Size	Nodes	Metastasis
IA	G_1 or G_2	T_1	N_0	M_0
IB	G_1 or G_2	T_2	N_0	M_0
IIA	G_3 or G_4	T_1	N_0	M_0
IIB	G_3 or G_4	T_2	N_0	M_0
III	Any G	T_3	N_0	M_0
IVA	Any G	Any T	N_0	M_1 (lung only)
IVB	Any G	Any T	Any N	Any M

metastasis, and the histologic grade of the tumour[5] (Table 20.4)

- The size of tumour is designated T_1 if it is ≤5 cm in maximum diameter and T_2 if it is >5 cm. Further description classifies superficial lesions as T_{1a} or T_{2a} and deep lesions as T_{1b} or T_{2b}
- Lymph node involvement is designated N_0 if there is no nodal involvement, and N_1 if there is regional lymphatic spread
- Metastasis is similarly labelled M_0 if there are no metastases, and M_1 if there is evidence of metastatic disease
- Grade is described as G_1 for well-differentiated tumours, G_2 for moderately differentiated tumours, G_3 for poorly differentiated tumours

Lymphatic spread?

NB. Soft-tissue sarcomas normally metastasise via haematogenous routes to the lungs. Lymphatic metastases are rare but are more often seen in the following five histological types:

- Angiosarcoma
- Synovial sarcoma
- Rhabdomyosarcoma
- Epithelioid sarcoma
- Clear-cell sarcoma

Table 20.4 AJCC staging system for soft-tissue tumours[5]

Stage	Grade	Size	Nodes	Metastasis
IA	G_1	$T_{1a, 1b.}$	N_0	M_0
IB	G_1	$T_{2a, 2b.}$	N_0	M_0
IIA	G_2 or G_3	$T_{1a, 1b.}$	N_0	M_0
IIB	G_2	$T_{2a, 2b.}$	N_0	M_0
III	G_3	T_{2a}, T_{2b}	N_0	M_0
III	Any G	Any T	N_1	M_0
IV	Any G	Any T	N_0	M_1

Primary bone tumours

Introduction[6]

Epidemiology

- Primary malignant bone tumours are rare
  - <600 per year in the UK
  - 0.2% of all malignancies, but 4% of malignancies in children under 14 years of age
  - More common in males. Males (58%): female (42%) = 13 : 10
  - Incidence is about 8/1 000 000 per year
  - Overall, 38% occur in the long bones of the lower limb, 16% in the bones of the pelvis, sacrum and coccyx and 14% in the scapula and long bones of the upper limb.
    - The pattern changes with age; however, such that in patients under the age of 20, 70% of bone tumours occur in the extremities, compared with 40% in patients over 40 (Figure 20.12)
  - The four most common tumours are osteosarcoma, chondrosarcoma, Ewing's sarcoma and chordoma (Figure 20.13)
    - Incidence of osteosarcoma peaks in childhood with a second peak in late adult life.
    - Ewing's sarcoma has a peak incidence in adolescence and early adult life but is extremely rare after the age of 30.
    - The incidence of chondrosarcoma and chordoma increases with age.
  - Overall, cumulative 5-year survival is 58% for men and 59% for women for all bone sarcomas.

How do bone tumours present?

- Swelling. Initially soft-tissue oedema. Later bony enlargement and soft-tissue extension
- Pain, typically worse at night. Often mistaken for growing pains
- Occasionally cachexia and weight-loss
- Coincidental injury. Not causative but may bring attention to swelling

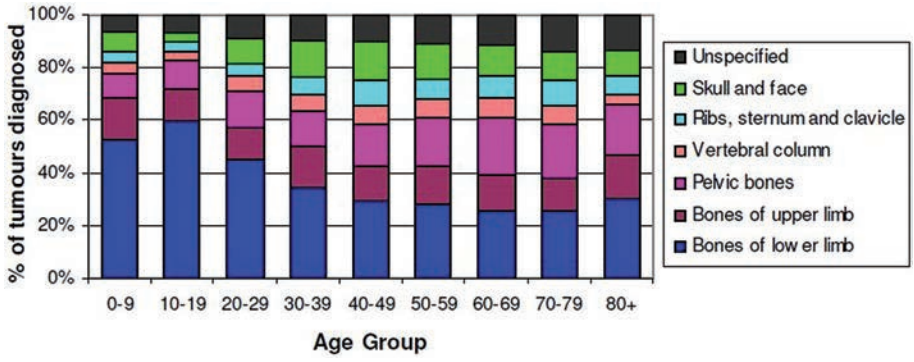

Figure 20.12 Proportion of bone sarcomas diagnosed by age group and anatomical site (England: 1985–2009). Reproduced with permission of Public Health England

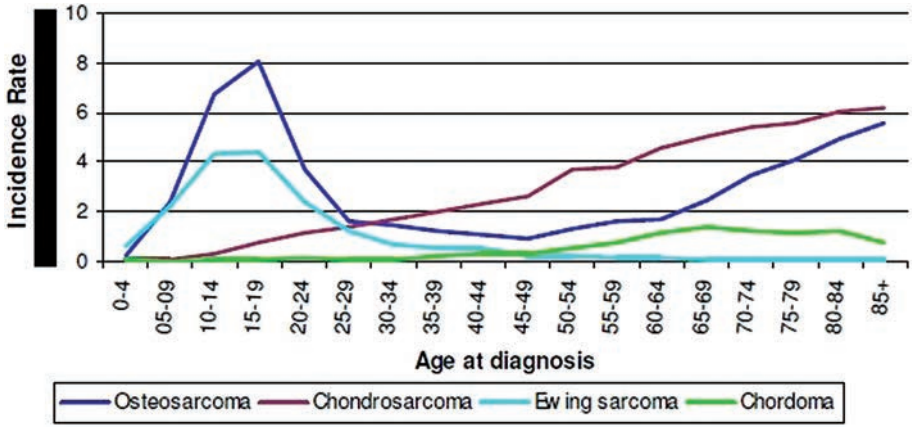

Figure 20.13 Age-specific incidence of the most common bone sarcoma variants (England: 1985–2009). Reproduced with permission of Public Health England

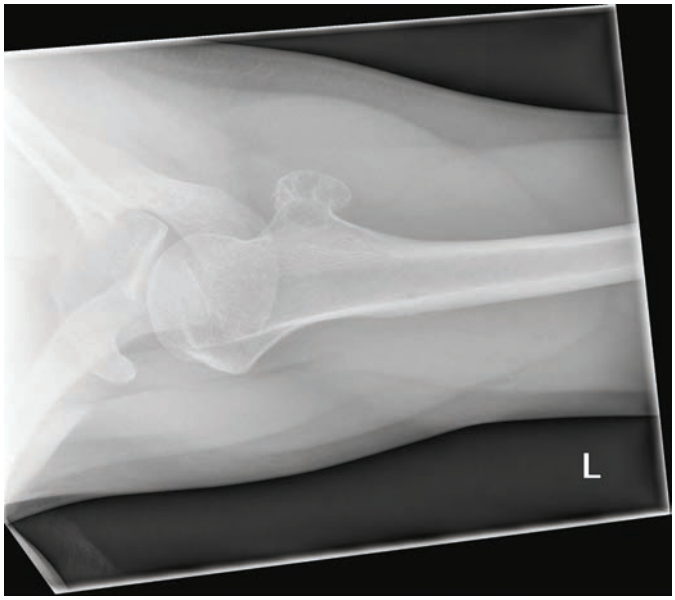

Figure 20.14 Osteochondroma left proximal humerus

- Pathological fracture (5–10%)
- Rarely, symptoms of metastasis to lung. Shortness of breath, chest pain, haemoptysis
- Occasionally neuralgia or paraesthesia from nerve compression

Types of bone tumours

The WHO classification is extremely detailed but bone tumours can more easily be classified in to four main categories:

- Cartilage-forming tumours
- Bone forming tumours
- Fibrous tumours
- Non-matrix producing tumours

Cartilage-forming tumours[7]

Developmental or hamartomatous tumours

Solitary osteochondroma

- Osteocartilaginous exostosis
- Follows an *EXT* gene mutation that leads to aberrant growth of a fragment of epiphyseal plate
- Persistent growth of the fragment by enchondral ossification leads to a cartilage-capped, subperiosteal, bony projection
- Manifests clinically during second decade of life, usually because of local irritation, trauma or fracture
- The cartilage cap grows under the same hormonal control as physeal plates and, therefore, ceases growing after skeletal maturity
- Most commonly affect the ends of long bones, especially around the knee. (Distal femur most common ≈25%)
- X-ray appearance is of a flattened (sessile) or pedunculated (on a stalk), juxta-articular protuberance, growing away from the adjacent joint. **The cortex of the lesion is continuous with the cortex of the bone, as is the medullary cavity** (Figure 20.14)
- If symptomatic, treatment is by surgical excision through the base of the lesion. Recurrence is more likely if the cartilage cap is incompletely excised and the patient has not reached skeletal maturity
- Malignant transformation to chondrosarcoma is exceptionally rare in solitary osteochondromas, but persistent growth after skeletal maturity or a thick cartilage cap (1.5–2.0 cm) are suspicious features which should prompt referral to a bone tumour centre

Multiple osteochondromas/hereditary multiple exostoses (Figure 20.15)

- Autosomal dominant trait of multiple osteocartilaginous exostoses
- Majority are familial but up to 20% occur due to sporadic mutations
- HME is caused by a mutation to one of three genes, *EXT1 (8q)*, *EXT2 (11p)* and *EXT3 (19q)*
- The most common skeletal dysplasia (and, therefore, likely to come up in clinical exams)
- Short stature with bony deformity and disfigurement (often a short ulna)

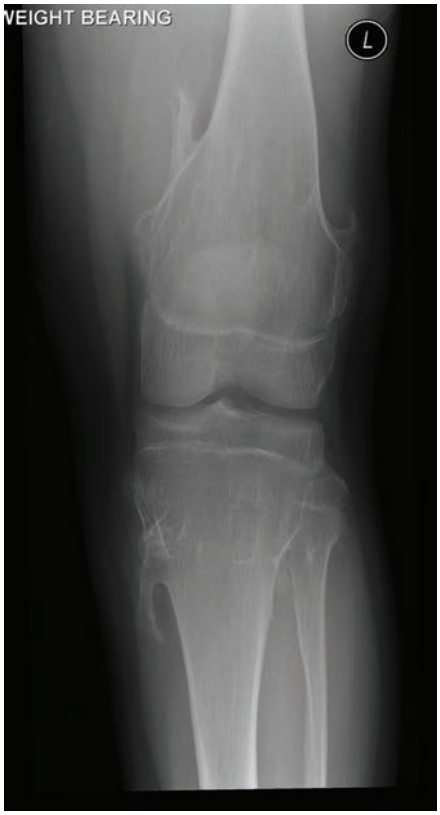

Figure 20.15 Hereditary multiple exostoses. Multiple ostoechondromas are seen around the left knee

- Grossly and radiographically similar to solitary osteochondromas but histologically are often more disorganized in structure with bosselated caps
- Treatment for symptomatic lesions is surgical excision as for solitary osteochondromas
- Lesions that continue to grow after physeal closure raise the suspicion of malignant transformation. Whilst still rare, this occurs more frequently than in solitary osteochondromas, arising in 1–5% of patients

Enchondromatosis

- Non-familial, sporadic, multiple cartilaginous tumours
- Often unilateral, confined to one limb. Usually peripheral affecting the small bones of the hands or feet
- Histologically more cellular and myxoid than solitary enchondromas (see benign tumours) and consequently the rate of malignant transformation is higher
- Enchondromatosis = **Ollier's disease** (Figure 20.16)
  - Incidence 1:100 000
  - Risk of bone malignancy is 10–15%
  - Also increased risk of visceral and CNS malignancies
  - Overall risk of malignancy is 25%
- Enchondromatosis + haemangiomas = **Maffuccis syndrome**
  - Risk of bony malignancy is 25–30%
  - Overall risk of malignancy, including CNS and visceral, approaches 100%

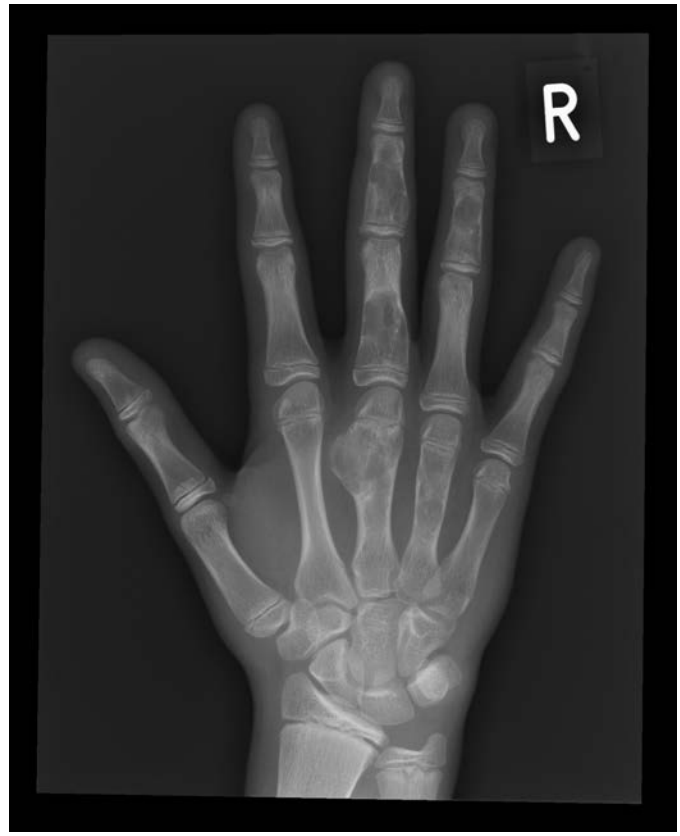

Figure 20.16 Hand x-ray of a patient with Ollier's disease showing multiple enchondromas of the small bones of the right hand

Benign cartilaginous tumours

Enchondroma

- Common, solitary, asymptomatic, intramedullary, cartilaginous tumours
- 50% occur in the hands
- When present in long bones, difficult to distinguish from low-grade chondrosarcoma
- Malignant transformation is rare, usually in large lesions in long bones
- Often present after pathological fracture, particularly in the small bones of the hands and feet
- X-ray shows a well-defined lucent lesion (short zone of transition) with stippled calcification (see Figure 20.7)
- Lesions found incidentally with no concerning features on MRI (oedema, cortical erosion) can be observed
- Biopsy is indicated if there is any doubt about the behavior of the lesion or it is symptomatic
- Surgical treatment is by curettage with or without grafting

Chondroblastoma

- Relatively rare entity, accounting for 1% of all primary bone tumours presenting in the second decade
- Most common around the hip, shoulder, or knee joint (Figure 20.17)

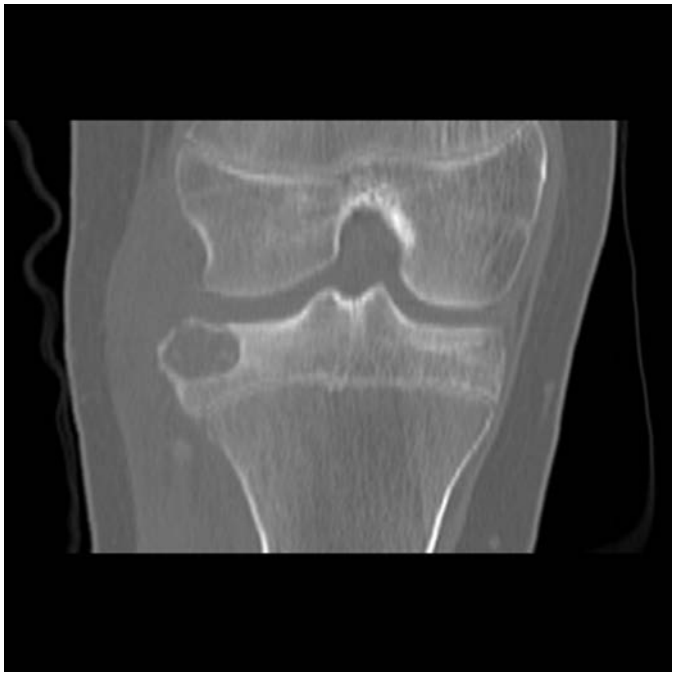

Figure 20.17 CT scan showing a well-circumscribed lesion in the epiphysis of a skeletally immature patient. There are flecks of calcification within the lesion which is classic in position and appearance of a chondroblastoma

- Well-defined lytic lesion with stippled calcification confined to epiphysis of a long bone
- Biopsy shows scattered giant cells in a field of mononuclear cell described as 'chicken wire calcification'
- Treatment by curettage with or without grafting

Malignant cartilaginous tumours[7]

Chondrosarcoma[8] (Figures 20.18–20.20)

- Malignant tumour producing a cartilaginous matrix (Figure 20.18)
- Most common in fifth and sixth decades
- Most common in pelvis, femur and humerus
- Present with persistent pain and swelling
- Lucent metaphyseal/diaphseal defect with endosteal scalloping and stippled calcification
- Bright on T2 MRI imaging because cartilaginous component has a high water content (like articular cartilage)
- Diagnosis critically depends on discussion of clinical, radiological and histological features. Can be difficult to distinguish enchondroma from chondrosarcoma on histological grounds alone

Grade I

- Low-grade chondrosarcoma
- Distinguished from enchondromas by location (long bones/pelvis/scapula and ribs), and microscopic evidence of haversian invasion
- Rarely metastatic
- 5-year survival >90%

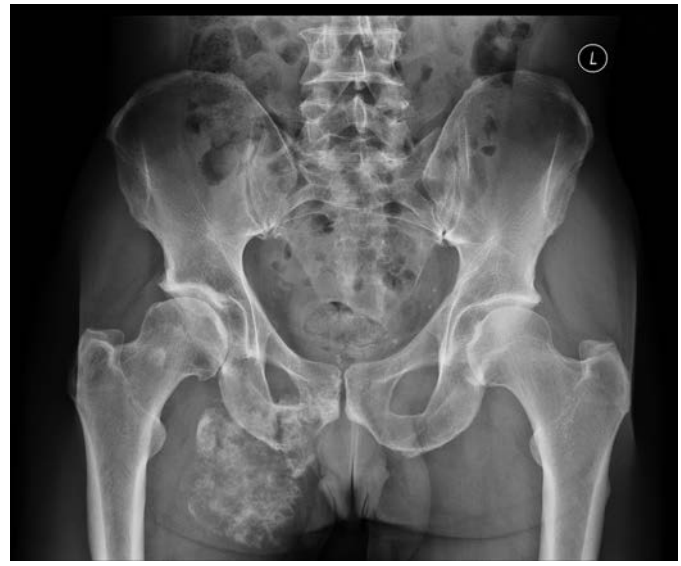

Figure 20.18 Chondrosarcoma of the right inferior pubic ramus

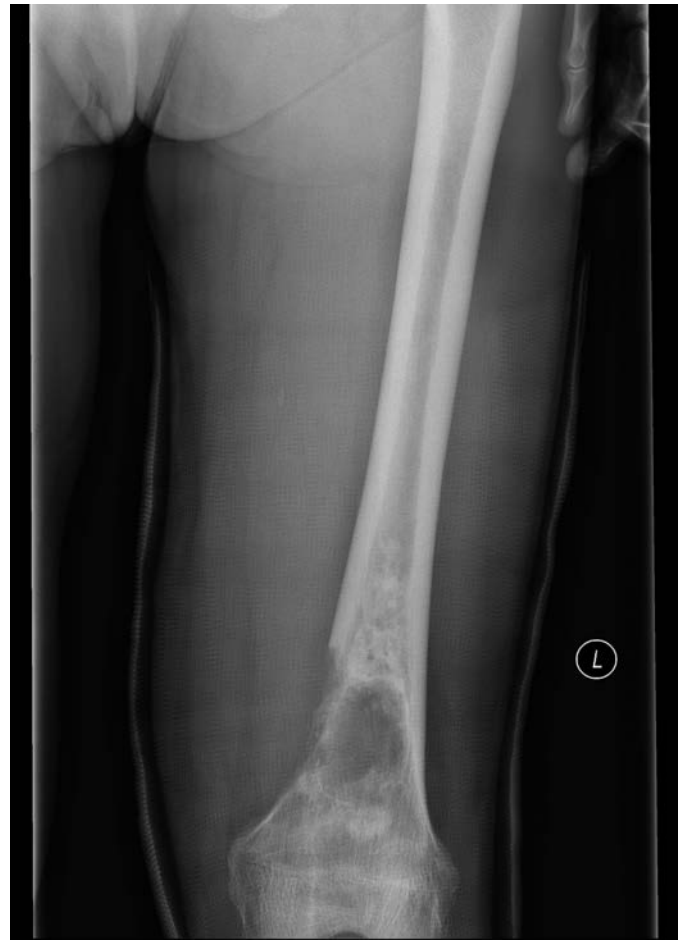

Figure 20.19 Dedifferentiated chondrosarcoma. An area of lucency with overlying cortical destruction is seen within a pre-existing enchondroma

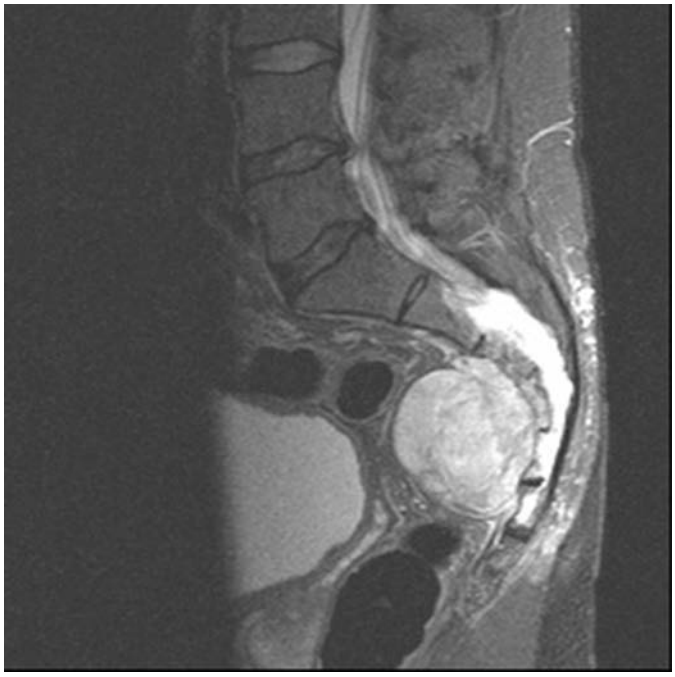

Figure 20.20 T2 sagittal MRI showing a lesion on the anterior surface of the sacrum which appears destructive with a wide zone of transition

Grade II

- Definite increased cellularity and nuclear size. Focal myxoid change is also seen
- Metastatic in 10–33%
- 5-year survival 80%

Grade III

- Marked hypercellularity, cellular atypia, and high mitotic activity
- Aggressive, rapidly enlarging
- Metastatic in 70%
- 5-year survival 30%

- Grade 1 tumours rarely metastasise and are often treated by intralesional curettage
- Higher grade tumours are treated by wide surgical excision. Chondrosarcomas are poorly sensitive to radiotherapy and there is no active chemotherapy treatments
- Ten per cent undergo de-differentiation in one area, becoming highly malignant sarcomas with spindle cells and bizarre giant cells (similar to fibrosarcoma or malignant fibrous histiocytoma), x-ray showing an area of lucency within classic stippled calcification. Dedifferentiated chondrosarcoma characteristically presents as a pathological fracture in the elderly (Figure 20.19). Prognosis is dismal
- Other rare variants include
  - **Mesenchymal chondrosarcoma**
  - **Clear-cell chondrosarcoma**

Bone-forming tumours[7]

Reactive or post-traumatic lesions

Subungual exostosis

- Osteocartilaginous lesion of the distal phalanx
- Treatment is by excision and recurrence is common if incomplete

Developmental or hamartomatous lesions

Bone islands

- Solitary enostosis. Small area of increased density within an area of cancellous bone
- Usually only 1–2 mm diameter
- **Osteopoikilosis** = multiple bone islands
  - Rare, autosomal dominant, asymptomatic condition
  - Sometimes seen with cutaneous nodules suggesting a generalized mesenchymal defect

Benign tumours

Osteoid osteoma

- Benign, painful, solitary diaphyseal lesions, usually of the femur or tibia (>50%) and located in the cortex
- Usually present in the third decade
- Pain is classically worse at night and often entirely relieved by non-steroidal anti-inflammatory drugs, reflecting the role of prostaglandins in the pain response
- X-rays and CT scan show a central lucent nidus within thickened sclerotic cortex (<1 cm). Isotope bone scan shows increased uptake
- These lesions usually 'burn-out' over time and, thus, conservative treatment with NSAIDs is an option. However, given the level of symptomatology experienced by patients, treatment is often required
- Treatment is by CT-guided radioablation or by surgical excision, often using a 'burr-down' technique

Osteoblastoma

- Benign, painful, osteoid-forming neoplasm, usually of the spine (40%) or long bones (Figure 20.21)
- Spinal lesions originate in the vertebral arch, rarely affect the vertebral body, and can lead to radicular or myelopathic symptoms
- X-ray and CT show a lucent lesion with a central density, similar to osteoid osteoma, but larger in size (i.e. >1 cm)
- Treatment is by curettage and grafting or *en bloc* excision

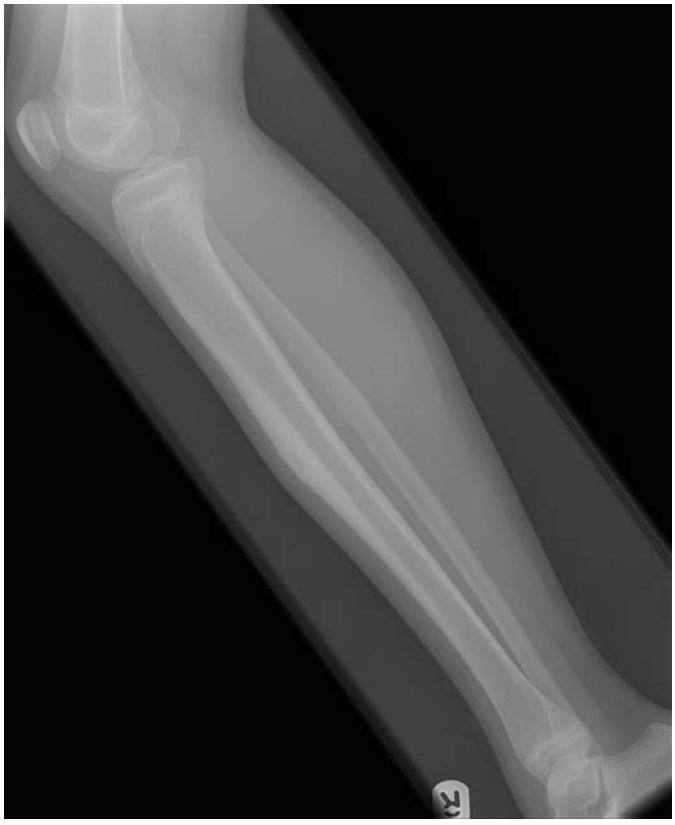

Figure 20.21 Osteoblastoma of the right tibia. Note the cortical sclerosis with a central lytic nidus. Very similar in appearance to an osteoid osteoma but with a bigger nidus

Malignant tumours

Osteosarcoma

- Second most common primary bone tumour (myeloma is first)
- Twenty per cent of all primary bone tumours (all ages), but 55% of all bone tumours in children and adolescents. The most common malignant bone tumour between ages 10 and 19
- Distal femur (35%) > prox. tibia (20%) > prox. humerus (10%)
- Bimodal distribution: first large peak during rapid growth in adolescence. Second smaller peak in sixth decade because of **Paget's sarcoma** and **radiation sarcoma**
- Seventy per cent do not have radiologically detectable metastases at presentation
- Ninety-five per cent intramedullary
  - of which 90% are high grade
    - Of these, 50% are osteoblastic, and the remainder are chondroblastic, fibroblastic, small-cell or telangiectatic/giant-cell.
- Five per cent surface
  - of which 90% are low grade
- Associated with retinoblastoma and pro-geroid/pro-cancerous syndromes: Li–Fraumeni, Rothmund-Thompson, Bloom's
- X-ray of 'classic' intramedullary osteogenic sarcoma shows a sclerotic, destructive lesion in the metaphysis of a long bone invading the cortex and extending into the soft tissues ('sunburst' pattern)
- Increased alkaline phosphatase levels are associated with a poorer prognosis as are elevated lactate dehydrogenase levels
- Treament is with neo-adjuvant chemotherapy (involving an anthracycline (doxorubicin), a platinum (cisplatin) and high dose methotrexate), surgery and adjuvant chemotherapy. Other chemotherapeutic agents (e.g. etoposide and ifosfamide) are sometimes added to the regime for poor responders during adjuvant chemotherapy
- Local treatment involves complete excision of the primary tumour. Limb sparing is usually feasible
- A good response to chemotherapy is >90% necrosis and is associated with a better prognosis than poor response
- Metastatic disease is treated by resection where possible
- **Mifamurtide,** muramyl tripeptide phosphatidylethanolamine (MTP-PE), is a synthetic analogue of a component of mycobacterial cell wall
  - It simulates a bacterial infection, stimulating the immune system, particularly white cells and causing the release of TNF-α and numerous interleukins
  - Activated white cells then attack cancer cells
  - Use granted by NICE in 2011 for non-metastatic disease, treated with complete excision and chemotherapy
  - Controversy around the clinical trial, which did show an absolute risk reduction of death of 8%
  - Infection post-resection, which MTP-PE simulates, has been shown in both human and veterinary literature to improve survival

Fibrous tumours[7]

Developmental or hamartomatous tumours

Non-ossifying fibroma

- Very common (up to 35% incidence in normal children)
- Asymptomatic (but can present after pathological fracture), well-demarcated, solitary, eccentrically placed metaphyseal lesion
- X-rays show a lucency with a sclerotic margin
- Treatment is by observation, unless risk of pathological fracture, as lesions usually resolve by adulthood

Fibrous dysplasia

- Common, usually monostotic, fibro-osseous lesion, affecting long-bones of the lower limb (50%)
- Often an incidental finding but can present with swelling or pathological fracture usually in adolescence

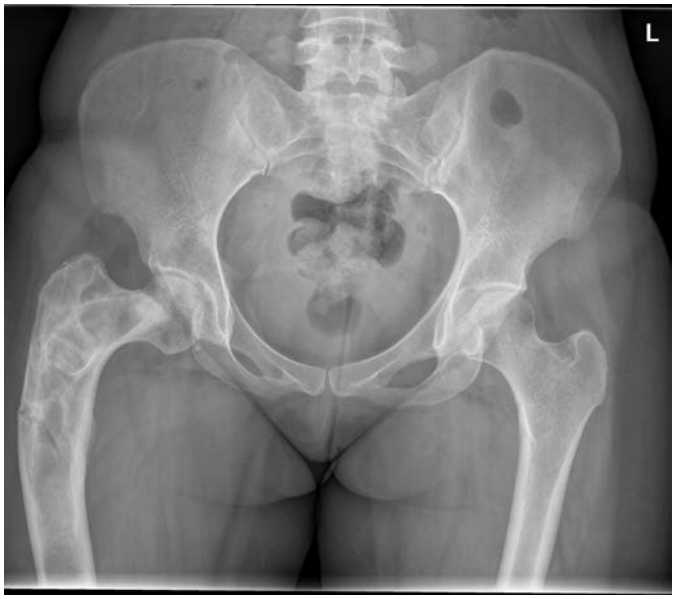

Figure 20.22 Fibrous dysplasia of the right proximal femur showing characteristic pathological fracture and developing shepherds crook deformity

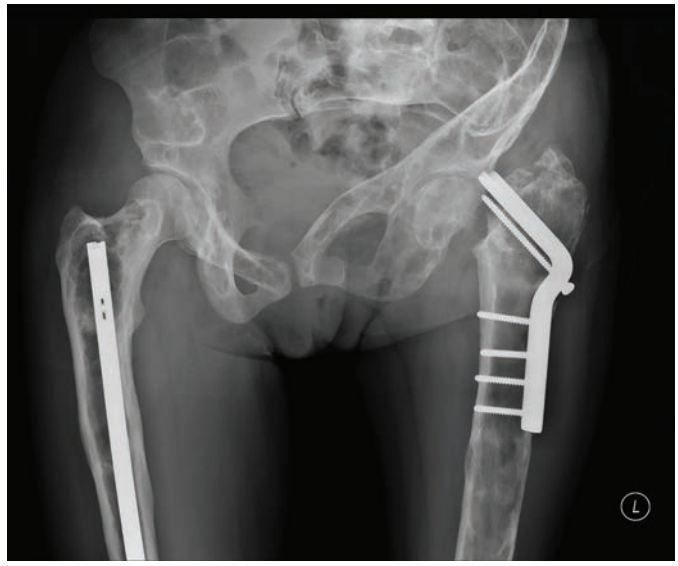

Figure 20.23 McCune–Albright syndrome. Polyostotic fibrous dysplasia is seen in the femurs and pelvis with widespread bony changes and deformity

- X-ray shows a well-demarcated, fusiform expansion, with ground-glass calcification and cortical thinning
- Histology reveals abnormal, thin bony trabeculae described as 'Chinese letters'
- The classic 'shepherd's crook', deformity of the upper femur, resulting in coxa vara, is caused by multiple, sequential pathological fractures (Figure 20.22)
- Polyostotic involvement is seen in **McCune–Albright syndrome** (Figure 20.23)
  - non-familial, resulting from a post-zygotic mutation to *GNAS1* (20q)
  - Syndrome characterized by polyostotic fibrous dyplasia, hormonal disturbances including precocious puberty, and café-au-lait spots, which have a jagged edge ('coast of Maine'), are unilateral, and do not cross the midline
  - Fibrous dysplasia most commonly affects the lower limbs and pelvis but can also frequently affect the skull and facial bones

Benign tumours

Osteofibrous dysplasia

- Painless swelling of the tibia (>85%)/fibula of young children affecting the metaphysis/diaphysis but not the epiphysis
- X-ray shows intracortical, multiloculated lesion with osteolysis and thinning often with anterior bowing
- Can undergo malignant transformation to adamantinoma
- Treatment is with curettage (± grafting) but there is significant risk of local recurrence, particularly if this is performed before skeletal maturity

Malignant tumours

Fibrosarcoma

- Rare, malignant spindle-cell tumour affecting the ends of long bones (50% around the knee), in the third to sixth decades
- X-rays show lucent lesions with cortical destruction and extension into the soft-tissues, a mottled appearance of the bone and indistinct margins
- Treatment is by wide excision and reconstruction

Malignant fibrous histiocytoma

- Rare, pleomorphic spindle-cell tumour (storiform or 'starry-night' pattern) affecting the metaphysis of long bones around the knee in adults (any age)
- X-ray shows a poorly defined lucent lesion with cortical destruction, but minimal periosteal new bone formation
- Treatment is as for osteosarcoma with chemotherapy, wide excision and reconstruction

Adamantinoma

- Rare, slow-growing (pain developing over many years), low-grade spindle-cell neoplasm affecting the tibia in >90% of cases (Figure 20.24)
- X-ray shows a multicystic ('soap-bubble') lucency with cortical thinning and expansion (this is different to the central, metaphyseal soap bubble appearance of unicameral bone cysts)
- Locally aggressive but can metastasise in 20% of cases late in its course
- Treatment is with wide excision and reconstruction

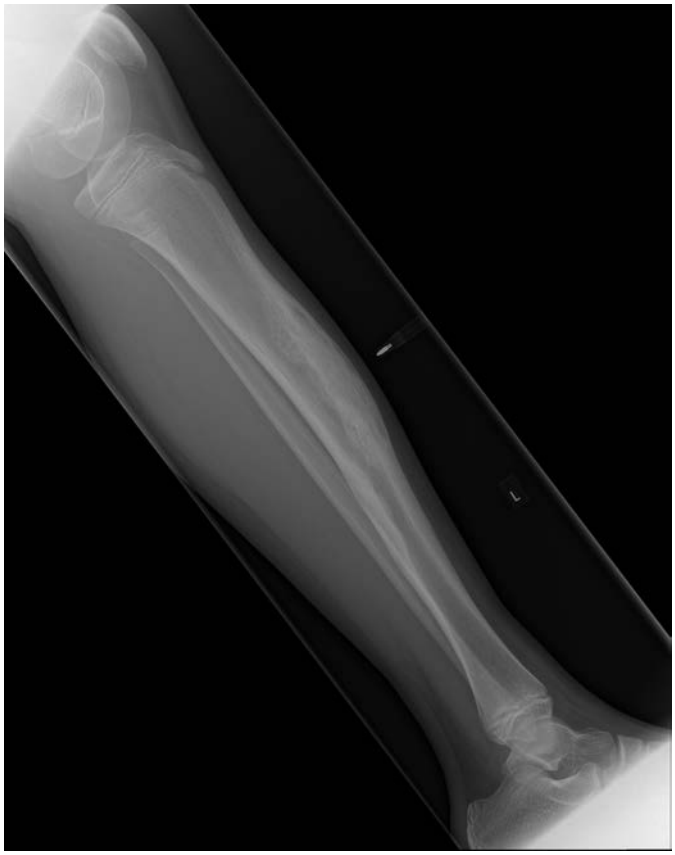

Figure 20.24 Adamantinoma of the left tibia, arising in an area of osteofibrous-dysplasia showing the characteristic anterior bow. With the 'eye of faith', the proximal extent appears like soap bubbles

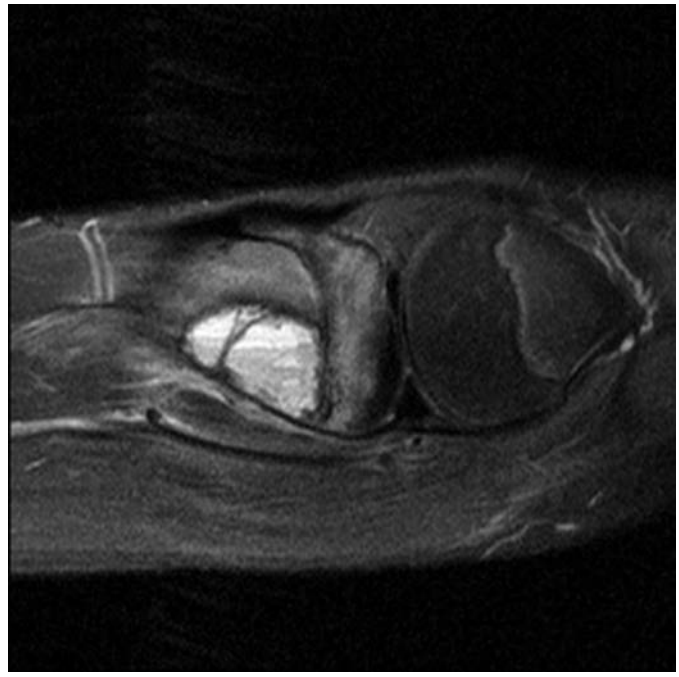

Figure 20.25 ABC of the proximal tibia in a skeletally immature patient. The lesion is metaphyseal, eccentric and has multiple fluid–fluid levels

Non-matrix-producing tumours[7]

Reactive or post-traumatic lesions

Unicameral bone cyst

- Solitary, cystic bone lesions found in the metaphyses of long bones in childhood and adolescence. Not seen in adults
- Classic site is prox. humerus (50%). Other common site is prox. femur (25%), fractures of which can be difficult to manage
- Usual presentation is with pain (usually indicates impending fracture), actual fracture or as an incidental finding
- Fractures usually heal, but the cyst usually only partly resolves
- X-ray shows well-defined lucent area with a geographical, sclerotic margin, sometimes with a 'fallen leaf' sign
- Treatment is by observation for the majority. Surgical intervention is indicated for those at risk of fracture. Options described include minimally invasive decompression and curettage (preferred option), curettage and grafting, surgical fixation through the cyst with an intramedullary nail or corticosteroid injection

Aneurysmal bone cyst

- Solitary, expansile, multiloculated, eccentrically placed, cystic lesion, usually of the long bones or spine (15%) (Figure 20.25)
- Presents with pain and swelling usually before the third decade
- X-rays show an expansile lesion with a trabeculated appearance. MRI shows loculations with fluid levels
- In many cases the lesion is secondary and reactive to another benign lesion (e.g. osteoblastoma, chondroblastoma, fibrous dysplasia, giant cell tumour)
- **Must distinguish** from **telangiectatic osteosarcoma** which can have aneurysmal change within it
- Treatment is by curettage and grafting but recurrence rate can be as high as 50%

Developmental or hamartomatous tumours

Haemangioma of bone

- Solitary, asymptomatic lesions, usually affecting the vertebral bodies or skull (lower thoracic most common) comprising thin-walled cavernous blood vessels
- X-rays show accentuated, thickened vertical trabeculae = 'Jail Bar' on coronal/sagittal imaging and 'salt and pepper' on axial imaging
- No treatment is usually required but pathological fracture can occur

- Widespread disease can occur (**skeletal haemangiomatosis/lymphangiomatosis**), but has no known familial tendency and is self-limiting

Benign tumours

Eosinophilic granuloma

- Solitary (80%) bony lesions of unknown etiology (possibly reactive or inflammatory) classically seen in males in the first decade usually in the proximal femur, spine, skull or ribs
- Can be multi-focal and include soft-tissues including skin, lymph nodes and lung
- X-rays show one, or more, well-defined lucencies. In the spine, secondary vertebral collapse is often seen (vertebra plana). There may be an associated periosteal reaction
- Microscopically contains histiocytes and multinucleated giant cells (Langerhan's)
- Unifocal lesions usually regress spontaneously, particularly after biopsy, but curettage and grafting may be required

Giant cell tumour of bone

- Rare (1–2 per million per year) solitary, locally aggressive lesion seen at the epiphyseal ends of long bones (typically distal femur 25%, proximal tibia 25% and distal radius 10%) in the third decade
- X-ray shows an aggressive lytic lesion in the metaphyseal/epiphyseal part of the bone, usually juxtaarticular. There is often complete cortical destruction with an associated neocortex (Figure 20.26)
- Histologically, the tumour comprises spindle-shaped tumour cells and multinucleated giant cells, which are indistinguishable from osteoclasts. These are recruited from the monocyte–macrophage population. Agents which interfere with osteoclast recruitment (e.g. bisphosphonates and RANKL inhibitors) may be helpful in difficult cases
- **Must distinguish** from **giant-cell rich osteosarcoma**
- Denosumab is a human monoclonal antibody designed to inhibit RANKL. It blocks the osteoclastic action of the multinucleated giant cells within the tumour, thus, leading to reossification of the defect. Relapse occurs when the drug is stopped
- Treatment is by curettage and grafting and the use of a surgical adjuvant (e.g. high-speed burr, liquid nitrogen, cement). Local recurrence rates can be high, particularly in difficult sites such as the pelvis, spine and distal radius
- Malignant transformation and metastasis is a rare (<5%) but well-recognised complication. Locally recurrent tumours are associated with a higher risk of metastatic disease

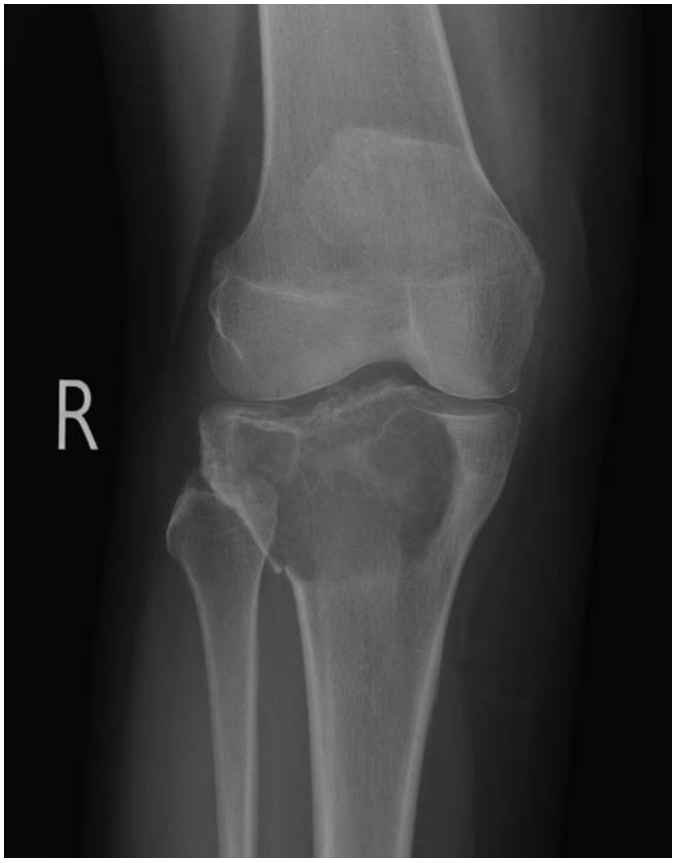

Figure 20.26 GCT of the proximal tibia showing locally aggressive behaviour but a narrow zone of transition. There is a pathological fracture but no obvious periosteal reaction or soft-tissue extension

Malignant tumours

Ewing's sarcoma/PNET

- Malignant, small round blue cell neoplasm of bone (rarely of soft tissue) presenting most commonly in the metaphyses/diaphyses of the femur (25%), tibia (10%), humerus (10%) and in the pelvis (10%). Most common malignant bone tumour under the age of 10. Second most common tumour between the ages of 10–19 (Osteosarcoma No. 1)
- Presentation is with pain and swelling, but anaemia, fever, a raised ESR and a leukocytosis may also be present incorrectly suggesting infection as the diagnosis
- X-ray shows a lytic, moth-eaten appearance with laminated periosteal bone reaction ('onion peel'). MRI shows the local extent of the tumour and depth of soft-tissue involvement
- Investigation for Ewing's also includes whole body isotope bone scan and a bone marrow biopsy to rule out metastatic disease. Bone marrow involvement is associated with a poorer prognosis
- Reciprocal translation between chromosomes is seen
  - **t(11;22)(q24;q12) = EWS-FLI1***
  - t(21;22)(q22;q12) = EWS-ERG
  - t(7;22)(p22;q12) = EWS-ETV1
  - t(17;22)(q12;q12) = EWS-FEV
  - t(2;22)(q33;q12) = EWS-E1AF
  - t(16;21)(p11;q22) = FUS-ERG

* Know this one for MCQ/EMQs

- Chemotherapy is with vincristine, doxorubicin, cyclofosfamide, ifosfamide and etoposide
- Local treatment is with wide surgical excision. Additional radiotherapy is added if margins are close. Radiotherapy is sometimes used as the primary local treatment modality where surgical excision is not possible
- Overall 5-year survival is 66% (75% with a good response to chemotherapy and 20% in those with a poor response)

Chordoma

- Arises from remnants of the notochord, so almost exclusively arise in the midline/axial skeleton (50% sacrococcygeal, 35% cranial) (see Figure 20.20)
- Slow-growing neoplasm, presenting in fifth decade
- Lytic lesion with bony destruction and focal calcification
- Systemic metastases in 50% to lymph nodes, lung, liver and bone
- Chordomas are generally considered radio-insensitive and radiation dose is limited due to adjacent neurological structures. However, proton beam therapy has shown early promise in this tumour type and there are good results using carbon ion radiotherapy
- Surgical excision is the only curative treatment, but needs to be balanced with the morbidity, given the proximity of vital neurological structures

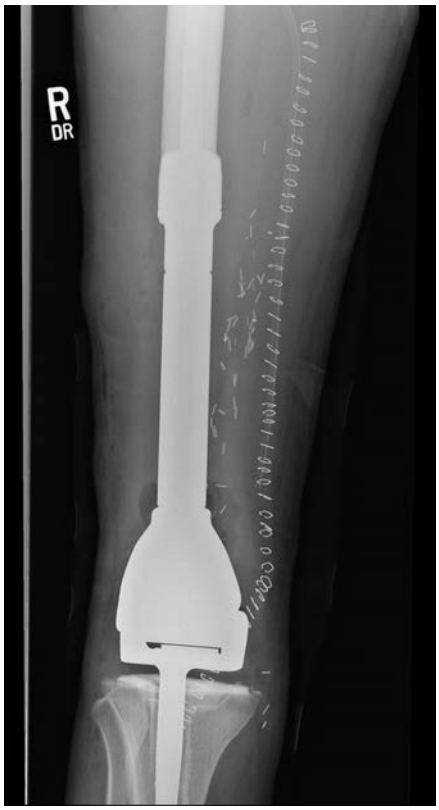

Figure 20.27 Right distal femoral replacement performed after resection of the osteosarcoma seen in Figure 20.4

Treatment of primary bone sarcomas

Reconstructive/ limb-salvage surgery[9,10]

- Safe margins must not be compromised for a preferred functional or reconstructive outcome
- Limb-salvage rates 90% in most centres
- Amputation is reserved for tumours that extensively involve neurovascular structures, or where safe margins cannot be achieved. In osteosarcoma, assessment of a safe margin depends on the response of the tumour to neoadjuvant chemotherapy as well as the thickness of the margin histologically
- Greatest experience of megaprosthesis use is in long bones esp. femur, tibia and humerus (Figure 20.27)
- Extendable prostheses are available to accommodate growth in children
- Ten-year implant survival around 75%, depending on anatomical site and length of resection. BUT: With revision procedures, durability of limb-salvage can be as high as 90% at 20 years. Survival of implants has been improved by the use of hydroxyapatite collars. Silver-coated implants may be more resistant to infection
- Secondary amputation, when required, usually follows deep periprosthetic infection or local recurrence

Adjuvant treatment

Radiotherapy

- Malignant bone-forming tumours (e.g. osteosarcoma) and malignant cartilage-forming tumours are poorly radiosensitive
- Ewing's sarcoma is classically radio and chemosensitive and radiotherapy can occasionally form the mainstay of treatment in areas where surgery is difficult (e.g. pelvis)

Chemotherapy[11]

- For **osteosarcoma**, randomised trials have demonstrated increased disease-free and overall survival in localized disease with the use of multiagent chemotherapy
  - It is recognised, however, that although most patients do not have radiologically detectable disease at presentation, many have occult micrometastatic disease. In the pre-chemotherapy era, survival was poor (20%)
  - Neo-adjuvant (pre-surgical) chemotherapy allows early and effective treatment of micrometastases and usually leads to a reduction in inflammation around the tumour, and sometimes in the size of the tumour, which helps the surgical resection
  - Standard treatment is with combination doxorubicin, cisplatin and high dose methotrexate, but where possible, patients are recruited into clinical trials
  - The response of the tumour to neo-adjuvant chemotherapy (measured as percentage necrosis on histology of the resected specimen) is prognostically

significant. Patients with >90% necrosis do better than those without
- Good responders are treated with the same agents after surgery but non-or poor responders may benefit from the addition of ifosfamide and etoposide

Immunohaematopoietic tumours

Plasmacytoma and multiple myeloma

- Most common primary bone tumour
- Multiple myeloma invariably presents in the spine, but may also present in the sternum, ribs, pelvis and skull and limbs
- Generally presents in or after sixth decade. Male 2 : 1 female
- Present with pain, anaemia, increased ESR and hypercalcaemia
- Serum electrophoresis identifies a monoclonal proteinaemia. Bence–Jones proteins (light-chain subunits of immunoglobulins) are found in the urine
- X-ray shows multiple round, lytic defects with no surrounding sclerosis or reactive bone. Classically do not show up on isotope bone scans so skeletal survey is needed
- Treatment is with steroids and chemotherapy ± bone marrow transplant. This generally improves survival only rather than effecting a cure
- A diagnosis of plasmacytoma depends on
  1. No other radiographic lesions
  2. A negative bone marrow biopsy from a separate site
  3. No significant protein or immunoglobulin abnormality on urine or serum assays
     - X-rays show a solitary, expansile, lytic lesion
     - Treatment is with radiotherapy or *en bloc* excision
     - The diagnosis of solitary myeloma is debated as 70% develop disease at multiple sites (i.e. multiple myeloma) and die within 5 years. Others can develop multifocal disease years after the index plasmacytoma

Primary non-Hodgkin's lymphoma

- Usually large B-cell lymphomas of the axial skeleton although 25% can occur in the femur
- X-rays show moth-eaten osteolysis and destruction with no periosteal reaction
- Local evaluation is by MRI and staging is by CT scan and isotope bone scan to look for multifocal disease
- Treatment is by radiotherapy ± chemotherapy
- Prognosis depends on histological subtype, grade and stage

Hodgkin's disease

- A specific type of lymphoma often presenting with pain (classically after alcohol consumption although this is actually very rare), weight loss, night sweats, lymphadenopathy, hepatosplenomegaly and 'cyclical' fever
- X-ray shows variable lesions (lytic, sclerotic or mixed), but the classic is a dense 'ivory' vertebra
- Pathognomic histopathological finding is the Reed–Sternberg cell (large with a 'mirror image' double nucleus. Looks like an owl!)
- Treatment is with radio- and chemotherapy

Leukaemia

- Haematopoietic disease widely affecting the bone marrow
- Bone pain is presents in 25% of children and 5% of adults with the disease but radiographic changes are present in up to 90%
- X-rays show
  1. Transverse lucent metaphyseal line in children
  2. Osteolytic destruction
  3. Generalized osteopaenia
  4. Periosteal elevation
  5. Focal sclerosis
- Treatment varies with subtype but includes chemotherapy, radiotherapy and, in some cases, bone marrow transplant

Treatment of immunohaematopoietic tumours

- Treatment of immunohaematopoietic tumours (e.g. myeloma, lymphoma and leukaemia) is usually with chemo- and radiotherapy, but rarely involves surgery
- Surgery is reserved for stabilisation of completed or impending pathological fractures

Vascular neoplasms

Angiosarcoma

- Usually present with a rapidly enlarging mass in the lower limb in males. Usually after radiotherapy/ lymphadenectomy (Stewart–Treves syndrome)
- X-rays show extensive bony destruction with erosion of the cortices and MRI shows extension into the soft tissues
- Aggressive tumours which metastasise early
- Treatment is with radical excision

Metastatic bone disease

- Best managed by an MDT in conjunction with the departmental lead for MBD[12]

Epidemiology

- 'Big 5': Lung, breast, prostate, kidney and thyroid
- >20 000 patients develop bony metastases per annum in the UK
- 11 500 deaths from breast cancer per year of which 70% have MBD

Preoperative assessment

- As for any suspicious lesion as described earlier

- **Do not assume a solitary lesion is a metastasis**
- Bone profile blood test important to look for hypercalcaemia
- Investigations to find primary cancer (see earlier section)
- Assess general fitness for surgery
    a. biological, as opposed to chronological, age
    b. functional ability or performance status
    c. medical co-morbidities or ASA grade
    d. patient motivation
    . Correct reversible conditions, e.g. anaemia
- Renal, thyroid and melanoma metastases can be extremely vascular. Preoperative angiography and embolisation can reduce blood loss during surgery

Biopsy

- Where diagnosis is in doubt or the metastasis is a solitary lesion
- Must follow principles as above and should be discussed with/performed by the bone tumour MDT
- Metachronous, solitary renal metastasis, after successful urological treatment of the primary, may be best managed by wide excision with curative intent

Prophylactic fixation

- Assess fracture risk using Mirels score[13]
    . **Site**: Upper limb (1 pt), lower limb (2 pts), inter/subtrochanteric (3 pts)
    . **Pain**: Mild (1 pt), moderate (2 pts), limits function (3 pts)
    . **Matrix**: Blastic (1 pt), mixed (2 pts), lytic (3 pts)
    . **Size**: (maximal cortical destruction) <1/3 (1 pt), 1/3–2/3 (2 pts), >2/3 (3 pts)
    . Scores are added together: ≥8 are at high fracture risk (>30%) and should undergo surgery before radiotherapy (Figures 20.2 and 20.28)
    . Other considerations include prognosis, mobility and likelihood that lesion may respond to non-surgical treatment
    . The Mirel score is a guide only and a clinical decision taking all factors into account is essential
- Radiotherapy is usually palliative, is given in a single fraction, but may prevent fracture if the lesion heals

Stabilisation of pathological fractures

Assume that fractures will **not** heal.

- Aim for
    . Immediate stability
    . Immediate weight-bearing
    . A durable reconstruction that will outlive the patient and will remain stable if there is local recurrence
    . Stabilise all lesions in any given bone if possible
    . If practical, local tumour debulking with PMMA cement packing, may increase stability and reduce complications of tumour progression postoperatively (Figure 20.28)
    . Adjuvant radiotherapy may reduce the risk of local recurrence
    . There is a high rate of failure with fixation of proximal femoral lesions; therefore, consider cemented hip replacement/tumour prosthesis
    . Pelvic and acetabular lesions should be treated by an experienced orthopaedic oncology surgeon

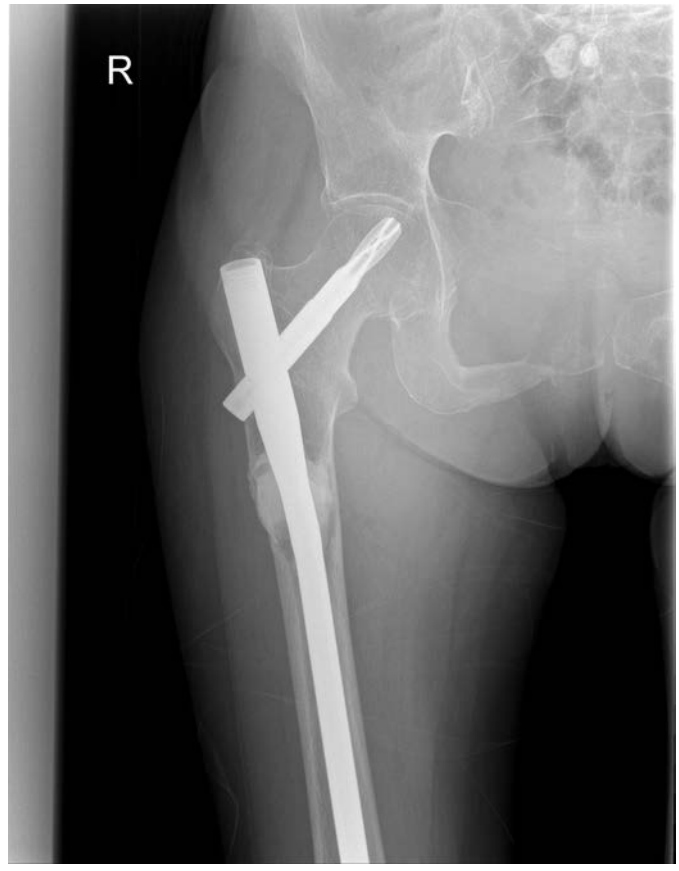

Figure 20.28 IM nail right femur for MBD as seen in Figure 20.2. The lesion in the femur would score 12/12 according to Mirel's criteria. It is in the subtrochanteric region of the femur (3 pts), the patient experiences pain at rest (3 pts), the matrix is lytic (3 pts) and more than two-thirds of the cortex is destroyed (3 pts). It has been treated with local curettage, cementation and nailing of the entire femur. The reamer, irrigator, aspirator (RIA, *Synthes*) system was used to minimize distal tumour seeding within the bone

Other treatments:

- Chemotherapy, bisphosphonates and hormonal manipulation may all have a role and, hence, a contemporaneous oncology input is mandatory with MDT discussion

Spinal metastases

- Commonest site for MBD
- Present with pain/neurological deficit
- Always look for signs or symptoms of cord compression

- Consider possibility of unstable spine
- MRI to assess extent of involvement – Whole spine
- Biopsy only on advice of spinal centre
- Radiotherapy as definitive treatment if
  - No instability
  - It is a radiosensitive tumour
  - Multi-level disease
  - Stable neurology
  - General condition precludes surgery
  - Poor prognosis
  - Or as adjuvant treatment postoperatively
- Surgery if
  - Unstable
  - Progressive neurological deficit
  - Intractable pain, unresponsive to non-operative management
  - Spinal cord tolerance reached after prior radiotherapy
- Objectives of surgery
  - Prevention of further neurological deficit. Recovery of neurological deficit can occur, but is uncommon
  - Restoration of spinal stability
  - Decompression of spinal cord and spinal nerves
  - Restoration of structural integrity and stability of the vertebral column
  - Tumour eradication if feasible
  - Surgery should ideally be undertaken before the patient loses the ability to walk and should be done to maximize useful function
  - The magnitude of the procedure should not exceed the patient's ability to survive it or the surgeon's level of competence
  - Decompression **AND** stabilisation is usually required

Soft-tissue tumours[7]

Benign synovial lesions

Synovial chondromatosis

- Multiple islands of metaplastic cartilage in the synovium of a major joint, which calcify (or even ossify) to a varying degree
- Patients complain of pain, swelling and decreased ROM or mechanical symptoms
- X-rays show numerous partially calcified loose bodies within the joint
- Treatment is by surgical excision and synovectomy, arthroscopically if possible, but recurrence is common
- Malignant transformation is very rare

Teno-synovial giant cell tumour

- Previously known as pigmented villonodular synovitis (PVNS) but renamed in the fourth edition of the WHO classification
- **NOT to be confused** with giant cell tumour of bone
- A locally aggressive synovial tumour affecting mainly large joints (diffuse type) and tendon sheaths (nodular type)
- Knee most commonly affected but is also seen in the hip, ankle, foot and wrist
- X-rays generally show only soft-tissue swelling, but juxta-articular erosions can sometimes be seen particularly in the hip. MRI may show small voids within the tumour caused by iron deposits, and 'blooming' on a gradient echo sequence
- Lesions are seen as tan coloured nodules with haemosiderin deposition (pigmented) and a dramatic hyperplastic villous response is seen in the adjacent synovium, particularly in large joints
- Treatment is by excision of the lesions ± total synovectomy. In diffuse disease, recurrence is $\geq$50%. Radiotherapy is sometimes used for recalcitrant disease and recent use of chromic phosphate (p32) has shown some encouraging results

Benign fibrous lesions

Fibroma of tendon sheath

- Small, well-circumscribed lesions affecting tendons in the hands and feet
- Treatment is by excision if symptomatic

Fibromatosis

- A generic term for a group of diseases characterized by a cellular, infiltrative growth of fibroblastic tissue. (Dupuytren's, Peyronie's, etc)
- Palmar fibromatosis/Dupuytren's disease is common (10% incidence), affects men 4 : 1 women, may be familial, and is associated with diabetes, epilepsy and alcoholic liver disease. Treatment is often by surgical excision. For more detailed summary of Dupuytren's disease, see the section in the hand oral topics
- Plantar fibromatosis is rare compared to Dupuytren's disease. It presents with large nodules but contracture is not a feature. Unlike Dupuytren's, surgery should be discouraged as recurrence is common and florid
- Fibromatosis can occur as a soft-tissue tumour (extra-abdominal desmoids tumour) in adolescents and young adults, typically around the shoulder or pelvic girdles. It is locally invasive and resection has a high rate of local recurrence. The natural history is unpredictable in that recurrent nodules can remain dormant for many years, or grow rapidly for some time and then plateau
- The pathologist will need clinical information when examining a surgical specimen of fibromatosis as differentiating it from low-grade fibrosarcoma can be difficult
- Radiotherapy may be helpful for recurrent disease
- Chemotherapy (methotrexate and vinorelbine) has shown good response in reducing tumour volume

Nodular fasciitis

- A proliferation of fibroblasts most commonly seen on the volar aspect of the forearm from the second to fifth decades
- It can be mistaken for a malignant condition because of the hypercellularity, atypia and mitotic rate seen within the rapidly growing lesion
- Despite the aggressive microscopic features, the condition is self-limiting and can be treated with local excision

Elastofibroma

- A firm, rubbery tumour, which occurs almost exclusively in the fascia between the ribs and the inferior portion of the scapula
- Patients present with pain as the lesion 'flicks' over the border of the scapula on shoulder elevation
- Microscopic examination reveals a collagenous, fatty lump, with numerous elastin fibres throughout
- Treatment is conservative or by surgical excision for symptomatic lesions

Peripheral nerve lesions

Traumatic neuroma

- An exuberant, non-neoplastic growth of nerve tissue, which occurs at the proximal end of a severed or injured nerve
- Treatment includes conservative measures (cortisone injection, physiotherapy) or surgery

Morton's neuroma

- Thickening and degeneration of an interdigital nerve in the foot
- Clinical diagnosis by pain on web-space pressure with metatarsal approximation, Mulder's click and digital nerve stretch
- Treatment is by steroid injection or surgical excision

Benign Schwannoma

- Encapsulated nerve sheath tumour with a highly ordered cellular component (Antoni A area) and a loose myxoid component (Antoni B area)
- Usually solitary, fusiform lesions on flexor surfaces of limbs
- Treatment by marginal excision to spare nerve fibres, which are usually splayed over the surface of the lesion. Even with intralesional excision (to spare nerve function), local control is usually good

Neurofibroma

- Most occur as solitary lesions in the third decade
- Non-encapsulated lesions although usually well circumscribed
- Treatment is by marginal excision

Neurofibromatosis

- Genetic disorders of nerve development and growth
- Nf-1 (von Recklinghausen's disease) and Nf-2

Nf-1

- 1 in 4000 incidence of which up to 50% are new mutations affecting gene on Ch 17.
- Diagnosed by two or more of the following
  - Five or more *cafe-au-lait* spots
  - Two or more neurofibromas or one plexiform neurofibroma
  - Tumour of the optic nerve (optic glioma)
  - Benign growth on the iris (Lisch nodule)
  - Scoliosis
  - Deformity of other bone (esp. tibial bowing)
  - Freckling of groin or armpit
  - Parent sibling or child with Nf-1
- Patients are kept under observation and treatment aims at symptomatic control. Surgical excision can be performed for large or unsightly lesions. Malignant transformation occurs in up to 5% and should be treated as per sarcomatous lesions

Nf-2

- 1 in 40 000 incidence
- Bilateral acoustic neurofibromas (CN VIII) which lead to tinnitus and progressive deafness, ± other gliomas, menigiomas, neurofibromas, schwannomas or early cataract formation
- Treatment is aimed at surgical debulking of the acoustic neuromas to relieve compression but avoid complete deafness

Miscellaneous lesions

Lipoma

- Tumours of mature adipocytes, identical to the surrounding adipose tissue, with minimal variation in cell size and shape
- Usually seen in the fifth decade as superficial lesions
- MRIs demonstrate circumscribed, bland lesions, identical in signal to the surrounding fat
- Treatment is by marginal excision
- Lesions showing variation of adipocyte size, nuclear atypia, fibrous septal stroma and lipoblasts are now termed atypical lipomatous tumours when they occur in the limbs or well-differentiated liposarcomas when they occur in the visceral cavities of the trunk. Unlike simple lipomas they carry a risk of local recurrence but have almost no malignant potential unless dedifferentiation occurs (Figure 20.29)

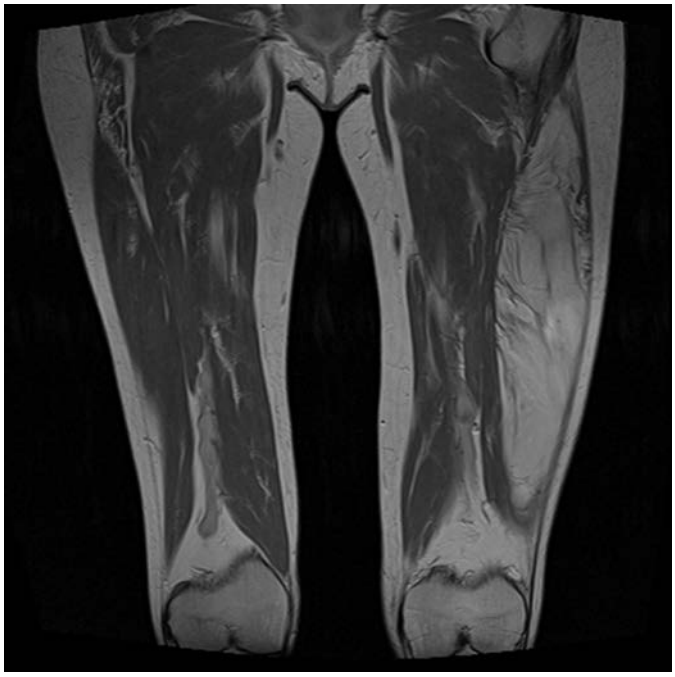

Figure 20.29 T1 MRI (fat is bright) showing a large, deep, intramuscular lipoma of the left thigh. It is made of largely bland fatty tissue. Although there is some streaking, there are no dark nodules to suggest an area of de-differentiation. (Contrast with Figure 20.30)

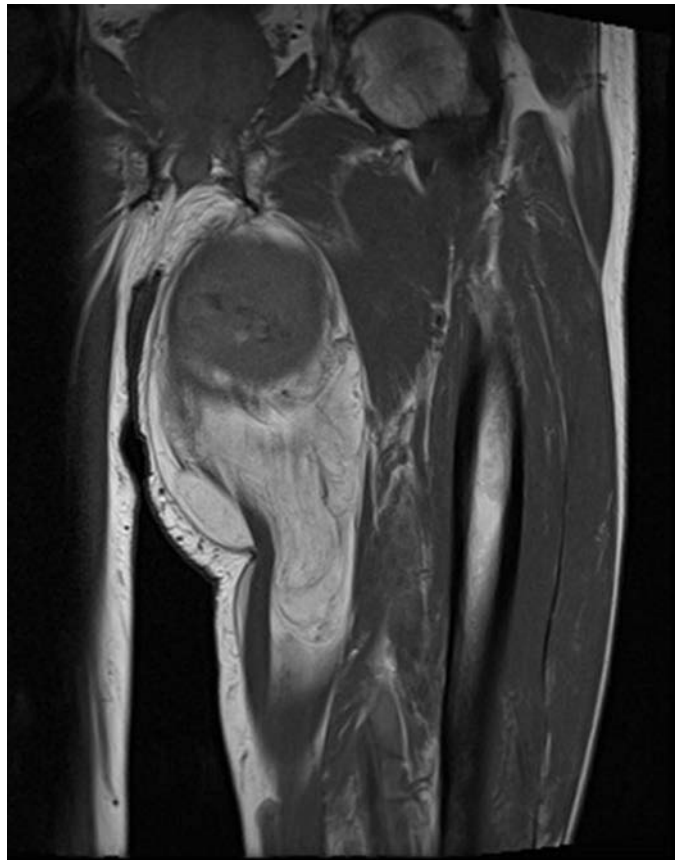

Figure 20.30 T1 MRI of a left thigh showing a large fatty lesion. However, within this lesion is a large dark nodule, which is bright on T2 (see Figure 20.31). This represents an area of dedifferentiation into a liposarcoma

Haemangioma

- Commonest soft-tissue tumour of infancy
- Multiple (usually small) lesions in Maffucci's syndrome
- Capillary and cavernous forms, most usually present on head or neck, consisting of a mass of 'knotted' blood vessels
- Diagnosed by ultrasound ± MRI
- Plain x-ray may show calcification within vessels,
- Treatment of peripheral lesions is usually by observation whilst the child grows, but can include laser treatment (for flat superficial lesions), embolisation or surgical resection

Myositis ossificans circumscripta

- Solitary, non-progessive, benign ossification of the soft tissues
- Usually presents as a painful lump within a muscle often following trauma
- X-ray shows calcification within the lesion after maturation but may be negative in early presentation
- Treatment is by surgical excision

Myositis ossificans progressiva

- Very rare genetic (autosomal dominant), progressive disease affecting groups of muscles, tendons and ligaments usually in the spine and upper limb, leading to progressive fibrosis, calcification and ossification, resulting in deformity. Biopsy leads to muscle trauma and should be avoided. Associated with infantile hallux valgus
- Often fatal secondary to pulmonary impairment
- No effective treatment is available but bisphosphonates and steroids can be beneficial during flares

Ganglion

- Fibrous-walled cysts containing mucinous fluid, commonly seen on the extensor surfaces of hands, wrist and feet, originating from the underlying joint
- Treatment (if large or symptomatic) is by aspiration and needling of the cyst, injection of a sclerosant (recurrence up to 70%) or surgical excision (recurrence up to 40%)

Soft-tissue sarcomas (STS)[14]

STS are a heterogenous group of tumours of mesenchymal or connective tissue origin. They are rare, with an incidence of around 45 per million per year, and comprise <1% of all adult cancers. The median age at presentation is around 55 years. Histological types include:

Undifferentiated pleomorphic sarcoma

- The term Malignant Fibrous Histiocytoma has been removed from the most recent WHO classification
- Pleomorphic, high-grade tumour presenting in the fifth to seventh decades more commonly in men

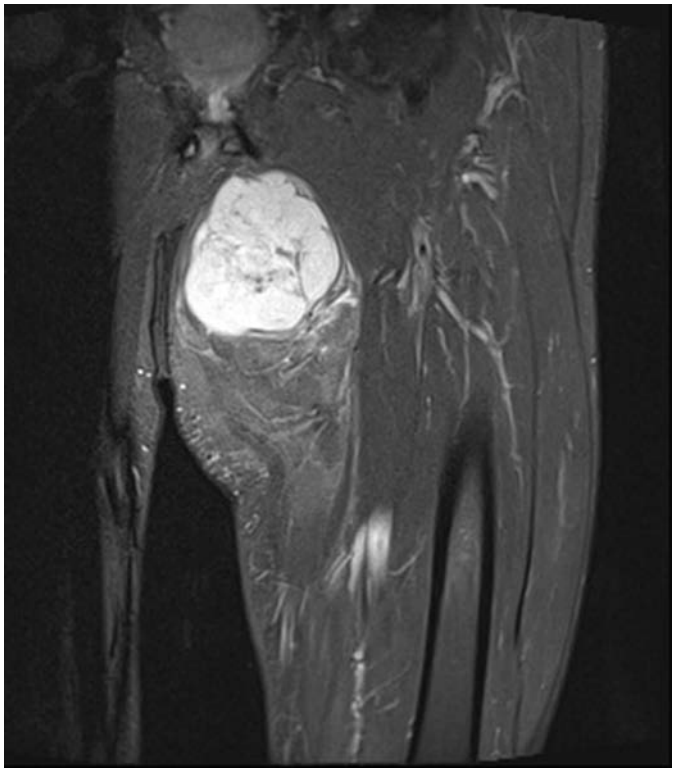

Figure 20.31 T2 MRI of the same lesion as Figure 30. Note that the fatty areas are dark and supress, while the area of dedifferentiation is bright

- A diagnosis of exclusion after other tumour types have been excluded by immunohistochemical staining and genetic testing
- Treatment is by wide surgical excision. Use of chemotherapy is controversial

Liposarcoma

- Malignant tumours of fat seen predominantly deep to the fascia of the lower limbs and in the retroperitoneal space
- Three major subtypes
  1. Atypical lipomatous tumours with dedifferentiation (40%). They are characteristically seen in fifth to seventh decades. Local recurrence is more common in retroperitoneal tumours and the risk increases if there are areas of dedifferentiation. Metastasis is generally only seen in tumours with dedifferentiation (Figures 20.30 and 20.31)
  2. Myxoid and round cell liposarcomas (50%). Generally present in the third and fourth decades. Cytogenetically, reciprocal translocation is seen between chromosomes 12 and 16. Metastasis occurs in 25% of myxoid and 50% of round cell tumours
  3. Pleomorphic liposarcomas (10% liposarcomas). Present in the fifth to seventh decades and have a poor prognosis with early metastasis

Rhabdomyosarcoma

- Malignant tumour of skeletal muscle. Histologically small round blue cell tumours
- Most common malignant soft-tissue tumour in children (87% <15 years), rare after the age of 40
- Three major subtypes
  1. Embryonal (55%): Generally seen from birth to 15 years, usually in the head, neck and truck. Cytogenetically show a loss of heterozygosity at chromosome 11p15.5
  2. Alveolar (20%): Generally seen between ages 10 and 25 years, usually in the extremities. Reciprocal translocation is seen between chromosomes 2 and 13 (t(2;13)(q35:14))
  3. Undifferentiated (20%): Generally seen in older patients (fourth decade and above). Poor prognosis
- Treatment includes neo-adjuvant chemotherapy and wide excision. Radiotherapy is given for close margins or otherwise unresectable tumours

Synovial sarcoma

- Classically spindle-cell tumours with an epithelial component seen between the ages of 15 and 40 years in the soft-tissues of the lower limb, usually adjacent to the knee
- **NOT** of synovial origin
- Can present with a long history of pain
- Reciprocal translocation t(x:18) is present in 90%
- Irregular calcification is seen in 20%
- Chemotherapy can cytoreduce some tumours improving resectability but does not affect overall survival

Fibrosarcoma

- Rare malignant tumour of fibroblasts generally seen in the fourth to sixth decades
- Well- and poorly differentiated types
- Well-differentiated tumours display the classic 'herring-bone' cell pattern histologically and have a 60% 5-year survival
- Poorly differentiated tumours have a much poorer prognosis

Malignant peripheral nerve sheath tumour

- Malignant spindle-cell tumour of nerve or neurofibromatous origin, presenting in the third and fourth decades
- Ten per cent of all soft-tissue sarcomas
- Fifty per cent arise in patients with neurofibromatosis type 1
- Can be difficult to differentiate histologically from leiomyosarcoma or fibrosarcoma
- Local recurrence and metastasis are common (both around 50%)

Epithelioid sarcoma

- Malignant soft-tissue tumour usually seen in the superficial subcutis or tendon sheaths of the hand and wrist in the second and third decades, most commonly in men
- Can be mistaken for squamous cell carcinoma or synovial sarcoma
- Probably of synovial origin
- Aggressive tumours which metastasise early either haematogenously or through the lymphatics
- Poor prognosis

Treatment of soft-tissue tumours[14]

Reconstructive/limb-salvage surgery

- Safe margins must not be compromised for a preferred functional or reconstructive outcome
- Randomised controlled trial showed increased rates of local recurrence in limb-salvage surgery compared to amputation but no significant difference in 5-year disease-free survival or overall survival. Hence, limb-salvage surgery has become the norm[15]
- Amputation is reserved for tumours that bridge several compartments, or extensively involve neurovascular structures, or those for which an amputation would be more functional than the salvaged limb (e.g. following complex resection of the foot)

Adjuvant treatment

Radiotherapy[16]

- Radiotherapy reduces the rate of local recurrence but has no effect on survival
- Preoperative or postoperative radiotherapy?
  - Preop radiotherapy can be given at a lower dose (50 Gy vs 65 Gy) and with a smaller treatment volume, but is associated with a higher rate of wound complications, particularly in the proximal thigh[17]
  - Postop radiotherapy has higher rate of long-term fibrosis and lymphoedema because of the higher doses and field sizes used potentially leading to a worse functional outcome[18]
- In UK, usually given postop to reduce rate of local recurrence except for radiosensitive tumours (e.g. myxoid liposarcoma) when preop treatment may shrink the tumour

Chemotherapy

- **Sarcoma Meta-Analysis Collaboration (SMAC)**: Cochrane review, 2000. Included data from 14 randomised trials and showed a small but significant reduction in both local and distant recurrence-free survival but only a trend towards increased survival for patients treated with chemotherapy in soft-tissue sarcoma[19]
- Pervaiz et al. *Cancer*, 2008. Updated SMAC using later studies which used higher doses of doxorubicin and ifosfamide and showed a benefit in local, distant and overall recurrence-free survival as well as an increase in overall survival equivalent to an absolute risk reduction in death of 11%[20]
- BUT: Side effects are significant (e.g. cardiotoxicity with doxorubicin) so use is limited, particularly in the elderly population at risk of soft-tissue sarcoma
- Some specific tumour types are chemo-sensitive and, hence, chemotherapy does form part of the treatment regime for these, e.g. rhabdomyosarcoma, soft-tissue Ewing's and primitive neurectodermal tumours (PNETs)

Examination corner

Basic science oral 1

Candidate shown a picture of a diseased and necrotic hand.

CANDIDATE: Due to the manginess of the hand I initially failed to spot that there was a digit missing. I started by discussing infection and osteomyelitis before realizing that it was a malignancy. I went for epithelioid sarcoma and ended up in a difficult discussion until the bell went.

EXAMINER: The candidate offered epithelioid sarcoma as the diagnosis. Although this should form part of the differential as the most common soft-tissue sarcoma of the hand, squamous cell carcinoma is much more common and was in fact the diagnosis.

Basic science oral 2

Candidate shown an x-ray of a pathological neck of femur fracture.

CANDIDATE: I was quickly moved on past history and examination to discuss investigations and management. I discussed the likelihood of primary verses metastatic and explained that there was no urgency to fix at the expense of diagnosis. This was the correct decision as the diagnosis turned out to be a renal cell metastasis. We then discussed further management including embolisation.

Basic science oral 3

'Describe this tumour.'

Common prop-based question usually using a plain radiograph and occasionally another imaging modality, e.g. MRI.

Basic science oral 4

Candidate shown a radiograph of an osteosarcoma of the femur.

Asked to describe the x-ray appearance.

Asked about principles of tumour staging and biopsy.

Asked about the surgical options available in the treatment of bone tumours.

EXAMINER: You are the tumour surgeon. How are you going to stage and manage this tumour?

Basic science oral 5

Candidate shown a radiograph of a lucent lesion of the proximal femur.

Asked to describe management, investigation and likely diagnosis.

Discussed scoring system of Mirel.

Basic science oral 6

Candidate shown a radiograph of a lytic lesion of the midshaft of the humerus in an elderly woman.

Asked to describe the x-ray and offer a differential diagnosis.

The diagnosis was myeloma.

EXAMINER: Where else do you get myeloma deposits? How do you confirm the diagnosis?

Basic science oral 7

Candidate shown a radiograph of a pathological fracture through a fibrous cortical defect in the distal tibia.

Asked to present a differential diagnosis.

EXAMINER: How do you manage the fracture? How do you confirm the diagnosis?

Adult and pathology oral 1

Candidates may be asked to describe how they would perform a biopsy of a suspicious bone lesion.

Answers should include the following principles:

1. A biopsy should only be performed after detailed history, examination, investigation and planning
2. A biopsy should only be performed by, or after discussion with, a surgeon working as part of a bone tumour MDT
3. Careful planning of the biopsy track is essential such that all contaminated tissue and the track itself can be excised *en bloc* during the definitive resection
4. Biopsies can be taken using a core needle percutaneously or by open incision
5. Biopsies can be taken with or without image guidance including CT, US or II
6. Where open biopsies are performed, the incision should be placed along the longitudinal axis of the limb
7. Close attention to haemostasis and minimal tissue dissection are important during incisional biopsy to minimize tissue seeding

Adult and pathology oral 2

Candidates are commonly asked to give an account of the typical workup for patients with a bony lesion?

In answering these types of question, start by saying that one would:

1. Take a history and perform a detailed examination
2. Take plain radiographs of the affected part

Primary bone lesion/tumour

1. Urgent discussion with a bone tumour MDT. Urgent referral must not be delayed by local arrangement of further investigations
2. Local imaging of the bony lesion
   (a) X-rays have usually been performed prior to referral, and often have alerted the referring unit to the possibility of a primary bone lesion, triggering the referral. X-rays should include the whole bone and include the joint above and below to screen for skip/satellite lesions
   (b) MRI gives good information about soft tissues. It is used to delineate the extent of the tumour, to seek out extracompartmental extension and/or skip lesions, and assess any involvement of critical neurovascular structures. Images can give diagnostic information in certain circumstances, but are generally used for biopsy planning and later to plan the definitive resection
   (c) CT scans can give more detailed information about the bony involvement and anatomy. Can be useful for diagnosis, e.g. osteoid osteoma
3. Biopsy
   (a) Biopsy is performed to obtain a definitive histological diagnosis. (For principles of biopsy see earlier question)
4. Staging
   (a) The lung is the most common site for metastasis and, hence, CT chest is the most important staging investigation, but often the abdomen and pelvis are included. Whole body bone scan is also helpful to look for other bone lesions
   (b) Certain tumours (as diagnosed from the biopsy) require specific staging procedures, e.g. bone marrow biopsy in Ewing's sarcoma
5. Treatment planning in the MDT

Metastatic lesions If the history, examination and radiographs point towards this being a metastatic lesion, further investigations can be undertaken locally. Malignant lesions in bone are more likely to be metastases in patients over 40 years of age. If there is any dubiety urgent discussion should be undertaken with a bone tumour MDT. Where there is a fracture, the candidate should not rush in to recommending surgical fixation when the diagnosis is not known.

Metastatic lesions should be investigated with a global aim of finding the primary cancer (if not already obvious) and a local aim of delineating the metastatic lesion and determining if orthopaedic intervention is required:

1. Laboratory studies
   (a) FBC, U&E, LFTs, bone profile

(b) Serum electrophoresis (myeloma screen)
(c) Tumour markers as appropriate to history, e.g. PSA

2. Imaging to 'hunt' the primary
   (a) Chest x-ray (looking for lung primary or lung mets)
   (b) Bone scan (looking for other metastases)
   (c) CT chest and abdomen (looking for primary tumour ± other mets, e.g. liver). CT pelvis may be included at this stage if surgical treatment of a lesion around the hip is being considered
   (d) Other imaging may be indicated by the original history and examination, e.g. mammogram, thyroid USS, etc.

   If all the investigations aimed at finding the primary cancer are negative a biopsy of the bone lesion will be required to obtain a definitive diagnosis. This must then be treated as a possible primary bone lesion and discussed with a bone tumour MDT.
3. Local imaging of the metastatic lesion
   a. Plain radiographs of the whole bone including the joint above and below to screen for other lesions in the same bone
   b. A CT scan shows calcified tissue better and can delineate the amount of bone erosion
   c. An MRI scan shows soft-tissue detail better and can delineate the size and extent of the metastatic lesion, in particular whether there is a soft-tissue mass

   Discussions on fixation of metastatic lesions should include:

1. The Mirel score
2. The general condition of the patient and conservative measures, including reference to team working with other specialists
3. Principles of fixation
   (a) Assume that any pathological fracture will not heal
   (b) Fix all lesions in a bone if possible
   (c) Aim for immediate stability, immediate weight-bearing and a durable reconstruction that will outlive the patient
   (d) In general, arthroplasty is preferred to fixation around joints

   Again, if there is any dubiety then discussion with a bone tumour MDT is mandatory. Remember too, that solitary metastases may, in certain circumstances be best treated as primary tumours and should be discussed with a bone tumour MDT.

Adult and pathology oral 3

Osteochondroma proximal femur
Differential diagnosis/management?

The radiological features should be diagnostic. X-rays should show cortical continuity between the bone and the prominent lesion. Lesions can be sessile (broad-based) or pedunculated (stalked). MRI scan should confirm the contiguous nature of the cortex with the lesion but will also delineate the cartilage cap. Bland cartilage <1 cm in depth indicates a benign lesion and excision biopsy is appropriate treatment if the lesion is symptomatic.

Red-flags include lesions which continue to enlarge after the cessation of skeletal growth and the closure of the physes, and a thickened cartilage cap. Malignant transformation is very rare in isolated lesions but is reported at between 1% and 5% in multiple hereditary exostoses. Biopsy of suspicious lesions, by or after discussion with a bone tumour MDT, must preceed surgical excision, as wide margins, including excision of the biopsy track, will be necessary if malignant change is diagnosed.

Staging?

Osteochondromas are benign lesions. If asymptomatic, they are labelled as latent, but, if they become symptomatic, they are then labelled as active. This is according to the system described by Enneking, classifying benign lesions as latent, active, or aggressive. (If asked to give an example of a lesion that would be labelled as aggressive, the classic is a GCT.)

Adult and pathology oral 4

X-ray osteoid osteoma in the femoral neck
How to investigate?

- **X-ray will show thickening of the cortex. A lucent nidus may be identified, but may be obscured by the by the cortical thickening.**
- **CT scan shows the classic lucent nidus (<1 cm) with a central point of sclerosis and is diagnostic.**
- **Bone scan is usually unnecessary, but will show increased uptake.**

To fix or not to fix?

Treatment is either conservative or operative. Conservative treatment is with NSAID medication which classically absolves the pain associated with this lesion. Operative treatment of oseoid osteoma is with CT-guided radioablation, 'burr-down', or surgical excision. Depending on the position of the lesion within the femoral neck, mechanical stabilisation with internal fixation may be required if an open procedure is performed.

Basic science oral 1

Candidates may be asked to differentiate between infection and neoplasm on radiographs.

- It may not be possible to distinguish between infection and tumour on the basis of radiograph

- Both may show diffuse changes, patches of lucency and sclerosis, and periosteal reaction
- The clinical picture may not be helpful either as fever, raised ESR, and swelling with localized dolor, rubor and calor may also be features of Ewing's sarcoma
- Definitive diagnosis often requires biopsy for histology and culture to distinguish between infection and tumour

Basic science oral 2

Candidates may be asked to describe the radiological differences between benign and malignant bone lesions. The differences are really between locally aggressive lesions and others.

Key features

- Zone of transition
  - Wide/diffuse in malignant lesions
  - Narrow in benign lesions
- Periosteal reaction seen in malignant lesions

Candidates may be shown various x-rays and asked to describe the salient feature and offer a differential diagnosis, e.g.

- Ewings sarcoma
  - Bony lucency with a wide zone of transition – Classically spreads through cortex without destroying it
  - Periosteal reaction
    - 'Onion peel'
  - Differential diagnosis
    - Osteomyelitis
- Osteosarcoma
  - Sclerotic, bone-forming lesion typically in the metaphysis of a long bone around the knee or shoulder with a wide zone of transition, invading the cortex and extending into the soft-tissues
  - Periosteal reaction
    - Codman's triangle (periosteal elevation)
    - 'Sunray' spiculation (indicative of extension into the soft-tissues)
- Paget's disease
  - In the earliest stages of the disease, osteoclastic resorption predominates and significant radiolucency may be seen
  - As the disease progresses overall density increases
    - Trabeculae become coarser and thicker
    - Cortical bone becomes less compact
    - Corticomedullary demarcation becomes less obvious
    - Diameter of the bone increases
  - Vetebral bodies may show uniformly increased density mimicking possible metastatic tumour
  - Pelvic lesions may show areas of sclerosis or lysis as well as areas of 'honeycomb' or striation
  - In long-bones, disease usually starts at one end and extends along the bone. The junction between diseased and normal bone is often seen as a 'flame-like' wedge of advancing rarefaction
  - Bone of the skull show areas of patchy sclerosis in the latter stages of the disease and the bone may appear significantly thicker than normal

Bibliography/further reading

Bullough's *Orthopaedic Pathology* provides an extensive overview of bony and soft-tissue lesions and includes numerous x-rays and histology slides. The clarity and order of his book has given much inspiration for the layout of this chapter[7].

Papp et al.'s immersion orthopaedic pathology article in *J Bone Joint Surg Am* is also very useful as further reading for the exam[21].

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Chapter 21

Hand oral core topics

David R. Dickson and John W. K. Harrison

Hand surgery syllabus for the FRCS (Tr & Orth) examination

'The hand' covers the hand and forearm and the structures anatomically contained within. Knowledge of the structural anatomy and the biomechanics of joint and tendon function is required.

Pathology

A working knowledge of the acute conditions and trauma of the hand is required, i.e. injury to the bones, joints, tendons, nerves, skin and vessels of the hand, and infective processes.

Knowledge of the non-acute congenital, degenerative, inflammatory (rheumatoid) and neoplastic conditions as well as benign tumours, e.g. ganglions, is also required.

Training in operative hand surgery

For the purpose of the examination, the trainee should have gained experience in the operative management of:

- The acutely injured hand
- Fractures and dislocations, including scaphoid non-union
- Nerve injuries
- Tendon injuries and common tendon transfers
- Skin grafts
- Infections

In elective surgery, the candidate must have sound knowledge of the procedures appropriate for carpal tunnel syndrome, trigger finger, Dupuytren's contracture, benign tumours, degenerative conditions of the thumb carpometacarpal joint and wrist joints, and surgery of the rheumatoid hand.

Intermediate cases

- Brachial plexus injuries
- Peripheral nerve injuries
- Rheumatoid shoulder/hand and wrist

Short cases

Rheumatoid hand and wrist

- Dupuytren's disease
- Ulnar nerve injury (high vs low lesion)
- Basal thumb osteoarthritis
- Hand swellings
- Carpal tunnel syndrome
- Kienböck's disease
- Median nerve injury
- Perilunate dislocation
- Radial nerve palsy
- Ulnar collateral ligament injuries

Basic science

Brachial plexus

- Nerve conduction studies
- Extensor compartments of the wrist
- Flexor tendon sheath/vinculae/radial and ulnar bursae
- Name muscles in deep flexor compartment of forearm
- Ulnar nerve anatomy at wrist
- Seddon's classification of nerve injury
- Factors influencing outcome in nerve repair

Children

- Syndactyly
- Camptodactyly
- Clinodactyly
- Congenital bands
- Delta phalanx
- Radial club hand
- Congenital absence of thumb

Trauma

- Distal radius fractures
- Scaphoid fracture/non-union/perilunate injuries
- Bennett's fractures (name deforming forces)
- Carpal instability
- Compartment syndrome
- Digital nerve injury
- Fingertip injuries
- Finger amputations
- Flexor tendon injuries: Repair, rehabilitation and late reconstruction

- 'Mangled hand'
- Pathoanatomy of MCP joint dislocation
- UCL injuries – Stener lesion

Rheumatoid

- Dropped fingers
- Boutonnière/swan-neck
- Elbow replacement
- MCP joint replacements
- Rheumatoid thumb

Others

- Dupuytren's disease: Associations, management of PIP joint contracture, treatment options
- Drainage infection
- Kienböck's
- Complex regional pain syndrome
- Enchondromata
- Tumours
- Tourniquets

MCQ paper

- Ganglions
- Trigger finger
- Management principles for the rheumatoid hand
- Causes of loss of extension in the rheumatoid finger

General guidance

Generally the oral is very straightforward and consists of a series of clinical photographs and radiographs of common hand conditions that the average trainee would have no difficulty in recognising. There is a large amount of material but, again, a working knowledge of all subjects is sufficient. It is a case of describing what you see, the diagnosis, differential diagnosis and management options. Do not expect any encouragement or any clues as to how you are doing throughout the oral.

It is a combined oral with paediatrics, with both orals last 15 minutes each. There are usually one or two paediatric hand trauma questions thrown in at some stage from either the hands or paeds examiner.

Most trainees will not have done a hand job even for 6 months. A practical, safe knowledge of hand surgery is required rather than textbook minutiae. Practising orals before the exam with an experienced hand surgeon will help greatly.

Anatomy

Bones

There are 19 in the hand (plus 2 sesamoids), and 8 in the wrist.

Physes

In the hand these are located distally in the second to fifth metacarpals, and proximally in the thumb metacarpal and all phalanges.

Flexor pulleys

Fingers

Facilitate sheath collapse and expansion during digital motion. There are five annular pulleys: A2 and A4 pulleys originate from bone and it is critical to preserve them to prevent bowstringing of the flexor tendons; A1, A3 and A5 originate from the volar plates. A1 pulley is released in trigger finger.

The three cruciate pulleys are not critical for flexor function. C1 is distal to the A2 pulley over the proximal phalanx; C2 and C3 are either side of the A4 pulley over the middle phalanx.

Thumb

In the thumb there are two annular pulleys and one oblique pulley. The oblique pulley is most important. A1 overlies the MCP joint, attached to the volar plate. A2 overlies the IP joint, attached to the head of PP. The oblique pulley overlies the shaft of PP. The tendon of adductor pollicis attaches to the A1 and oblique pulleys.

Flexor sheaths

The ulnar bursa can connect to the radial bursa through the space of Parona – The space between the flexor tendons and the pronator quadratus muscle – Causing a 'horseshoe' abscess.

Vinculae

The vinculae are folds of mesotendon carrying the blood supply to both tendons from transverse branches of the digital arteries. There is a short vinculum (vinculum brevis) and a long vinculum (vinculum longus) to each FDP and FDS tendon. Nutrition of the tendons is also derived from diffusion through the synovial fluid.

Anatomy of the intrinsic muscles

The intrinsic muscles have their origins and insertions within the hand. These are the thenar muscle group, hypothenar muscle group, adductor pollicis, lumbricals and interossei.

Lumbrical muscles (four)

The lumbrical muscles are the workhorse of the hand. They are the only muscles to originate on a tendon (FDP) and insert on a tendon (radial lateral band of the extensor expansion). The radial two lumbricals are innervated by the median nerve and the ulnar two lumbricals by the ulnar nerve. Action is MCP joint flexion and IP joint extension. Laceration of FDP distal to the lumbrical origin leads to a 'lumbrical plus' finger.

Interosseous muscles (seven)

Four dorsal interossei abduct the index, middle and ring fingers away from the axis of the middle finger (Dorsal ABducts, or DAB). Three palmar interossei adduct the index, ring and little fingers towards the axis of the middle finger (Palmar ADducts or PAD). The interossei flex the MCP joint and extend the PIP joint through the lateral bands. They arise from the metacarpal bones. The dorsal interossei insert into the lateral sides of the index and middle fingers and to the medial sides of the middle and ring fingers. The palmar interossei insert into the medial side of the index finger and to the lateral sides of the ring and little fingers. All are supplied by the ulnar nerve. An occasional variant is for the first dorsal interosseous muscle to be supplied by the median nerve.

Anatomy of the extrinsic muscles

The extrinsic extensor muscle bellies of the hand overlie the dorsum of the forearm and their tendons pass over the dorsum of the wrist to insert in the hand.

Dorsal extensor compartments of the wrist (six)

There are fibro-osseous tunnels through which the extensor tendons pass and are numbered from radial to ulnar.

1. **APL/EPB:** Located on the radial surface of the radial styloid. Chronic tenosynovitis here is known as de Quervain's disease. Both tendons are in separate synovial sheaths
2. **ECRL/ECRB:** ECRL inserts on to the base of the second metacarpal, and ECRB onto the base of the third. Intersection syndrome
3. **EPL:** Lister's tubercle separates the second from the third compartment. Delayed rupture following distal radius fractures
4. **EIP/EDC:** Extensor tenosynovitis and ruptures. Contains the posterior interosseous nerve
5. **EDM**: Double tendon. Floor is dorsal capsule of DRUJ. Vaughan–Jackson syndrome – Rupture in rheumatoid arthritis
6. **ECU:** Lies over the head of the ulna. Tendonitis or instability of ECU

Extensor tendons and hood

The extensor tendon broadens before dividing into three slips over the dorsal surface of the proximal phalanx. The central slip inserts into the base of the middle phalanx. The two lateral bands receive attachments from the lumbrical and interossei tendons to form a broad extensor expansion or hood, which overlies the metacarpal head and the proximal part of the PP. Over the middle phalanx the lateral slips are held dorsally by the **triangular ligament** and volarly by the **transverse retinacular ligaments.** Imbalance of the lateral bands results in a swan-neck or boutonnière deformity. The transverse retinacular ligaments attach to the volar plate.

Oblique retinacular ligament (ORL)

Passive extension of the DIP joint (tenodesis effect) – Bilateral strong narrow bands that originate from the periosteum of the PP and A2 pulley and insert into the extensor tendon at the base of the DP. Active extension of the PIP joint tightens the volarly placed ORL, leading to DIP joint extension.

Sagittal band

Connects the extensor tendon to the volar plate of the MCP joint to extend the MCP joint. In hyperextension of the MCP joint the IP joints fall into flexion because the extensor tendon distal to the sagittal band becomes lax. In this position the IP joints can only be extended by the intrinsics.

Boundaries of the anatomical snuffbox

The 'snuffbox' is a hollow area distal to the radial styloid on the dorsal–radial aspect of the wrist.

Floor: Scaphoid
Radial border: Extensor pollicis brevis (and abductor pollicis longus)
Ulnar border: Extensor pollicis longus
Proximally: Radial styloid
Distally: Base of thumb metacarpal

The radial artery courses through the snuffbox on its way to the dorsal first web space. The cephalic vein originates over the ASB.

Cleland's ligament (dorsal – Ceiling)

A fibrous ligament between the phalanges and the dermis (skin) that is dorsal (posterior) to the neurovascular bundle. These fibrous bands are located between the middle of the PP and the DIP joint.

Grayson's ligament (volar – Ground)

A very fine membrane, which lies in front of the neurovascular bundle. Originates from the anterior layer of the fibrous flexor tendon sheath and inserts into the skin.

FDS of the small finger

Absent in 30% so remember that it may be absent in flexor tendon injury of the little finger. Comparison with the opposite side is essential when evaluating for an FDS laceration in the small finger.

EDC of the small finger

EDC may be absent in half of people in whom extension is achieved by the EDM.

Nail anatomy

The nail plate is supported by the nail bed. This consists of the germinal matrix (lunula is the visible portion) that produces the nail, and the sterile matrix that produces keratin to thicken the nail. The eponychium is the proximal nail fold and the paronychium is the lateral nail fold.

Blood supply of the hand

The hand has a generous blood supply. Eighty per cent is from the ulnar artery.

Superficial palmar arch

Lies 2 cm distal to the deep arch. Surface anatomy is the distal palmar crease. The superficial arch is the curved continuation of the ulnar artery and is incomplete in 80% as there is no anastomosis with the superficial palmar branch of the radial artery.

From its convexity a palmar digital artery passes to the ulnar side of the little finger and three common palmar digital arteries run distally to the web spaces between the fingers, where each vessel divides into proper palmar digital arteries that supply adjacent fingers. The arteries lie superficial to the nerves in the palm and deep to the nerves in the digits.

Deep palmar arch

Surface anatomy is Kaplan's line (from the hook of hamate to the base of the first web space). The deep palmar arch is an arterial arcade formed by the terminal branch (deep branch) of the radial artery anastomosing with the deep branch of the ulnar artery and is complete in 98%. Lies deep to the long flexor tendons and superficial to the interosseous muscles.

From its concavity three palmar metacarpal arteries pass distally and join with the common palmar digital branches of the superficial arch.

Radial artery

The radial artery passes into the hand between the two heads of the first dorsal interosseous muscle. Lying between the first dorsal interosseous and adductor pollicis muscle, it gives off two branches.

- The *radialis indicis artery* passes distally between the first dorsal interosseous and adductor pollicis muscles to supply the radial side of the index finger
- The *princeps pollicis artery* passes distally along the metacarpal bone of the thumb and divides into the two palmar digital branches of the thumb at the metacarpal head

The main trunk of the radial artery passes into the palm between the oblique and transverse heads of adductor pollicis to form the deep palmar arch.

Examination corner

Hand oral 1

- Intraoperative photograph of the volar aspect of the wrist – Asked to identify various anatomical structures
  - State the relevant part of the wrist shown
  - Be systematic. If the carpal tunnel contents are shown state the structures you see and state that these make up the contents of the carpal tunnel, which may lead into the boundaries of the tunnel

Hand oral 2

- Similar to oral 1 with an interactive photograph of the back of the wrist – Asked to identify labels to various anatomical structures
  - State the relevant aspect of the wrist shown
  - Be systematic – E.g. if shown all the extensors state there are six compartments and then start to name them from one side to the other
  - Can conclude that this anatomy is important for a dorsal approach to the wrist, which can lead the examiner to the next question such as approach for wrist arthrodesis

Hand oral 3

- Shown a radiograph of a distal radius fracture that you've been asked to plate on the volar surface
- Describe the technique, landmarks and structures at risk
  - FCR approach
  - Tendon is usually palpable and incision placed over it. If not palpable the line is between the scaphoid tubercle and distal biceps tendon
  - Care should be taken with the radial artery on the radial side and the median nerve ulnarwards
  - FPL retracted
  - Pronator quadratus elevated
  - Brachioradialis can be released as it is a deforming force
- What is the innervation of pronator quadratus?
  - Anterior interosseous nerve
- Do you repair this muscle at the end?
  - Benefit is to cover the plate and, thus, protect the flexor tendons
  - Often the suture pulls through but worth a try. Just beware the radial artery when trying to repair the muscle
- What is the watershed line and what is the relevance of it?
  - This is the ridge just distal to the pronator quadratus to which the volar wrist capsule attaches
  - It is thought that using this as the distal limit to plate application will reduce the risk of flexor tendon attrition form the plate
- What is the shape of the dorsal surface of the radius and what is the relevance when placing screws and checking screw placement on a lateral radiograph?
  - The dorsal surface of the radius is not flat, it is peaked with Lister's tubercle being the summit. This three-dimensional structure is not appreciated on a lateral radiograph and dorsal penetration can occur but, unless the screw is longer than the peak of the distal radius, it can 'appear' OK
  - The risk can be reduced by measuring for screw length without penetrating the dorsal surface, taking oblique views and also an axial view

- If the patient also had carpal tunnel syndrome – How would you decompress this – Separate incision or just extend the approach to the distal radius? What structure do you need to take care of?
  - Safer to use two incisions – FCR approach for the distal radius and standard approach for the carpal tunnel
  - One incision risks damage to the palmar cutaneous branch which can result in painful neuroma

Anatomy of the median nerve

Nerve roots

- Formed by the joining of the lateral and medial cords of the brachial plexus in the axilla (C6, C7, C8, T1)
- The median nerve receives its sensory contribution predominantly from the lateral cord and the motor contribution predominantly from the medial cord

Course

- Lies lateral to the brachial artery in the arm, then crosses anteriorly to lie medial to the artery in the antecubital fossa, passing beneath the bicipital aponeurosis
- There are no branches before the elbow
- It descends between the two heads of pronator teres and is separated from the ulnar artery by the deep head of pronator teres
- Then passes beneath the fibrous arch of FDS in the proximal third of the forearm
- About 5–6 cm distal to the elbow it gives off the anterior interosseous branch (motor to FPL, FDP index finger and pronator quadratus)
- In the forearm it descends between FDS and FDP roughly in the midline
- It becomes superficial just above the wrist where it lies between the tendons of FDS and FCR
- Palmar cutaneous branch (sensory to thenar skin) arises 5 cm proximal to the wrist joint, ulnar to FCR and passes over the flexor retinaculum
- Main nerve passes deep to the flexor retinaculum, to the radial side of the tendons of FDS
- The recurrent motor branch to thenar muscles arises at the distal end of the carpal tunnel (see below)
- The nerve median nerve finally ends by dividing into medial and lateral branches to supply the radial 3½ digits

Branches

Near the elbow

- PT
- FCR
- PL
- FDS

In the forearm (anterior interosseous branch[1])

- FPL
- Radial half of FDP
- PQ

In the hand

- Motor – The 'LOAF' muscles of the thenar eminence
  - **L**ateral two lumbricals
  - **O**pponens pollicis
  - **A**bductor pollicis
  - **F**lexor pollicis brevis

Sensory

- Flexor surfaces and nails of the radial 3½ digits
- Skin thenar eminence supplied by the palmar cutaneous branch, which is given off 5 cm above the wrist
- Abnormal connections
- Martin–Gruber (17%) – Median to ulnar nerve in forearm
- Riche–Cannieu (77%) – Deep branch ulnar to median in hand
- Clinically may present as ulnar nerve lesion but no intrinsic deformity, or as severe carpal tunnel syndrome but with no muscle weakness

Carpal tunnel anatomy

- Fibro-osseous tunnel formed by the concavity of the anterior surface of the carpus and roofed over by the flexor retinaculum
- Knowledge of the anatomy of the carpal tunnel is essential to undertake carpal tunnel decompression

Boundaries of the carpal tunnel

- Radial wall: Tubercle scaphoid, ridge of trapezium
- Ulnar wall: Pisiform, hook of hamate
- Floor: Carpus, proximal metacarpals
- Roof: Flexor retinaculum

Contents

- FPL – The most radial structure
- Median nerve (just deep to the flexor retinaculum and lateral to FDS)
- FDS – Lies on the profundus tendons arranged 2-by-2 (middle and ring lie superficial to index and little). *Remember, 34 (third and fourth) is more than 25 (second and fifth)*
- FDP – All together on a deeper plane (lie side-by-side on the floor of the carpal tunnel)
- The FCR tendon lies in a separate fibro-osseous tunnel deep to the flexor retinaculum

Flexor retinaculum

Attachments

- Pisiform and hamate on the ulnar side
- Trapezium and the tuberosity of the scaphoid on the radial side
- There is also a deep slip, which is attached to the medial lip of a groove on the trapezium

Functions

- Prevents bowstringing of the long flexor tendons
- It gives partial insertion to some muscles (PL, FCU)
- It gives partial origin to some muscles (thenar and hypothenar muscles)

Variations of the motor (recurrent) branch of the median nerve

- A key surgical landmark and major surgical danger in carpal tunnel release
- Surface landmark is the intersection of the flexed middle finger tip with Kaplan's line
- There are several variations to the motor branch in the palm, the three most common are
  1. **Extraligamentous branch** (50%) arises distal to the transverse carpal ligament and recurrent to the thenar muscles. The nerve hooks radially and upwards to enter the thenar muscle mass between the FPB and APB muscles
  2. **Subligamentous branch** (30%) arises within the carpal tunnel, emerging distal to the flexor retinaculum and recurrent to the thenar muscles
  3. **Transligamentous branch** (20%) arises from the nerve within the carpal tunnel and pierces the flexor retinaculum
- More rarely the motor branch can either originate from the ulnar side of the median nerve or lie on top of the flexor retinaculum
- NB. Patients with rare variations usually have a large palmaris brevis muscle

Variants of the palmar cutaneous branch of the median nerve

The course of the palmar cutaneous branch of the median nerve may vary in four important ways:

1. Normally the nerve is given off 5 cm proximal to the wrist and runs along the ulnar side of FCR before crossing the flexor retinaculum. The nerve divides into two major branches, medial and lateral, whilst crossing the flexor retinaculum
2. There are two distinct branches, which travel separately across the wrist
3. Within the carpal tunnel and penetrates flexor retinaculum
4. Nerve may be replaced by a branch from the radial or ulnar nerve

Kaplan's cardinal line

- A line drawn from the distal border of the abducted thumb to the hook of hamate
- The recurrent motor branch of the median nerve is estimated by the point where the middle finger tip flexes onto Kaplan's line
- The deep palmar arch lies deep to Kaplan's cardinal line
- The superficial palmar arch lies 2 cm distally, deep to the distal transverse palmar crease

Examination corner

Hand oral

- Clinical photograph of a typical carpal tunnel scar with wasting of the thenar eminence
- This woman has had recent surgery to her hand. What do you think she has had done? (settler question)
- Demonstrate the lines on the examiner's hand that you would make when deciding where to make your incision

- **What are the contents of the carpal tunnel?**
  - As described in the text
- **What is the distal extent of the incision?**
  - In line with Kaplan's line. The carpal tunnel is release when I see the fat pad at the distal end of the retinaculum. I may need to enlarge the skin incision as necessary to ensure I have achieved this.
- **What is the proximal extent of the wound?**
  - The flexor retinaculum should be released. Using a McDonald I assess for any residual proximal to my release and if need be continue the release more proximally until the McDonald can easily be rotated.
- **If you need to extend the incision proximally across the wrist crease how would you do this?**
  - Do not cross the wrist crease at 90° to it – This can cause a scar contracture which can affect function
- **What are the variations in the path of the motor branch?**
  - As described in the text
- **What would you tell the patient would happen with the thenar wasting? How would you manage this?**
  - Very unlikely to recover.
  - Would not routinely address this at the same surgery. Most people function well from a combination of other functioning muscles. Can be addressed down the line with a transfer
- **When would you get nerve conduction studies?**
  - In cases where there is doubt about the diagnosis
  - Recurrence
  - Failure to improve following decompression
  - Negative carpal tunnel provocation test
  - Diabetic patient
  - Neck symptoms
- **Interpret a set of nerve conduction studies (NCS)?**
  - latency = demyelination
  - amplitude = axonal loss

- **What complications would you warn the patient of?**
  - Infection, bleeding, nerve damage, stiffness, swelling and CRPS
  - Scar sensitivity
  - Pillar pain
  - If they have altered sensibility present all the time before surgery this may not improve with surgery but the periods of increased tingling should settle.
- **When can the patient expect to return to work?**
  - Depends on the job
  - Most office based jobs can return at about 2 weeks
  - More manual workers may take 4 weeks
- **When will pinch and grip strength return?**
  - Thumb pinch returns on average at 6 weeks.
  - Grip strength returns on average at 12 weeks.
- **How long can the scar sensitivity take to settle?**
  - Vast majority settle within 6 months.
- **How would you investigate and manage a patient who still had symptoms following surgery?**
  - Need to reassess
  - Was the diagnosis correct?
  - Possible double-crush phenomenon
  - Incomplete release at the time of surgery.

Carpal tunnel syndrome[2]

Incidence

- One per cent in the general population (14% in diabetics)

Pathophysiology

- Believed to be related to pressure applied to the median nerve within the carpal tunnel
- Normal nerve conduction is disrupted by pressure on the myelin sheath of the nerve
- Severe compression may result in Wallerian degeneration of axonal fibres
- Visual inspection at the time of surgery often reveals deformity of the nerve ('hour-glass' appearance) and evidence of an inflammatory reaction woth oedema and hyperaemia
- Any condition that decrease the space and, thus, increase pressure within the tunnel can result in carpal tunnel syndrome such as inflammatory arthropathies, flexor tenosynovitis, generalized oedema of pregnancy or haematoma from trauma
- The vast majority of cases are idiopathic
- The acronym ICRAMPS helps you to remember the causes of carpal tunnel syndrome
  - I – Idiopathic
  - C – Colles', Cushing's
  - R – Rheumatoid
  - A – Acromegaly, amyloid
  - M – Myxoedema, mass, (diabetes) mellitus
  - P – Pregnancy
  - S – Sarcoidosis, systemic lupus erythematosus

Diagnosis

- No agreement on the gold standard for establishing the diagnosis
- Relative importance of history, examination and NCS is unclear. Investigating studies have defined carpal tunnel syndrome on either one or a combination of these criteria
- Concern on relying on NCS is due to:
  - Test only being abnormal when the nerve compression has caused structural damage to the nerve
  - A number of patients having abnormal NCS but being asymptomatic
- In practice most clinicians rely on a combination of history and examination findings. NCS are not routinely performed by most clinicians but reserved for:
  - Good history but negative provocation tests
  - Consideration of 'double crush' phenomenon
  - Failed carpal tunnel surgery
  - Recurrent carpal tunnel syndrome following previous successful decompression,
- Symptoms
  - Paraesthesia in radial 3½ digits
  - Worse at night
  - Weakness in the hand, dropping things
  - Pain
  - 40% bilateral involvement
  - Male : Female ratio: 1 : 6
  - Not always classical
- Examination
  - Examine neck movements
  - Swelling over volar aspect of the wrist
  - Wasting of thenar muscles
  - Sensation (light touch, two-point discrimination)
  - Power (APB)
- Provocative tests
  - Median nerve compression test (Durkin's) – Apply direct pressure with your thumb over the nerve, with the patient's elbow extended and wrist flexed 60° (86% sensitivity, 83% specificity)
  - Tinel's sign (74% sensitivity, 90% specificity)
  - Phalen's test (positive if signs <60 s) (61% sensitivity, 83% specificity)
- Nerve conduction studies (NCS):
  - Abnormal if:
    - Sensory conduction is prolonged >3.5 ms (demyelination)
    - Increased distal motor latency >4.0 ms
    - Decreased amplitude (axonal loss)
    - Accurate 85–90%
    - False-negative rate 10–15%

- Differential diagnosis
  - Cervical disc disease
  - Peripheral neuropathy – Alcohol, diabetes
  - Pronator syndrome (discussed below)
  - Spinal cord lesions – Tumour, syrinx, MS
  - Thoracic outlet syndrome
  - Collagen vascular disorders
  - Raynaud's disease
  - CRPS

Management

Non-operative

Indicated in those with mild intermittent symptoms without neurological impairment, who have had symptoms <1 year, and who have no muscle wasting. The classic indication is a temporary, reversible carpal tunnel syndrome (pregnancy). Patients with pure nocturnal symptoms may also benefit from a trial of non-operative treatment.

- Splintage in neutral (extension increases tunnel pressure)
  - Evidence it is more effective than no treatment for symptom relief and function for at least 3 months
  - No evidence that full time splinting is better than nocturnal splinting
- NSAIDs have no clear evidence of benefit
- Steroid injections
  - 80% transient relief
  - 20% symptom-free at 12 months
- Ultrasound
  - Evidence of benefit compared to placebo in 2 studies for at least 6 months
- Physiotherapy
  - Not proven
- Combination therapy
  - A study comparing NSAIDs, splinting and hand therapy vs surgery revealed better outcome at both 6 and 12 months

Surgical

Indicated in those with progressive persistent symptoms with neurological defects.

Open carpal tunnel decompression

- Ninety-five per cent of patients have good or excellent results
- Allows good visualisation of the median nerve and the contents of the tunnel
- The physiological state of the nerve can be assessed

Incision – A safe incision is offered (Figure 21.1): Longitudinal from the distal wrist crease and just ulnar to palmaris longus (found by opposing the thumb to the little finger and flexing the wrist), in line with the radial border of the ring finger, and distally to Kaplan's line. It is not routinely recommended to cross proximal to the wrist crease Some recommend extending your incision proximal to the distal wrist crease to make sure the fascia of the distal forearm is released as this can cause compression of the nerve. Most feel that this structure can be adequately decompressed with a smaller incision using scissors and retractors to protect the median nerve. Still others feel that decompressing the transverse carpal ligament is sufficient. However, you should not cross perpendicular to a flexion crease

Adjunctive surgical procedures – No demonstrable benefit of additional synovectomy or internal neurolysis following carpal tunnel release and may lead to adhesions

Endoscopic carpal tunnel release

- Introduced to reduce the incidence of pillar pain but this has not been demonstrated
- Use either the Agee (one-incision) or Chow (two-incision) technique. Steep learning curve with increased early complication rate, including actual injury to the median nerve
- Pain less at 3 months compared to open release, but no difference at 1 year[3]

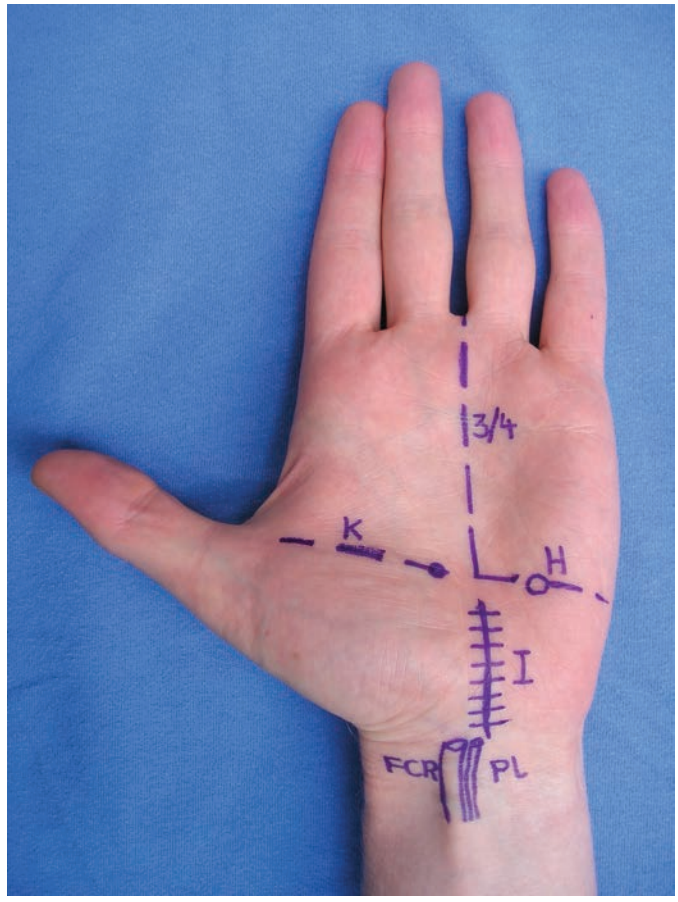

Figure 21.1 Landmarks for open carpal tunnel decompression. I, Incision; H, hook of hamate; K, Kaplan's line; black spot, motor branch; PI, palmaris longus; FCR, flexor carpi radialis

Complications of carpal tunnel release

- **Infection:** <1%
- **Tender scar:** Owing to division of very fine terminal branches of the palmar cutaneous nerve
- **Haematoma:** Major bleeding is rare if safe anatomical landmarks are observed
- **Dehiscence:** Sutures removed too early
- **Damage to nerves:** Recurrent motor branch of the median nerve, palmar cutaneous branch median nerve, ulnar nerve
- **Pillar pain:** Pain felt when pushing down on the base of the hand following carpal tunnel release. Aetiology uncertain, possible because of gradual stretching of intercarpal ligaments, which are no longer de-tensioned by the flexor retinaculum. Others suggest that division of the flexor retinaculum disturbs the alignment of the pisotriquetral joint, which is the source of pillar pain
- **Complex regional pain syndrome:** Rare but always mention in consent
- **Weakness of pinch:** Returns to preoperative levels in a mean of 6 weeks
- **Weakness of grip:** Returns to preoperative levels in mean of 3 months
- **Bowstringing of flexor tendons:** More a theoretical complication than a practical one

Failed carpal tunnel release

- Recurrent or persistent symptoms in up to 20%
- Need to consider whether the persistence of symptoms os due to inadequate release, wrong diagnosis, 'double crush' phenomenon or due to the severity of the nerve compression prior to surgery
- Patients that had altered sensibility all the time before surgery should be aware that this may not imrpove following surgery or may take several months to do so
- Persistence of night-time symptoms should raise the suspicion of incomplete release
- Consider other differential diagnoses including cervical radiculopathy at C5/6, compression of the upper trunk brachial plexus and proximal median nerve compression, double crush phenomenon relates to more than one site of compression that, if in isolation, would not cause symptoms, but together cause symptoms. Perform NCS after at least 3 months
- **Symptoms unchanged:** Wrong diagnosis, inadequate decompression, postoperative fibrosis, double crush phenomenon
- **New symptoms:** Normal structures damaged at surgery, new diagnosis

Re-exploration is indicated if:

- Marked symptoms
- Other causes of symptoms ruled out
- Positive provocative tests
- Positive NCS after 3–6 months
- ?Relief of symptoms after steroid injection into carpal tunnel

Pronator syndrome[1]

Background

- Compression of the median nerve in the elbow or forearm
- Results in altered sensation in the median nerve distribution and thenar eminence
- Much less common than carpal tunnel syndrome
- Reported in individuals who do repetitive upper extremity activity

Sites of entrapment

- Ligament of Struthers – 1% of population (supracondylar process)
- Bicipital aponeurosis (lacertus fibrosus)
- Origin of the pronator teres (abnormal anatomy, tight fibrous bands)
- Proximal arch of FDS

Clinical

- Pain or ache in the volar proximal forearm and tender to palpation
- Paraesthesia of the median nerve-innervated digits and thenar eminence (palmar cutaneous branch – Not involved in carpal tunnel syndrome)
- Usually no night symptoms of paraesthesiae or tingling
- Weakness of the forearm or clumsiness of the hand. Phalen's test generally negative as the site of compression is proximal to the transverse carpal ligament. Tinel's test is negative at the wrist but may be positive at the proximal anterior aspect of the forearm

Provocative tests

- Resisted elbow flexion with forearm supination (bicipital aponeurosis)
- Resisted forearm pronation with elbow extended (two heads of pronator teres)
- Isolated long finger PIP joint flexion (FDS origin)

Investigations

- NCS/EMGs usually negative
- Radiographs of the distal humerus may show the (rare) supracondylar process

Management

- Usually responds to non-operative treatment – Modification of activities, NSAIDs, heat and massage
- If not settled after 6–12 months, surgical decompression of all potential sites of compression

- Decompression
  - 10 cm lazy 'S' incsion from the antecubital fossa heading distally (extended proximally if a ligament of Struthers is found)
  - Divide bicipital aponeurosis
  - Identify the superficial head of pronator teres which should be 'step cut' to lenghten the tendon and, thus, relieve any compression
  - Deep head of pronator teres is released as it arches around the median nerve
  - The tendinous portion of the FDS arch is divided

Anterior interosseous syndrome[4]

Background

- A pure motor entrapment. There is no sensory disturbance
- AIN supplies FDP to index and middle fingers, FPL and PQ

Tests

- Direct pressure over the nerve may elicit symptoms (Tinel's sign negative)
- Symptoms occur acutely with sudden onset of dull non-specific pain in the proximal third of the volar forearm
- Weakness of flexion at the DIP joint of the index finger and IP joint of the thumb (OK sign)
- The middle finger profundus may have some weakness but usually has some function because of cross-over innervation from the ulnar nerve
- Mild weakness of pronation
- 'OK' sign – Tip-to-tip pinch. Tests FDP to the index finger and FPL. If patient makes a square instead of a circle this is called the Kiloh–Nevin sign
- To isolate PQ – Test resisted pronation with the elbow maximally flexed (elbow flexion eliminates pronator teres)

Differential diagnosis

- Parsonage–Turner syndrome (bilateral AIN syndrome – Viral brachial neuritis, motor loss preceded by intense pain in the shoulder region)
- Mannerfelt–Norman syndrome (FPL rupture)

Sites of constriction

- Deep head of the pronator teres muscle (most common site of compression)
- FDS arcade
- Enlarged bicipital bursa
- Gantzer's muscle (accessory head FPL)

Investigations

- NCS usually unhelpful

Management

Non-operative management

- Elbow splinting in 90° of flexion, NSAIDs, etc
- Many symptoms and signs will gradually resolve in time

Surgical decompression

- This is indicated following the failure of conservative treatment for 6 months
- Surgery involves complete exposure of the AIN from its origin from the median nerve – Approach as for pronator syndrome
- Results are unpredictable

Examination corner

Hand oral: clinical photograph of the OK sign – Patient making a square instead of a circle

- Spot diagnosis (Kiloh–Nevin sign)
- **How would you exclude an FPL rupture?**
  - Tenodsis test
  - USS
- AIN-innervated muscles
  - FPL, FDP index ± middle and pronator quadratus
- **What are the sites of compression?**
  - Deep head of the pronator teres muscle (most common site of compression)
  - FDS arcade
  - Enlarged bicipital bursa
  - Gantzer's muscle (accessory head FPL).
- **Management?**
  - Splints and non-operative for 6 months
  - Consider decompression at 6 months.

Anatomy of the ulnar nerve

Nerve roots

The nerve arises from the medial cord of the brachial plexus (C8, T1).

Course

- Travels medial to the brachial artery in the upper arm
- Passes through the medial intermuscular septum at the midpoint of the arm to enter the posterior compartment
- Through the arcade of Struthers (band of fascia from the medial head of triceps to the medial intermuscular septum) approximately 8 cm proximal to the medial epicondyle
- Passes posterior to the axis of the elbow joint between the medial epocondyle and olecranon in the cubital tunnel
- Enters the forearm between the humeral and ulna heads of FCU

- Passes distally on the medial side of the forearm on FDP and deep to FCU
- Gives off the dorsal cutaneous branch 5 cm proximal to the wrist
- At the wrist lies between FCU tendon and ulnar artery (ANT – Artery–nerve–tendon)
- Passes through Guyon's canal between the pisiform and the hook of hamate and divides into superficial and deep branches at the distal end
- Superficial branch supplies palmaris brevis and is sensory to the ulnar 1½ digits
- Deep (motor) branch passes ulnar to the hook of hamate and then deep between the heads of origin of flexor and abductor digiti minimi

Branches

Motor

- FCU
- FDP to little and ring fingers
- All small muscles of the hand except LOAF (see Anatomy of the median nerve above)

Sensory

- Ulnar 1½ digits
- Medial skin palm

Examination corner

Hand oral

- **Describe the course of the ulnar nerve?**
  - As described above.
- **Explain the ulnar paradox?**
  - Normally you would expect a higher nerve lesion to create greater deformity. The opposite is true with the ulna nerve. In both a high and low ulnar nerve lesion the intrinsics are lost. In the low ulnar nerve lesion the long flexors and extensors are not adjusted by the intrinsics and so a claw appears. In a high ulnar nerve the long flexors are also lost so the fingers are less clawed.
- **How would you differentiate between an ulna or T1 lesion?**
  - Sensory loss in T1 distribution
  - Motor loss will affect the thenar eminence too.
- **What are the sites of compression at the elbow?**
  - See text.
- **What technique you will use and why, i.e. simple decompression, medial epiconylectomy or transposition?**
  - There is currently no conclusive evidence for one treatment over another.
  - Proponents of the transposition site the traction element of injury to the nerve on elbow flexion.
  - Certainly if the nerve is unstable (i.e. it flicks over the epicondyle on elbow flexion) the nerve should be transposed.
- **What structure do you need to look out for and protect during the approach to the cubital tunnel?**
  - Antebrachial cutaneous nerve.
- **How would you address the nerve if it subluxed?**
  - Transpose.
- **What functional problems do the patients have and what tendon transfers could you use to address these?**
  - Depends on the level of compression.
  - Discuss with the patient on the functional problems.
  - See the tendon transfers section later.

Ulnar nerve compression

Aim to diagnose whether the patient has a high or a low lesion. Cubital tunnel syndrome is far more common (95%).

Symptoms

- Paraesthesia of the ulnar 1½ digits (and ulnar dorsal aspect of the hand if the lesion is proximal to the wrist)
- Difficulty in fine motor activities
- Weakness of pinch grip (adductor pollicis)

Signs

- Little finger escape (Wartenberg's sign) – Abduction of the extended little finger in the line of pull of EDM owing to weakness of the third palmar interosseous muscle
- Ulnar claw hand
- Wasting of the small muscles of the hand (hypothenar eminence, metacarpal guttering, first dorsal interosseous)
- Wasting of the ulnar border of the forearm (FDP and FCU)
- Decreased sensation in the ulnar 1½ digits ± dorso-ulnar hand
- Positive Tinel's test behind the medial epicondyle of the elbow
- Weakness of the interossei and ulnar two lumbrical muscles
- Positive Froment's test – FPL (anterior interosseous nerve) compensating for weakness of adductor pollicis; see Figure 21.2)
- Jeanne's sign – Hyperextension of the thumb MP joint (involvement of FPB)
- Weakness of FDP of little and ring fingers (Pollock's test)
- Positive elbow flexion test – Maximally flexing the elbow produces pain and paraesthesiae in the ulnar nerve distribution within 60 seconds

Ulnar paradox

- The expectation with a nerve lesion is that a more proximal lesion would result in a greater deformity. However, there is less clawing of the hand with the more proximal the nerve lesion

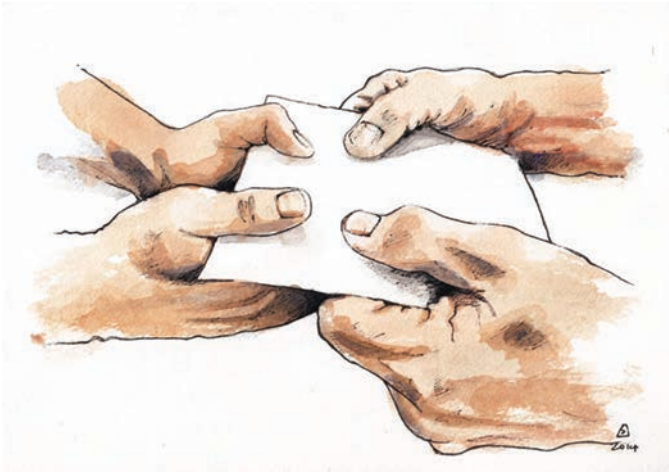

Figure 21.2 Froment's test. The patient is on the left. Notice how on he can hold the paper with thumb adduction on the right hand (ulna nerve) but needs to use thumb IPJ flexion (AIN) on the left hand. (Picture Courtesy of ©DonaldSammut 2014)

- In a distal lesion only the intrinsics are weak and so the stronger pull of the flexors overpowers the extensors resulting in a claw hand
- In a more proximal lesion the flexors are also weakened, thus, reducing the amount of flexion at the IP joints of the little and ring fingers by leaving the extensors unopposed and so the hand appears less clawed

Cubital tunnel syndrome[5]

- Second most common compression neuropathy after carpal tunnel syndrome
- Chronic compression of the ulnar nerve may occur as a result of ischaemia or mechanical compression by repeated elbow flexion, direct compression or post-traumatic scarring
- Subluxation of the nerve may also add to the direct trauma to the nerve
- The size of the cubital tunnel decreases with elbow flexion
- Both tunnel and intraneural pressure increases with elbow flexion

Sites of entrapment

1. Medial intermuscular septum
2. Arcade of Struthers – A thick fascial structure arising from the medial head of triceps to the medial intermuscular septum. It arises 8–10 cm prixmal to the medial epicondyle
3. Osborne's ligament – A fibrous aponeurotic arch (Osborne's ligament/cubital tunnel retinaculum)
4. Fibrous arch connecting the two heads of FCU
5. Between the two heads of FCU (flexor carpi ulnaris aponeurosis)

Other causes of compression

- **Bony abnormalities:** Osteophytes, cubitus valgus
- **Anconeus epitrochlearis muscle** (accessory muscle): Vestigial muscle originating from the medial border of the olecranon and inserting into the medial epicondyle, and crossing over the cubital tunnel. It is an uncommon cause of ulnar nerve compression at the elbow, which may be bilateral
- **Constricting fascial bands**
- **Tumours, ganglions**
- **Scarring**
- **Recurrent subluxation of nerve around medial epicondyle**

Differential diagnosis

Suspected cubital tunnel syndrome may be mimicked by other disorders, including:

- Cervical disc disease
- Spinal tumours
- Thoracic outlet syndrome
- Apical lung tumour
- Post-radiotherapy brachial plexopathy

Oral question

- How do you differentiate a T1 nerve root lesion from an ulnar nerve palsy?
- **A patient with a T1 root lesion may have a Horner's syndrome, paraesthesia over the medial aspect of the forearm and weakness of all small muscles of the hand with clawing of all four fingers.**

Management

Conservative management

- This should be the first step in treating patients in the absence of severe continuous symptoms with clinical signs of sensory loss or muscle weakness
- Patients with mild, intermittent symptoms should be educated to avoid direct pressure to the nerve, use elbow pads and modify activities to prevent prolonged periods of elbow flexion
- Night splints to hold the elbow in 40–50° of flexion (studies have shown this position to thave the lowest intraneural pressures)
- Conservative treatment may relieve symptoms of ulnar nerve dysfunction at the elbow in as many as 50% of patients
- Corticosteroid injections at the elbow are best avoided because of the high incidence of fat necrosis

Surgery

Indicated following failure of non-operative measures or clinical evidence of sensory and/or motor loss or severe neuropathy on NCS.

Operations used include:

1. **Simple decompression** (release of the cubital tunnel retinaculum)
2. **Decompression with medial epicondylectomy:** Theoretical advantage of a more extensive decompression than simple release without the disturbance in blood supply of the nerve. It is also thought to help negate the traction forces with elbow flexion. Concerns with this technique are medail elbow tenderness (up to 70%) and valgus instability (due to damage to the MCL origin). These concerns have been reduced by using a partial epicondylectomy (5–7 mm only)
3. **Anterior transposition** of the ulnar nerve, which may be to the subcutaneous, submuscular or intramuscular positions. Anterior transposition risks devascularization of the nerve. It is best indicated when either:
   - A bony deformity is present in the groove behind the medial epicondyle, or
   - The nerve exhibits a tendency to sublux or dislocate with elbow flexion and extension

Transposition of the ulnar nerve can be to:

- The subcutaneous tissues above the fascia of the flexor pronator group
- Within the musculature of the flexor pronator group itself with the fascia repaired
- Beneath the flexor pronator group with the origin repaired to the medial epicondyle

There is currently no evidence that one surgical technique is superior to any other regardless of the subjective clinical or objective nerve conduction assessments preoperatively. In most straightforward cases there is very little difference in outcome whether the nerve is treated by simple decompression, medial epicondylectomy, subcutaneous transposition or submuscular transposition. A satisfactory outcome is achieved in approximately 80% of patients using any of these techniques and function generally returns within 6 months.

> 'There are several surgical options available to treat this condition, my preference would be . . . because . . .'

The nerve is safely found proximal to the cubital tunnel and decompressed distally into FCU.

Complications from surgery

- Infection
- Scar tenderness
- Neuromas (medial antebrachial cutaneous nerve)
- Complex regional pain syndrome
- Failure to relieve symptoms following decompression owing to either the presence of severe intraneural fibrosis or inadequate decompression
- Disruption of blood supply to the nerve
- Irritation of superficially placed nerve

Following the failure of a previous decompression of the ulnar nerve at the elbow, treatment generally consists of some form of anterior transposition, most commonly utilizing a submuscular or intramuscular technique. Success in improving symptoms and function is reported in as many as three out of four patients.

Ulnar tunnel syndrome

This is caused by ulnar nerve compression in Guyon's canal. It is much less common than entrapment of the nerve at the elbow. Pain is usually less significant when compared to ulnar nerve compression at the elbow or carpal tunnel syndrome.

Signs

- Local tenderness, Tinel's test, Phalen's sign, severe ulnar clawing, weakness, atrophy, paraesthesia of the ulnar 1½ digits (Figure 21.3)
- Dorso-ulnar sensory branch spared

Anatomy of Guyon's canal

- Roof: Volar carpal ligament
- Ulnar wall: Pisiform
- Radial distal wall: Hook of hamate and ADM
- Floor: Transverse carpal ligament and pisohamate ligament

The ulnar nerve and artery lie beneath the volar carpal ligament on top of the transverse carpal ligament in Guyon's canal.

Causes of compression

The causes of compression are numerous and include:

- Typically ganglion or lipoma
- Tumours
- Thrombosis/pseudoaneurysm of the ulnar artery
- Pisiform instability
- Pisotriquetral arthritis

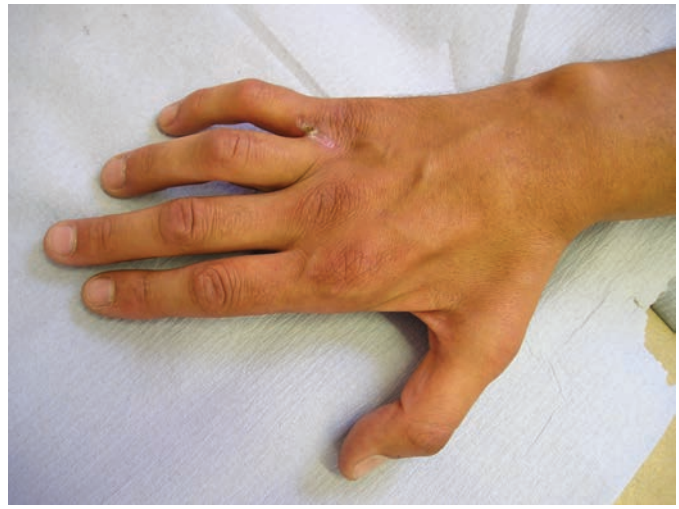

Figure 21.3 Ulnar nerve palsy

- Fractures distal to the radius/ulnar, hook of hamate, pisiform
- Palmaris brevis hypertrophy
- Muscle anomalies

Symptoms

Symptoms may be pure motor, sensory or mixed based on the location of compression within the tunnel. The tunnel is divided into three zones.

Zone 1

Area proximal to the bifurcation of the nerve. Combined motor and sensory symptoms.

Zone 2

Surrounds the deep motor branch and has pure motor symptoms only. Ganglions and hook of hamate fracture are the most likely aetiology in zones 1 and 2.

Zone 3

Surrounds the superficial sensory branch of the ulnar nerve and has sensory symptoms only. Thrombosis or aneurysm of the ulnar artery is the most likely aetiology in zone 3. Allen's test and Doppler studies are useful in making the diagnosis.

Differential diagnosis

- Cubital tunnel syndrome
- Cervical disc disease
- Thoracic outlet syndrome
- Motor neurone disease (motor signs only)

Management

The key to management is identifying the aetiology:

- NCS
- MRI for ganglions
- CT for hook of hamate fractures
- Doppler US for ulnar artery thrombosis

Conservative

- Wrist splinting
- Avoidance of repetitive trauma

Surgical

- Decompression of both motor and sensory branches ± excision of the pisiform/hook of hamate
- Release of the volar carpal ligament, isolating the ulnar nerve proximal to the wrist initially with a longitudinal incision radial to FCU
- Occasionally patients are seen who have carpal tunnel and Guyon's canal compression syndrome simultaneously. The volume of Guyon's canal increases after carpal tunnel release, and ulnar compressive symptoms improve in approximately one-third of patients following carpal tunnel release alone

Radial nerve compression

Anatomy

- Arises from the posterior cord of the brachial plexus (C5, 6, 7, 8)
- Passes through the triangular space to enter the spiral groove where it lies on the medial head of triceps
- Pierces lateral intermuscular septum ~7 cm proximal to lateral epicondyle
- Passes deep to the mobile wad in the radial tunnel, giving off a superficial branch
- Becomes the posterior interosseous nerve (PIN) as it passes deep to the superficial head of supinator

Sites of entrapment

- Axilla (Saturday night palsy)
- Humeral shaft fracture – It was thought that a radial nerve palsy has a higher incidence with a Holstein–Lewis fracture (junction of middle and distal third humerus), but a meta-analysis[6] has shown that it is more likely with a midshaft fracture. This paper showed an overall incidence of radial nerve palsy of 11.8% with a humeral fracture, and 88% recover

Examination corner

Hand oral

- A patient demonstrates a wrist and finger drop following a humeral shaft ORIF

- **How will you manage this patient intially?**
  - If a posterior approach was used and the nerve has been clearly seen at surgery and is known to be intact, this is most likely to be a neuropraxia. In this case I would manage the patient non-operatively with a wrist extension splint.
- **When would you re-explore the nerve?**
  - If the nerve had not been clearly seen or any evidence of a haematoma.
- **If so, what would you do if the plate was sat on top of the nerve?**
  - I would remove the plate to release the nerve, re-fix the fracture and discuss the patient with a peripheral nerve surgeon. This section of the nerve will probably need excision and then repair, probably by grafting.
- **If not, at what stage will you get nerve conduction studies and EMGs?**
  - Four to six weeks should show evidence of reinnervation.
- **What test can you perform in the clinic to check for reinnervation?**
  - Advancing Tinel's.
- **What muscle will be the first to re-innervate?**
- **What are the tendon transfers for this patient?**
  - Pronator teres for wrist extension
  - Palmaris Longus for thumb extension
  - FCR for finger extension.

Posterior interosseous nerve compression

Introduction

- Pain at the lateral elbow
- Weakness of wrist extension with radial drift (ECRL innervated higher than PIN take-off)
- No sensory loss

Clinical features

- Onset often insidious
- Dull aching of the proximal forearm
- Difficulty extending fingers and thumb
- Wrist extension is still possible (ECRL not affected) but it is weak plus there is an element of radial deviation (ERCB)
- Able to extend the IP joints due to interossei

PIN innervates nine muscles

- ECRB, supinator, EIP, ECU, EDC, EDM, APL, EPB, EPL

Sites of compression/entrapment

- Thickened fascia at radiocapitellar joint
- **R**adial artery recurrent leash of Henry
- **E**dge of ECRB
- **A**rcade of Frohse (tendinous proximal border of supinator)
- Distal edge of Supinator

Unusual causes

- Parsonage–Turner syndrome
- Chronic radial head dislocation
- Fracture of the radial head or neck
- Synovitis of the radiocapitellar joint
- Mass lesion (lipoma, ganglion) at the elbow

Differential diagnosis

- C7 radiculopathy
- Lateral epicondylitis

Management

- Conservative initially as many patients will spontaneously improve
- Avoidance of aggravating activities
- NSAIDs
- Full surgical decompression of all potential compression sites if no improvement after prolonged non-operative management

Radial tunnel syndrome

Introduction

- This is a pain syndrome and a clinical diagosis
- There is no motor or sensory dysfunction
- Because of the close proximity of the nerve to the lateral epicondyle, the condition can be difficult to differentiate from a 'resistant' tennis elbow (coexists in 5% of patients)

Anatomy of the radial tunnel

- Medial: Biceps tendon and brachialis
- Lateral: Brachioradialis, ECRL and ECRB
- Roof: Brachioradialis
- Floor: Radiocapitellar joint capsule and supinator muscle

Clinical features

History

- Deep-seated dull aching/pain in the extensor muscle mass, often radiating to the wrist

Examination

1. **Localized tenderness** directly over the PIN distal to the lateral epicondyle
2. **Middle finger extension test** – Each finger is tested under resisted extension. Testing the middle finger (firm pressure over the dorsum of the PP) increases the pain because the ECRB inserts into the base of the third metacarpal. The test is positive if it produces pain at the edge of the ECRB in the proximal forearm. It is performed with the elbow and middle finger completely extended and the wrist in neutral position
3. **Resisted active supination test** – The radial tunnel begins at the radiocapitellar joint and extends to the end of the supinator muscle. In radial tunnel syndrome the maximal tenderness is distal to the radial head in a line from the lateral epicondyle through the radial head to a point 2–3 cm more distal over the radial tunnel

Causes

As for PIN syndrome but not usually any mass lesions:

1. **F**ibrous bands tether nerve to the radial humeral capsule
2. Radial **R**ecurrent leash of vessels (the leash of Henry)
3. Fibrous medial edge along **E**CRB
4. Fibrous **A**rcade of Frohse (proximal superficial edge of supinator)
5. **S**upinator (distal border)

Investigations

- Diagnostic injection of local anaesthetic into the radial tunnel
- NCS not particularly helpful, as they are usually normal, which is in contrast to the PIN compression syndrome

Management

Non-operative

- Long non-operative approach warranted
- Activity modification

- Temporary splinting
- NSAIDs

Surgical

- Operative release is often disappointing, with only 50% satisfactory results
- Coincidental undiagnosed tennis elbow can lead to failure of radial tunnel decompression

Wartenberg's syndrome (cheiralgia paraesthetica)

- Uncommon condition
- Originally described in 5 patients by Wartenberg in 1932
- Radial sensory nerve is superficial in the distal forearm which renders it suscpetible to external compression
- Neuritis/compression neuropathy of the superficial sensory branch of the radial nerve

Causes

- Typically it is due to external compression from watches or jewellery
- In pronation the nerve can be compressed between the tendinous insertions of brachioradialis and ECRL
- The nerve undergoes traction when the wrist is moved from radial extension to ulnar flexion
- Anomalous fascial bands
- Thrombosis of the radial recurrent vessels
- Haemorrhage in the proximal forearm

Clinical features

- Burning, numbness or pain in the distribution of the superficial radial nerve (outer aspect of the distal forearm)
- Tenderness along the course of the nerve proximal to the wrist
- The most useful sign is a positive Tinel's test directly over the nerve as it exits from beneath brachioradialis
- Dellon's sign – Active forceful pronation of the forearm and ulnar deviation of the wrist with the elbow extended by the side for 60 seconds may provoke symptoms of numbness or tingling in the territory of the sensory branch

Management

Conservative

- Remove watches, jewellery or any garment compressing the region
- NSAIDs
- Wrist splint
- Steroid injection
- Approximately 50% of patients respond to conservative treatment

Surgery

- Should only considered in patients with continual troubling symptoms following a prolonged period of non-operative management
- Involves exploration and release of constricting tissue
- The nerve is susceptible to damage and neuroma formation and a very careful release with a 'no touch technique' is required
- Results of surgical exploration are unpredictable

Examination corner

Short case 1

- Lipoma in the forearm causing pressure neuritis of the superficial radial nerve
- Clinical findings: Numb over the dorsum of index finger and first web space

Dupuytren's disease[7–14]

This is an absolutely must-learn topic for both the clinical short cases and hands oral.

Epidemiology

- A benign fibromatosis of the palmar and digital fascia
- It is thought to be inherited through an autosomal dominant pattern with variable penetrance, though this is unproven
- Greater than 25% of men of Celtic origin over 60 years of age have evidence of Dupuytren's disease
- The male : female reported ratios vary from between 4 : 1 and 10 : 1.
- Those of oriental origin and diabetics tend to have palmar disease but not joint contracture.
- There is no reported predilection for side or dominance.
- Unilateral disease is more commonly a sporadic finding without a family history and is usually less severe.
- In women, Dupuytren's disease is typically seen later and is usually less severe.

Cellular pathology and pathophysiology

- Classically described in two forms: Nodules and cords
- Nodules
  - Palpable subcutaneous lumps that may be fixed to the skin and/or palmar fascia
  - Dense hypercellular and hypervascular
- Cords
  - Highly organized parallel collagen fibre structures
  - Relatively hypocellular
  - Increased cellularity is associated with higher recurrence rates

  - Predominantly type III collagen (in contrast to the type I in normal fascia)
- Nodules are commonly found throughout cords
- Trauma, ischaemia and microvascular angiopathy are thought to play key roles in the development of the disease
- There are two main theories on the mechanism of pathological change
  1. **Intrinsic theory** – Metaplasia of the existing fascia
  2. **Extrinsic theory** – A subdermal origin for the diseased tissue that attaches itself to and grows on underlying fascial bands
- The myofibroblast is the key cell and contains actin, allowing active contraction. These cells produce fibronectin, to link to other myofibroblasts, and increased amounts of type III collagen
- Oxygen free radicals that occur in hypoxic tissue are thought to play a role via fibroblast stimulation
- Cytokines (TGF-β, platelet-derived growth factor, fibroblast growth factor) are also probably involved, and stimulate transformation of fibroblasts into myofibroblasts, resulting in fibrous hyperplasia of the palmar fascia
- There are three stages described by Luck
  1. **Proliferative:** Large myofibroblasts, very vascular, minimal extracellular matrix, random cell proliferation
  2. **Involutional:** Dense myofibroblast network, increased amounts of type III collagen fibres compared to type I
  3. **Residual:** Myofibroblasts disappear and are replaced by fibrocytes

Anatomy[11]

- Dorsal skin is loosely attached where as the palmar skin is firmly attached to the underlying fascial tissue and skeleton. An understanding of the normal fascia is crucial to managing this disease
- Palmar fascia consists of radial, ulnar and central palmar aponeuroses
  - Radial aponeurosis (least commonly affected in Dupuytren's disease) is subdivided
    - Thenar fascia
    - Pretendinous band of the thumb
    - Proximal commissural ligament (thenar extension of the transverse ligament of the palmar aponeurosis)
    - Distal commissural ligaemtn (thenar extension of the natatory ligament)
  - Ulnar aponeurosis is subdivided
    - Pretendinous band to the little finger
    - Hypothenar fascia
    - Abductor digiti minimi confluence
  - Central palmar aponeurosis (the most affected part in Dupuytren's disease) is subdivided into three layers: Longitudinal, vertical and transverse
    - Longitudinally this appears as a triangular structure that fans out distally into the pretendinous bands. The pretendinous bands bifurcate and have three distinct layers of insertion
      - Superficially – Into the dermis
      - Middle layer – Wraps around the metacarpal head by twisting and travelling adjacent to the MCP joint capsule to form the spiral band. This continues deep to the NV bundle and emerges distally as the lateral digital sheet
      - Deep – Into the flexor mechanism
    - Vertical layer consists of Grapow fibres and the septa of Legueu and Juvara
      - Grapow fibres anchor the dermis to the palmar aponeurosis
      - Eight vertical septa of Legueu and Juvara create seven fibro-osseous compartments deep to the palmar fascia
      - Four compartments each containing the paired tendons to each finger
      - Three compartments containing the NV bundles and associated lumbrical
    - Transverse fibres consist of the superficial transverse ligament (Skoog's ligament) and the more distal natatory ligament
  - Digital
    - Neurovascular bundles are surrounded by four structures
      - Lateral digital sheet located on both radial and ulnar sides of the digit is a continuation of the spiral band and natatory ligament
      - Grayson ligament volarwards
      - Cleland ligament dorsally
      - Retrovascular fascia medially
- The bands are normal facial structures but are referred to as cords when they become diseased
- A spiral cord (Figure 21.4) is one which entwines the neurovascular bundle pulling it towards the midline where it can be easily injured by the ill-prepared surgeon. It occurs when several structures are involved including
  - Pretendinous band
  - Spiral band
  - Lateral digital sheet
  - Grayson's ligament

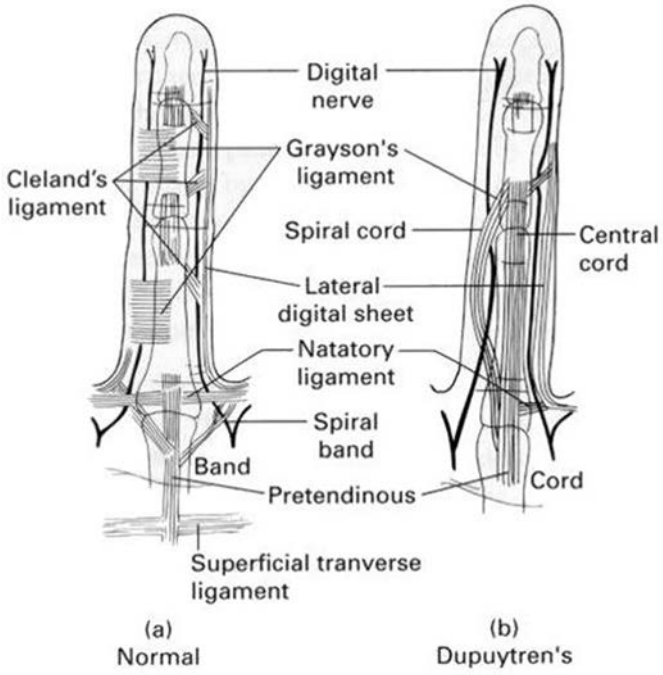

Figure 21.4 Spiral cord

Risk factors for Dupuytren's disease

- Positive family history
- Northern European heritage
- Male
- Alcohol excess
- Diabetes
- Chronic lung disease
- Smoking
- AIDS
- Epilepsy (this is disputed and may be related to the medication)
- Trauma (in a genetically suscpetible individual)

Dupuytren's diathesis

- This is an aggressive form of the disease and should be considered in patients presenting with

  Early onset disease
  Involvement of the radial side of the hand
  Both hands
  Ectopic disease

  - Ledderhose's disease – Plantar fibromatosis
  - Peyronie's disease – Penis
  - Garrod's pads – Nodules over the dorsal surface of the PIP joints

Symptoms

- This is not a painful condition and other pathology should be considered if pain is a signifcant feature. Patients can, however, describe pain in some nodules which are thought to be in the proliferative phase
- A progressive digital deformity as a result of cord formation
- Difficulty in undertaking activities of daily living
- Decreased manual dexterity

History

- Hand dominance
- Family history
- Rate of progression
- Diabetes
- Epilepsy
- Alcohol
- Foot/penis involvement
- Smoking
- Trauma
- Previous treatment
- Assessing the impact of functional difficulty
- Ascertain the expectations of treatment

Examination

- Previous scars
- Skin pits
- Digits involved and cord type
- MCP angle (measure with PIP joint fully flexed as cord can cross both joints)
- PIP angle (measure with MCP joint fully flexed)
- Garrod's pads over the dorsal PIP joint
- Sensation
- Digital Allen's test
- Mention Ledderhose's and Peyronie's
- The combination of PIP joint in a fixed flexion deformity with the MCP joint in flexion signifies a severe deformity and a poor prognosis

Indications for treatment

- Classically the Hueston's tabletop test
  - Involves placing the hand and fingers prone on a tabletop
  - The test is positive when the hand will not go flat
  - Rarely alters management decisions, but is of value as a screening test for general practitioners to identify those patients requiring referral
- Consideration of treatment should be given when
  - MCP joint contracture >30°
  - PIP joint contracture >15°
  - First web space contracture
- Remember
  - A severe MCP joint contracture will correct with excision of the cord as the collateral ligaments are not in a shortened position

  - However, a PIP joint contracture leads to a stiff joint as the collateral ligaments and volar plate tighten
- Excision of an early nodule may cause a flare-type reaction, leading to an early return of the disease

Informed consent

- Aims – Excision of the diseased tissue to restore full movement. The operation is not curative
- General/regional anaesthetic
- Complications
  - Recurrence rate 30–50% at 10 years[8]. Not all require further surgery
  - Incomplete correction of PIP joint contracture
  - Wound healing delay
  - Temporary or permanent digital nerve impairment (1.5%)
  - Splinting regimen postoperative (night splints up to 6 months)
  - Cold intolerance
  - Amputation

Treatment options

Collagenase injections[13,14]

- Currently licensed for use in Dupuytren's disease with a palpable cord
- Consists of two different collagenase enzymes (AUX-1 and AUX-II) taken from *Clostridium histolyticum*
- These enzymes primarily have action on collagen types I and III
- Injections are placed in the cord through a single puncture site with the enzymes working synergistically to cleave the collagen chains at the injection site
- The injection should be performed optimally where the cord is easily palpable and furthest away from the tendon. Only one cord should be treated at any one time. The MCP joint should be corrected beforing attempting correction of a PIP joint contracture
- The patient returns the next day for manipulation which is usually undertaken under LA for patients comfort
- Greater efficacy in MCP joint than PIP joint contractures

Fasciotomy

- Fasciotomy is indicated only for a well-defined pretendinous palmar cord causing an MCP joint contracture
- It is quick and can be performed under LA
- It may also be used in severe multiple digital contractures if the palmar skin is macerated to allow the fingers to be opened away from the palm
- There is a risk of digital nerve injury and recurrence
- This can be undertaken percutaneous with a needle

Limited fasciectomy

- This is the most widely performed surgical technique. The digital neurovascular bundles are identified proximally and traced distally
- Only the longitudinal contractile cords and nodules that are responsible for a joint contracture are excised
- Moerman's described a segmental fasciectomy[9]

Complete fasciectomy

- McIndoe popularized this technique using a transverse palmar incision to excise the entire palmar fascia
- The procedure has now been abandoned as it is impossible in practical terms to excise the entire palmar fascia, and attempting to do so does not necessarily prevent recurrence
- Secondly, the extensive dissection is a major assault on the hand, resulting in swelling, joint stiffness, haematoma formation and, possibly, skin necrosis

Dermofasciectomy

- This is indicated for recurrent disease with skin involvement and occasionally in the primary management of a digit in a young diathesis patients
- Involves excision of overlying skin and diseased tissue
- Proponents argue that diseased tissue is not isolated to the cord but involves the skin, which if left behind may cause recurrence
- The defect is replaced with a full-thickness skin graft from the forearm or groin (hair-free)
- The skin graft acts as a 'firebreak' to reduce recurrence
- Recurrence rate is 8% at 5.8 years follow-up[10]

Staged distraction and fasciotomy followed by fasciectomy

- First described by Messina, this is usually reserved for severe PIP joint involvement
- It is a two-stage procedure, which involves applying an external fixator to provide tension along the cord
- The DD regresses under stretch and then a planned fasciectomy is performed after a few weeks
- Not commonly undertaken in the UK

Amputation

- Consider for severe recurrent disease

Incisions

A good incision should provide well-vascularized skin flaps, extensile exposure and access for identification and preservation of the digital nerves and arteries.

Brunner

- Zigzag with apex made at the midaxial point of each finger flexor crease
- Allows excellent exposure laterally
- Raised skin flaps should be full-thickness and flap apex >60° angle to prevent tip necrosis

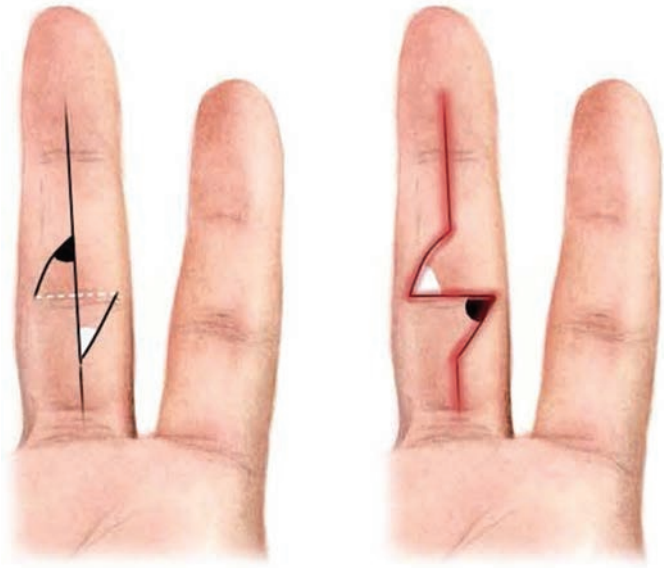

Figure 21.5 How to draw a Z-plasty. 1. Draw perpendicular (white dotted line) to longitudinal incision. 2. Mark flaps (white and black angles are at 60°). 3. Cross over flaps as shown

- Take care at the apex to avoid damage to the underlying neurovascular bundle

Straight longitudinal incision closed with Z-plasties

- Midline incision with Z-plasty of the flaps at closure (Figure 21.5). Be prepared to draw the Z-plasties if you mention this in the exam
- It has the advantage of lengthening the wound by up to 75%, which can help with skin coverage following correction of a large contracture

McCash open technique

- Transverse palmar incision with digital extension, either Brunner or Z-plasty, along the digit
- The wound provides excellent access to the diseased tissue and is left open at the end of the procedure
- This is a good technique in elderly patients, as it causes less oedema and haematoma formation, it is useful if you are short of skin, and it causes less pain and stiffness postoperatively; however, it takes a relatively long time to heal (6–8 weeks)

Oral question

- **Show me (on the examiner's hand) the incision you would make for a fasciectomy from the palm to middle phalanx?**
  - Brunner with apices out to the midaxial line.
- **Have you seen a Z-plasty? Why do we use them? Show me where you would make the flaps.**
- **What complications would you warn patients of before a limited fasciectomy?**
  - Scar tenderness, infection, bleeding, neuro-vascular damage, stiffness, CRPS, incomplete correction and recurrence
- **What would you do if you cut the digital nerve? How would you repair this? What suture would you use?**
  - Nerve should be repaired under magnification to prevent neuroma formation, and try and restore protective sensation. Requires a fine suture e.g. 9–0.
- **Describe the postoperative rehabilitation programme.**
  - Volar POP splint for 72 hours
  - Seen by the hand therapists for a wound check and apply a removable thermoplastic splint
  - Start ROM exercises
  - ROS at 10 days
  - Begin scar therapy at 2–3 weeks
  - Night splintage for 6 months.

PIP joint release (sequential)

- Prior to surgery it should be discussed with the patient about what degree of residual PIP joint contracture would be acceptable following removal of the disease
- A joint release can be undertaken which can intially obtain a straight finger. However, it requires more dissection and confers greater morbidity in terms of swelling, pain and stiffness. The patient typically takes longer to recover and the final result is often not the straight finger that was intially achieved
- The sequential approach involves releasing the following structures until an acceptable contracture is achieved
  - Release the check rein ligaments (proximal attachment of the volar plate that normally prevents hyperextension)
  - Release of accessory collateral ligament
  - Incise fibrous flexor sheath
  - Volar plate release (contentious)
  - Full collateral ligament release is not recommended
  - + transarticular K wire (4 weeks)
- A full release of the volar plate may cause excessive scarring and result in loss of flexion, which is more disabling than a flexion contracture

Postoperative management

Apply volar splint with digits extended for 2–4 days. Then actively mobilize and apply a night splint for up to 6 months.

Complications

Intraoperative

- Digital nerve injury (1.5%)
- Digital artery injury (actual incidence not known as there are good anastomoses to compensate if one artery is damaged)

- Overstretching of the finger and spasm in the digital arteries may lead to digital ischaemia. Positioning in flexion, patience and application of warm saline usually return the perfusion

Early postoperative

- Haematoma (release tourniquet before closure)
- Skin flap necrosis
- Small areas of skin loss will heal spontaneously, but healing of larger areas is slow and may result in scar contracture
- Finger loss – More common in revision surgery with prior damage to a unilateral digital vessel; hence, the need to assess sensation and perform a digital Allen's test preoperatively to identify potential hazards
- Infection

Late postoperative

- Stiff hand
- Loss of grip strength 6/52
- Complex regional pain syndrome type 1 (4% males, 8% females) – Rare but serious complication. Cardinal features are excessive pain, stiffness and vasomotor instability
- Inadequate release
- Scar-related problems
- Recurrence disease
- Flare reaction (combination of tenderness, shiny redness of the wound, swelling)

Examination corner

Hand oral 1: Clinical picture of severe DD affecting the little and ring finger

- **Who gets Dupuytren's disease?**
  - It occurs predominantly in males of northern European origin. It is associated with excessive alcohol intake, diabetes, chronic lung disease, chronic pulmonary tuberculosis and trauma most have a positive family history.
- **So what will you tell the patient about a limited fasciectomy procedure?**
  - I would tell the patient that surgery is under general anaesthetic or an axillary nerve block as a day case. Postoperatively their hand wrapped in a heavy dressing. This will be reduced at 48 hours and a smaller dressing applied. Sutures will be removed at 10 days and they will then be referred for physio. The surgery is not curative, and the disease can recur. I would mention that he has severe contractures of his PIP joint and we may not be able to get the deformity fully corrected. There is a small risk of a skin graft to cover any defect and this would be taken from the inner forearm. I would mention the risk of digital nerve injury and vascular injury that, in the worst-case scenario, may result in amputation.

Hand oral 2: clinical picture of a hand (Figure 21.6)

A pretendinous cord to the ring finger causing an MCP joint contracture.

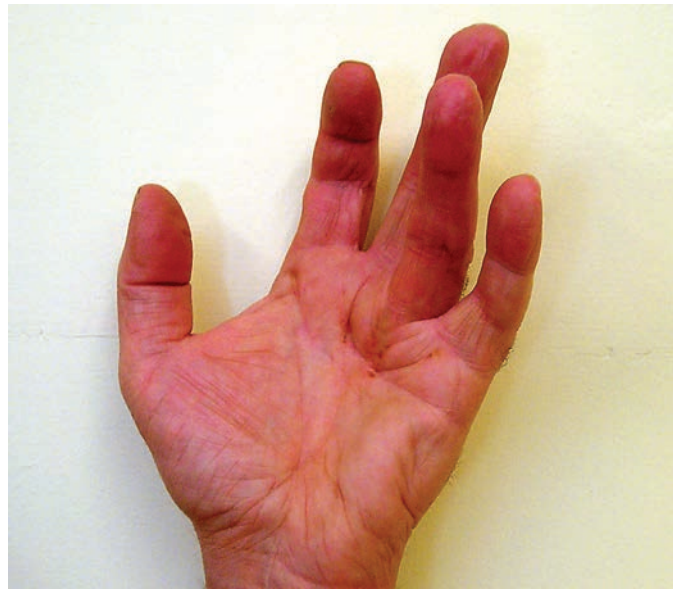

Figure 21.6 Clinical picture of Dupuytren's disease with a pretendinous cord to the ring finger causing an MCP joint contracture

- **What is the diagnosis?**
  - Dupuytren's disease.
- **How would you manage this patient?**
  - This would be based upon the patient's functional problems, expectations and medical well-being.
  - A pretendinous cord affecting solely the MCP joint will fully correct with surgery and so I will perform surgery when the patient has significant functional problems. This usually occurs with MCP joint contractures >30°.
- **Where would you start?**
  - In the palm where the nerve and vessels can be more easily and reliably found under Skoog's ligament. The nerve and vessels are then traced distally to allow safe excision of the disease.

Hand oral 3: Clinical picture severe DD affecting the PIP joint of the little and ring finger

- **What do you see here?**
  - This is a clinical picture, which shows a severe contraction of the PIP joint of the little and ring fingers. There appear to be cords present in the palm extending into the digits.
- **Is there anything that may guide you clinically as to how much correction can be obtained at surgery?**
  - It is important to access the PIP joint with the MCP joint fully extended as well as flexed. The PIP joint deformity often improves with MCP joint flexion by relaxing the MCP joint deformity and the intrinsic muscles. In addition, there is a high likelihood of midline displacement of the neurovascular bundle (spiral cord). I would estimate 1–1½ hours for the surgery when planning my list
- **What incision would you use?**
  - Brunner with the apices extending right out to the mid-axial line

Hand oral 4: Clinical picture of DD

- **Details on aetiology and presentation.**
  - Most are male of north European origin
  - Associated with several medical conditions
  - Most have a family history but as yet no clear gene has been identified
- **What tissue is involved?**
  - Skin and palmar fascia.
  - The palmar fascia is divided into three layers (talk into the detail given above).
- **What are the named cords?**
  - Pretendinous
  - Spiral
  - Natatory
  - Lateral
  - Abductor digiti minimi
  - Commissural (thumb web space).
- **Surgical indications?**
  - Functional problems
  - Generally MCP joint <30° and even less in PIP joint as less easy to achieve full correction.
- **When might you perform a dermofasciectomy?**
  - Extensive skin invovlement
  - Revision surgery
  - Some would consider this in a primary surgery for diathesis patients.
- **Revision options?**
  - Revision fasciectomy
  - Dermofasciectomy
  - Joint release
  - Arthrodesis
  - Amputation.

Kienböck's disease[15,16]

Most surgeons have radiographs showing Kienböck's disease and they often show them during the hand oral for the FRCS (Tr & Orth). This leads on to the usual questions about classification and treatment.

Introduction

There is an agreement that the histological and radiographic appearances are consistent with avascular necrosis of the lunate. First descibed in 1910 Kienböck felt 'this condition is due to a disturbance in the nutrition of the lunate caused by ruputre of the ligaments and blood vessels during contusion, sprain or subluxation'. It is typically unilateral although bilateral cases have been reported.

Aetiology

The aetiology of Kienböck's disease is unknown. There are several theories have been put forward:

- Trauma
- Mechanical overloading
- Metabolic abnormality
- Haematological disorder

There is no strong evidence to support any theory and it is, therefore, probably multifactorial. Many patients present with a history of trauma but over half of patients in one study could not recall any injury[15]. Others have suggested an occupational 'recurrent micro-trauma' but the evidence for this is poor.

Whilst the aetiology is unclear there are several well-established risk factors:

- Ulnar-negative variance (i.e. the ulna is shorter than the distal radius). It is thought that this leads to an abnormal loading of the lunate and has lead to the development of the surgical options
- NB. Ulnar variance can only be measured on a standard PA view of the wrist (shoulder flexed/internally rotated 90°, elbow flexed 90°, neutral forearm rotation)
- Shape of the lunate. There are three shapes and it is more common in type 1
  - Type 1 – Trapezoid
  - Type 2 – Rectangular
  - Type 3 – Square
- Blood supply (Gelberman). There are three patterns of supply. There is no definite association with a particular pattern, but it is thought that the I pattern have the greatest risk
  - Y pattern
  - X pattern
  - I pattern
- Male sex

Classification (Lichtman)

- **Stage I**
  - Plain radiographs are typically normal though a linear or compression fracture may be noted
  - MRI demonstartes diffuse T1 signal decrease
  - Positive bone scan
- **Stage II**
  - Sclerosis with or without multiple fracture lines
  - No collapse of the lunate
- **Stage IIIA**
  - Lunate collapse
  - Normal carpal height
- **Stage IIIB**
  - Lunate collapse
  - Capitate migrates proximally causing a reduction in carpal height
  - Scaphoid in a flexed position
- **Stage IV**
  - Radiocarpal and/or midcarpal arthritis

Clinical presentation

- Can be an incidental finding with the patient being asymptomatic

- Typically males aged between 20 and 40
- Insidious onset of central wrist pain with stiffness and weakness of grip strength
- Tenderness over the lunate and there maybe evidence of a radiocarpal effusion

Management

The surgical options are based on the aforementioned theories and none are proven to prevent progression of disease. Consequently surgical management is and indicated for patients who fail to respond to non-operative management in the form of rest, analgesia and splintage.

The severity of symptoms and radiological appearances do not correlate well. In planning treatment one needs to assess the patient's pain and disability thoroughly. The surgical options vary according to the stage of the disease. A wrist denervation incorporating both the anterior and posterior interosseous nerves just proximal to the wrist joint (as described by Berger) can be a useful adjunct in the surgical management regardless of the stage of the disease.

Stage I

- Simple immobilization to 'unload' the lunate and allow any revascularization to occur unimpeded
- Usually by use of a cast which is required for 3 months
- Progress can be followed on MRI
- If symptoms fail to respond can consider treatment outlined in stages II and IIIA

Stage II and IIIA

- Surgery aimed at trying to enourage direct or indirect revascularization
- Indirect is achieved by 'unloading' the lunate by shielding it from excessively high sheer stresses
  - In an ulna minus deformity, this is achieved by a radial shortening osteotomy to 'level' the joint (Figure 21.7)
  - In a neutral or ulna positive variance treatment is controversial. A capitate shortening can be undertaken to 'unload' the lunate. It has been shown to reduce the load on the lunate by 66% but increases the scaphotrapezial load by 150%
- Direct can be achieved through vascularized bone grafts
  - 2,3 – Intercompartmental supraretinacular artery (ICSRA), or 4,5 – Extensor compartment artery (ECA) are used
  - Technically demanding but studies have suggested satisfactory pain relief and improved range of movement

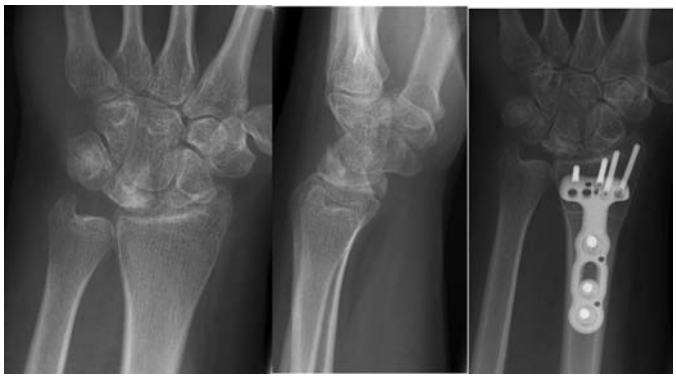

Figure 21.7 AP and lateral radiographs showing sclerotic and fragmented lunate (type IIIA), and ulnar minus. Treated by a joint levelling procedure (a radial shortening)

Stage IIIB

- The carpal instability resulting from the collapse is the focus of treatment
- This is managed by either a limited intercarpal arthrodesis or proximal row carpectomy
- Scaphocapitate or scaphotrapeziotrapezoid (STT) or fusion repositions the scaphoid into a neutral posture and re-establish carpal height. Load transfer subsequently occurs predominantly through the scpahoid rather than lunate fossa
- Proximal row carpectomy has shown no benefit to an STT fusion for stage IIIB disease, possibly because of pre-existing damage to the lunate fossa, which will articulate with the head of the capitate

Stage IV

- A salvage procedure in the form of a total wrist fusion is indicated

Examination corner

Hand oral: Radiographs demonstrating Kienböck's disease

- **A 42-year-old businessman comes to your clinic complaining of mild left wrist pain. These are his wrist x-rays. What do you see?**
  - This has the appearances of Kienböck's disease as shown by the marked sclerosis of the lunate. There are no osteophytes present and the lunate is not collapsed. The architecture of the wrist is well preserved. I note he is ulnar minus.
- **What do you think of his MRI scan?**
  - The MRI scans show decreased signal intensity consistent with the avascular necrosis of Kienböck's disease.
- **How do you classify Kienböck's disease?**
  - Kienböck's disease is usually diagnosed and staged on plain radiographs. Lichtman graded the disease into four stages radiographically.
  - Stage I has a normal lunate. Stage II sclerosis. Stage III shows collapse and fragmentation. It is subdivided into IIIA with no carpal collapse, and IIIB with carpal collapse (carpal index <0.54) and fixed scaphoid rotation. Stage IV is generalized arthritis of the wrist.
- **How do you manage Kienböck's disease?**
  - There is no correlation between staging and symptoms, and no conclusive evidence that surgery prevents progression of the disease.
  - I would treat this patient based on the level of symptoms.

- I would initially treat the patient non-operatviely
- If he did not settle I would offer him a joint levellling procedure with a radial shortening and plate fixation through a volar approach.

Hand oral 2: *Radiograph of Kienböck's disease*

- **What condition do you see here?**
  - Keinbocks
  - As evidenced by . . .
- **What is the natural history of this disease?**
  - Unclear
  - No conclusive evidence that people will progress through the stages
  - Pain does not correlate with the radiographic severity
- **Why is the aetiology?**
  - Unproven
  - Several theories (as listed above)
- **What is the classification and what stage is this (in the initial radiograph)?**
  - Lichtmann classification
- **What is the indication for surgery?**
  - Failure of non-operative management
- **What are the surgical options in this patient (clear ulna negative variance)?**
  - Joint levelling procedure
  - Revascularization
- **What if the joint was already level?**
  - Unload the lunate with a capitate shortening
  - Lunate revascularization

Ganglion[17]

Introduction

Ganglion cysts are the commonest soft-tissue swelling of the hand. It is not a true cyst as it is does not have an epithelial lining. It is a fibrous swelling usually attached to an underlying synovial cavity of a joint or tendon. It contains clear mucinous fluid, which is a mixture of glucosamine, albumin, globulin and hyaluronic acid. It is unclear if ganglion fluid is simply synovial fluid which has escaped from the joint, or if it is formed by cells in the synovium at the origin of the ganglion. They are most common in women and most occur between the second and fourth decades of life.

Pathology

- Remains unclear
- Theories of mucoid capsular degeneration and joint capsule synovial herniation have been proposed
- Consist of compressed collagen fibres with no true epithelial or synovial lining membrane
- Whilst some present following a history of trauma – There is no good evidence to support a traumatic or inflammatory aetiology

Most common sites

- Dorsal wrist ganglion (from scapholunate ligament)
- Volar radial ganglion (radioscaphoid or trapeziometacarpal joints)
- Flexor sheath ganglion (appears in the A1 or A2 pulleys)

Clinical presentation

- Concern over the presence of a lump
- Cosmesis
- Pain
- Variation in size, especially a reduction in size
- Wrist weakness
- Extrinsic compression of adjacent nerves (ulnar nerve Guyon's canal, median nerve in the carpal tunnel)

Management

It is very important to tailor the management to the patient and discover whether the problem to be addessed is concern, pain or cosmetic.

Non-operative management

- Reassurance – Whilst many patients know they have a ganglion a surprising number of patients have concern over the presence of a lump. Spend time reassuring the patient this is not and will not become a cancer. It is a harmless swelling which may resolve spontaneously in time. Useful to mention the Bible was used as an old-fashioned treatment method and that surgery would be exchanging a swelling for a scar, etc

Aspiration ± steroid injection

- May be effective in upto 30% of wrist ganglions
- Useful in confirming the diagnosis of a ganglion
- Can be helpful in the 'painful ganglion' because if the pain resolves on aspiration then excision should similarly relieve pain if the ganglion recurs

Surgical excision

- Excise for cosmesis, pain or functional disability. Recurrence rate 5%
- Reports of arthroscopic ganglion excision as yet have failed to prove superior results

Dorsal wrist ganglion

- Accounts for up to 70% of all ganglions
- Arises from the scapholunate ligament
- Occult ganglions may only be visible/palpable with wrist flexion though patient may only complain of pain in extension when the ganglion 'impinges' on the dorsal rim of the distal radius

- Transverse skin incisions are preferrable for cosmesis
- Success depends on identification of the pedicle connecting the ganglion to the joint and excision of the surrounding capsule
- The joint capsule should not be closed after surgical excision

Volar radial wrist ganglion

- Second most common ganglion accounting for up to 20%
- Two-thirds arise from the radiocarpal joint at the scapholunate interval
- One-third from the scaphotrapezial joint
- Ganglion lies under the volar crease between the FCR and APL
- An Allen's test before surgery is mandatory as the ganglion is often intimately adherent to the radial artery and requires careful dissection from it
- Inadequate collateral circulation from the ulnar artery may contravene surgery
- The recurrence rate is higher than for dorsal wrist ganglia

Flexor sheath ganglion

- Also known as a volar retinacular or seedling ganglia
- Third most common ganglion in the hand (~10%)
- Firm swelling felt at the base of the finger in the web space that can cause discomfort when gripping objects
- Small, firm, hard and tender mass ~2–5 mm in diameter
- Arises through the A1 or A2 pulleys and does not move with finger flexion, unlike the flexor tendon nodule seen with trigger finger
- Excise through a small Brunner's type incision with a margin of tendon sheath. Protect the neurovascular bundle
- Recurrence after excision is very uncommon

Mucous cyst

- A mucous cyst is a dorsal digital ganglion arising from an osteoarthritic DIP joint
- The cyst tends to lie to one side of the extensor tendon
- A dorsal osteophyte is commonly present and must be excised
- May present as ridging of the nail plate or recurrent infection with discharge
- Generally occurs between the fifth and seventh decade
- A transverse incision should be used to protect the germinal matrix of the nail bed
- A horseshoe ganglion may grow on either side of the extensor tendon
- The ganglion is mobilized and traced back to the joint with trimming of any dorsal osteophytes present
- Skin closure is either primary, a local rotation flap or a full-thickness skin graft

Examination corner

Short answers/notes

- Write short notes on the management of a 21-year-old woman who presents with wrist ganglia
- Confirm the diagnosis of a ganglion

History

- Usually painless swelling
- Examination
- Exclude worrisome features
- Transillumination

Management options

- Reassurance
- Especially if asymptomatic
- 'You haven't got cancer'
- Aspiration (±steroid injection) – High chance of recurrence
- Can be performed as an outpatient procedure and repeated if necessary
- Excision
- Indications would include a persistent, painful or enlarging ganglion
- Inform the patient that they would be substituting a scar for a swelling, that the scar may be painful, and that there may be an area of numbness around the scar
- Can be performed under either local or general anaesthetic
- Recurrence can occur in ~5%
- Trace the pedicle down to the joint capsule and excise part of the joint capsule
- Do not close the defect in the joint capsule

Hand oral: clinical photograph of either a dorsal and palmar wrist ganglion

- Spot diagnosis
- Usual questions about management

Hand oral: clinical photograph of a mucous cyst

- **What is the diagnosis?**
  - Mucous cyst
- **How would you treat this?**
  - Reassurance if that is all that is needed
  - Can attempt aspiration but high recurrence rate
  - Excision
- **You excise the cyst. How would you close the defect. Draw the rotation flap you would use to close.**

Osteoarthritis of the base of the thumb[18–25]

Background

Peritrapezial osteoarthritis commonly affects postmenopausal women, with 80% having radiographic changes although many have few symptoms.

Anatomy

- The thumb carpometacarpal (or trapeziometacarpal) joint is saddle-shaped and has little inherent stability but allows for a wide range of flexion–extension, abduction–adduction and rotation
- Opposition is a composite movement involving flexion and pronation
- Stability is predominantly provided by the three main ligaments stabilise the joint
  - Lateral ligament
  - Dorsal ligament
  - Volar-ulnar or beak ligament
- The beak ligament is the most important
  - It is extremely strong and is the primary static stabiliser of the joint
  - It is thought that degeneration of this ligament leads to joint instability and early disease

History

- Pain
  - Constant dull pain around the base of thumb and radial side of the wrist
  - Worse with use
  - Particularly aggravated with pinch or strong grip activities such as removing a tight jar lid
  - May affect MCP joint from compensatory hyperextension
  - Night pain is unusual
- Difficulties with ADLs (undoing screw top jars, doing up buttons, writing)
- Carpal tunnel symptoms

Examination

- Look
  - Squaring-off of the base of the thumb (shoulder sign – Dorsal subluxation of the thumb's metacarpal base)
  - Adduction contracture of the first web space
  - Thenar muscle wasting
  - Compensatory hyperextension of the MCP joint to increase span owing to the adduction contracture (Figure 21.8)
  - Look for trigger fingers and carpal tunnel syndrome (43% association)

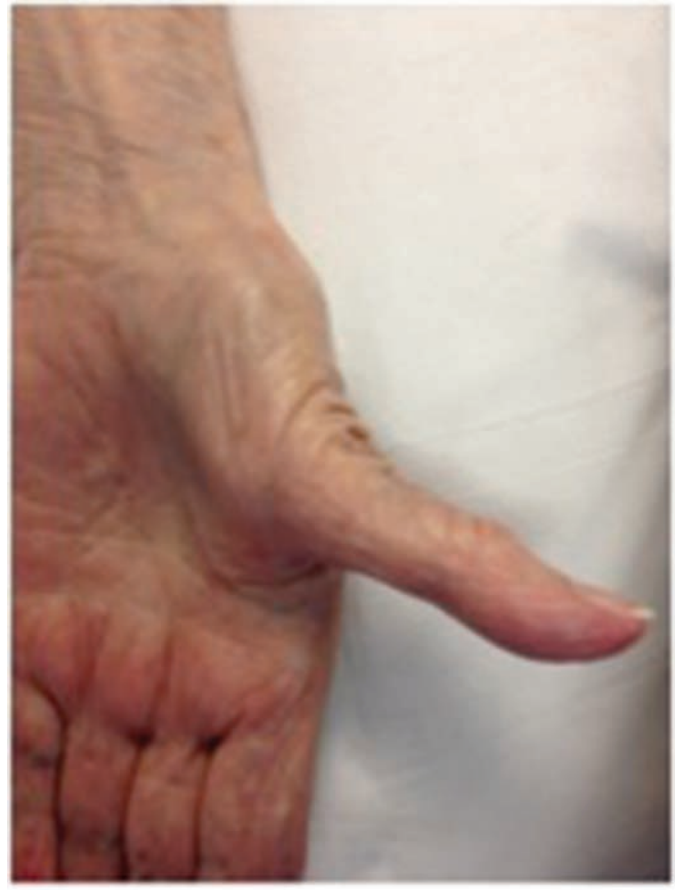

Figure 21.8 Typical basal thumb osteoarthritis. Note the shouldering of the CMC joint with a compensatory hyperextension of the MCP joint

- Special tests
  - Painful and unlikely to be performed in the exam
  - Grind test – Pain with axial loading of the thumb metacarpal and rotation of the CMC joint; positive if pain disappears with repeat test with distraction of the joint. May feel crepitus
  - Crank test – Axial loading of the thumb with passive flexion and extension of the metacarpal
  - A further test consists of longitudinal traction and pressure over the base of the thumb metacarpal to reduce the subluxed joint. Reproduction of pain strongly suggests disease at the thumb CMC joint

Radiographs

- AP hand and Robert's view (Figure 21.9 – True AP view of the thumb. Taken with the thumb fully abducted and the forearm fully pronated)
- Consideration of other diagnoses
  - Thumb CMC joint laxity
  - De Quervain's
  - Trigger Thumb
  - Scaphoid non-union

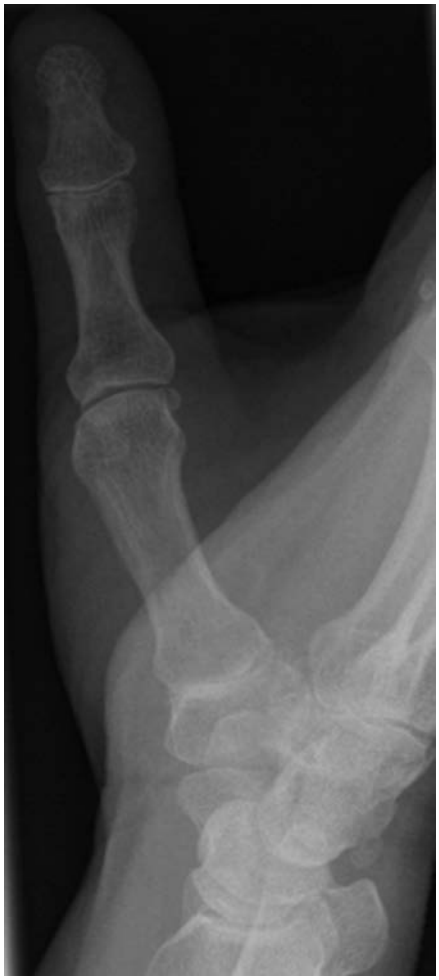

Figure 21.9 Robert's view of thumb CMC joint

- SNAC/SLAC wrist
- Kienböck's

Classification (Eaton and Littler)

- Radiological classification for staging thumb CMC joint OA
- Corresponds poorly to clinical symptoms
- Poor interobsevor corroboration

Stage 1

- Radiographs demonstrate widening of the joint space
- Synovitis and joint effusion
- Pre-arthritis stage

Stage 2

- Slight narrowing of the joint space
- Mild subluxation
- STT joint normal

Stage 3

- Joint space markedly narrowed
- Often sclerotic and cystic change
- Moderate subluxation

Stage 4

- Pantrapezial arthritis
- Severe subluxation
- Joint space is narrow, cystic and sclerotic subchondral bone changes

Management

Thumb CMCJ OA is extremely common and a significant proportion of patients can be managed without surgery.

Non-operative

- A trial of non-operative treatment should be tried in all patients regardless of the severity of the radiographic changes
- Options
  - Activity modification
  - NSAIDs
  - Intra-articular steroid/local anaesthetic injections
    - One RCT found no benefit of steroid over saline[23]
    - A different study showed 59% were satisfied and only 28% had required surgery at 3 years[24]
  - Thumb splinting – Can offload degenerate parts of the joint
  - Physiotherapy
- In reality a combination of these options should be tried
- One prospective study showed 70% of patients listed for surgery no longer required this following 7 months of therapy, splints and analgesia
- No evidence for the use

Surgery

The indications for surgery are disabling symptoms unresponsive to conservative treatment.

Early stages of the disease (stage 1)

- If instability is present an Eaton–Littler procedure is performed (soft-tissue reconstruction of the beak ligament using half of FCR passed through a hole drilled across the base of the thumb metacarpal). Contraindicated if degenerative changes are present
- More recently an extra-articular 30° extension osteotomy of the thumb has been described as an alternative method of management
- In reality few patients will present at this stage

Late stages of the disease

There are numerous surgical treatment options for OA of the base of the thumb (Table 21.1).

Table 21.1 Surgical treatment options for osteoarthritis of the trapeziometacarpal joint

Trapeziometacarpal arthroplasty without excision of the trapezium
Trapeziectomy
± Ligament reconstruction and tendon interposition
± Soft-tissue interposition
Osteotomy
Arthrodesis
Joint replacement arthroplasty

The choice depends on three main factors:

- Whether there is isolated CMC joint disease or pantrapezial disease
- Patient's activity level
- Surgeon's experience

Procedures that preserve the trapezium or aim to maintain thumb length will theoretically preserve function. However, loss of trapezial height has not been shown to correlate with thumb strength postoperatively[18].

1. Trapeziectomy
   - Generally provides reliable pain relief but may be accompanied by thumb weakness
   - It is not a technically demanding procedure but there is protracted rehabilitation time (6 months)
   - Requires 4–6 weeks in a thumb splint postoperatively
   - Instability of the base of the thumb metacarpal is a possible complication
   - Numerous modifications to simple excision have been devised to try to prevent this (haematoma distraction, APL sling)
2. Excision plus ligament reconstruction and tendon interposition (LRTI)
   - More popular in USA than Europe
   - Time-consuming and there is no proven benefit over simple trapeziectomy[20]
   - Ligament reconstruction using FCR is done to support the base of the first metacarpal and to prevent thumb shortening, and the remaining tendon is rolled up to act as a spacer
3. Arthrodesis
   - Possibly indicated for younger, manual workers with isolated trapezometacarpal disease to maintain better grip strength
   - This is a technically more difficult procedure, with a higher incidence of serious complications[22]
   - It places increased demands on the triscaphe joint, which may become painful
   - The joint is fused in a clenched fist position (10–20° radial and 30–40° palmar abduction). In reality this is where the thumb tip lines up with the index fingetip for a pinch
   - The arthrodesis can be stabilised with an AO 2.4 mm compact hand set T-plate, K-wires or a tension-band wire
4. Joint replacement arthroplasty: Both cemented and uncemented designs
   - Total joint replacements generally have a constrained ball and socket design with the stemmed ball inserted into the metacarpal and the socket anchored to the trapezium
   - This is gaining in popularity despite only short-term results to date in a small series
   - Currently no evidence that it is superior to trapeziectomy and it is not in common usage
5. Osteotomy: A number of osteotomies have been described at the base of the first metacarpal
   - Good results have been reported with an abduction–extension osteotomy for stage II and early stage III disease
   - It is suggested as a more durable procedure than an arthroplasty and restricts motion less than an arthroplasty, but has not gained widespread popularity

- Despite the wide variety of surgical options exist and none is clearly superior. Overall, expect 80–90% good results whatever procedure is used
- Therefore, length of surgery and rehabilitation time are important. Patients should be warned that several months might be needed to gain the full benefit from the procedure
- A recent publication by Gangopadhyay showed the results at 5–18 years for simple trapeziectomy, trapeziecotmy with tendon interposition and trapeziecotmy with LRTI[19]. There was no difference in terms of pain relief, grip or pinch strength, range of motion or complication
- A Cochrane review in 2009[22] found marginally better pain relief with LRTI but an amount that could have happened by chance. They did report a significantly greater number of complications with LRTI

Approach

- Dorsal
  - Longitudinal incision over the anatomical snuffbox
  - Take care not to damage the branches of the superficial radial nerve
  - The dissection is taken down on to the capsule between EPB and EPL
  - The radial artery crosses the floor of the anatomical snuffbox and has to be carefully mobilized dorsally
  - A longitudinal capsular incision is then made before subperiosteal dissection of the trapezium, which can be removed whole or piecemeal

- Modified Wagner
  - Curved incision between glabrous and non-glabrous skin
  - Elevate the thenar muscles of the trapezium
  - Less likely to encounter bracnhes of the radial nerve or the radial artery

MCP joint hyperextension[25]

- This may disappear with correction of the adduction contracture at the thumb metacarpal
- Some advocate treatment with hyperextension of >30°
  - Either a volar capsulodesis if the joint surfaces are intact
  - Or a fusion if there are painful degenerative changes
- Others choose to treat the CMC joint and evaluate the MCP joint following rehabilitation

Examination corner

Hand oral 1: Clinical photograph of hand demonstrating shoulder sign

- **What do you see here? What is the diagnosis?**
  - Thumb held in an adducted position
  - Shouldering of the base of thumb in keeping with OA
- **What problems do the patients usually have?**
  - Common joint to get arthritis
  - Not always symptomatic
  - Symptoms are worse on pinch and twisting activites of the thumb such as lifting a kettle or twisting a door knob or using a knife
- **How would you manage the patient intially?**
  - Activity modification
  - Splints
  - Steroid injection
- **How would you manage this patient surgically?**
  - Trapeziectomy
  - (For you the candiate to decide but need to justify why – Probably easier to do a simple trapeziectomy)
- **Would you perform LRTI?**
  - (Your decision as the candidate)
  - No I would not. There is no clear evidence that it is beneficial, is time consuming and can lead to increased complications
- **What literature can you quote to support your surgical approach?**
  - Cochrane review

Hand oral 2: a patient is listed for a trapeziectomy

- **Which approach would you use**
  - Dorsal or modified Wagner
  - (Increased rate of superficial radial nerve irritation with dorsal approach)
- **Show on the examiner where you would make your surgical incision**
  - Clearly demonstrate landmarks and the incision line
- **What are the structures at risk?**
  - Superficial radial nerve
  - Radial artery
  - FCR
- **How do you know the bone you are about to excise the trapezium and not the scaphoid?**
  - Saddle shape trapezium but get an x-ray if any doubt before you excise
- **Patient returns to your clinic 1 year later and an x-ray shows the thumb metacarpal is now articulating with the scaphoid**
- **What would you do now?**
  - Depends on symptoms.
  - Migration of the thumb proximally does not always denote pain and functional problems
- **What if they have symptoms?**
  - Assess the cause of the pain – Remaining osteophytes, ST arthrosis or arthrosis between the thumb metacarpal and the scaphoid
  - No proven treatment – Consider steroid injections, splints, stabilisation procedure or even a silastic implant

Small joint arthritis

History for this should cover any skin, eye or bowel problems.

Osteoarthritis

- Involves base of thumb and DIP joints mainly
- Heberden's (DIPJ) and Bouchard's (PIPJ) nodes are painful dorsal osteophytes

Systemic lupus erythematosus (SLE)

- Chronic inflammatory disorder with joint involvement in 75%. Malar rash, fever, pericarditis. In the hand there is a similar deformity to rheumatoid deformity with joint subluxations and dislocations but normal joint spaces and no erosions. Soft-tissue procedures are unsuccessful, and require arthrodesis (or arthroplasty)

Gout

- Urate crystal deposition from various causes (idiopathic, thiazide diuretics, renal failure, malignancy). Causes acutely inflamed joints and characteristic punched-out lesions. Gouty tophi and kidney stones may occur. Crystals are negatively birefringent (yellow) on polarized light microscopy

Psoriasis

- Arthritis present in 20% of those with psoriasis. HLA-B27 in 50%. Other manifestations include extensor and scalp plaques, and nail pitting. In the hand, there is asymmetric arthritis with marked deformity ('pencil in cup')

Haemochromatosis

- With osteoarthritic changes to the metacarpal heads this should be considered as a rare cause and a full blood count performed

Rheumatoid arthritis of the wrist and hand[26–32]

Introduction

- The most common inflammatory arthritis affecting 0.5–1.0% of the population
- Women are three times more often affected than men
- A chronic progressive symmetrical polyarthropathy and systemic disease of unknown aetiology
- Rheumatoid factor (IgM autoantibody to IgG) is present in 80%
- The main structures requiring treatment are painful, arthritic joints, tendon rupture and subluxation, and nerve compression
- The continuing improvement in the medical management of this disease has markedly reduced the number of patients requiring surgery
- Management of these patients should be with a multi-disciplinary approach involving rheumatologists, hand therapists, hand surgeons and the patient

Pathophysiology

- Chronic inflammatory autoimmune process that causes joint inflammation, cartilage destruction and ligament wekaness
- The formation of pannus, caused by synovial inflammation, in areas or increased vascularity. The pannus invades into terminal vessels resulting in soft-tissue ischaemia and stretching of tissues
- Activated neutrophils from the pannus release lysosomal enzymes and free radicals that destroy the articular surfaces
- Volar subluxation and supination of the carpus occurs due to laxity of the strong extrinsic volar carpal ligaments. This, with distal radioulnar joint disease and ulnar subluxation of ECU, leads to prominence of the ulnar head (caput ulnae)
- There is radial deviation of the metacarpals, altering the line of pull of the EDC tendons. This, combined with capsular laxity from synovitis, leads to volar–ulnar subluxation at the MCP joints
- Tightness of the ulnar intrinsics causes imbalance of the digits, leading to swan-neck and boutonnière deformities
- Rheumatoid nodules are present in 25% and consist of a collagen capsule, and fibrous and central necrosis if large

History

- Pain
- Weakness
- Loss of function (it should be noted that, despite advanced disease, patients maintain an excellent level of function)[24]
- Swelling
- Cosmetic deformity
- Difficulty in activities of daily living and hobbies

Clinical

Pancarpal disease

- Synovial proliferation and inflammation involves the whole wrist joint, causing pain, stiffness and swelling
- The inflammatory synovitis causes ligament laxity and destruction of articular cartilage, and invades bone, causing cyst formation and bone destruction
- The end stage is either spontaneous fusion of the wrist joint or palmar dislocation and ulnar translocation of the radiocarpal articulation

Periscaphoid disease

- Synovitis disrupts the radiocarpal and intercarpal ligaments, leading to rotatory instability of the scaphoid and carpal instability (DISI pattern)
- The intercarpal ligaments and wrist capsule become stretched and weakened
- The scaphoid assumes a flexed position, leading to loss of carpal height, the carpus drifts into radial deviation and there is volar subluxation of the radiocarpal joint
- The carpus ultimately dislocates in a volar and ulnar direction. Power grip is weak; the wrist is no longer stable

Distal radioulnar joint instability

- The ulnar subluxates dorsally (caput ulnae syndrome)
- Prominence of the ulna gives rise to the piano key sign owing to destruction of the TFCC

MCP joint

- Volar–ulnar subluxation
- Synovitis causes capsular laxity
- Compensatory ulnar deviation at MCP joints from longitudinal pull of extensor tendons with radial deviation of metacarpals
- Ulnar intrinsics then shorten

Extensor tenosynovitis

- Attrition over the prominent ulnar head causes extensor tendon ruptures initially affecting the little finger (Vaughan–Jackson syndrome)
- The EPL can rupture around Lister's tubercle

Flexor tenosynovitis

- Pain and volar swelling
- The FPL can rupture in the carpal tunnel from synovitis of osteophytes over the scaphotrapezial joint
- An anterior interosseous nerve syndrome is the differential

Carpal tunnel syndrome

- Secondary effect of swelling at the wrist .joint

Assessment

- Looking is the most important part as these patients commonly have marked pain
- Quickly screen neck, shoulder and elbow movements. Place hands on a pillow
- Swellings – Nodules, MC heads, caput ulnae
- Obvious deformity – Subluxed ulnar head/carpus, deviation metacarpals, Z-deformity of the thumb, swan-neck/boutonnière, dropped fingers
- Scars (three most common rheumatoid patient scars: Dorsal midline from wrist arthrodesis, transverse over MC heads from MCP joint arthroplasty, and longitudinal over thumb from MCP joint fusion)
- Muscle wasting
- Feel any obvious swellings over joints for synovitis and along subcutaneous border of ulna for nodules
- Active movement – Forearm rotation for DRUJ (loss of supination as the ulnar head is subluxed dorsally), prayer position for wrist extension, back of hands together for wrist flexion, global screening finger movements – 'can you make a fist then straighten out your fingers?'
- Functional assessment – Different grips
  - Power – 'squeeze my fingers'
  - Tripod – 'hold a pen'
  - Key – Pulp to pulp
  - Precision – Tip to tip, 'pick up a coin'

Radiographs

- PA and lateral wrist radiographs plus PA of the whole hand to assess the severity of arthritis throughout the hand
- Rheumatoid wrist disease can be staged using either the Larsen (stages 0–5) or Wrightington classification (grades 1–4)

Management

Medical[26,27]

- In addition to anti-inflammatories and steroids, there are disease-modifying anti-rheumatioid drugs (DMARDs)
- DMARDs can reduce the extent to which rheumatoid arthritis damage bones and cartilage, decreasing disease impact and disability
- Early diagnosis of rheumatoid arthritis is vital as DMARDs have the greatest effect when started early in the disease
- Traditional DMARDs consist of methotrexate, hydroxychloroquine, sulfasalazine and azathioprine
- These can be used individually or in combination and can often require up to 6 months to have full effect
- Newer DMARDS include TNF-α (e.g. Etanercept, Infliximab) and recombinant monoclonal antibodies such as Rituximab. These newer drugs are generally well tolerated and can prove efficacious as early as 2 weeks
- Side effects such as infeciton, hepato-toxicity, vision loss and pancytopenia have all been reported
- Medication and surgery
  - Methotrexate was previously thought to increase the risk of postoperative infections and surgical complications during elective surgery
  - Suddenly stopping the drug often results in a flare-up of the disease, making movement painful and rehabilitation difficult
  - The continuation of methotrexate treatment does not increase the risk either of infections or early surgical complications in rheumatoid patients and so it should be continued[28]
  - TNF medications such as etanercept that should be stopped prior to elective surgery
  - Rituximab may be stopped prior to surgery but speak to your rheumatologist

Planning treatment

- Surgery is indicated for pain, deformity and loss of function
- Treatment should be individualized based on the type and severity of the local destructive process, the involvement of other joints in the upper and lower extremities, the overall status of the patient's disease and the patient's background and expectations from surgery
- In the rheumatoid patient, lower limb problems should in general be treated before upper limbs. Retention of walking ability is of overriding importance and periods of crutch walking following lower limb surgery are best avoided after reconstructive procedures on the upper limbs
- More proximal joints (shoulder and elbow) should be treated before distal joints (wrist and hand) – The hand has to be positioned to carry out appropriate tasks that require good function at the shoulder and elbow
- Urgent procedures – In the hand there are some procedures that should be carried out urgently. These include tenosynovectomy to prevent tendon ruptures or to release nerve compression

Operative management

The most common rheumatoid hand operations – Wrist fusion, finger MCP joint replacements, fusion thumb MCP joint.

The wrist

- Commonly affected – Up to 90% of patients at 10 years
- May manifest itself at the radiocarpal, midcarpal, DRUJ or any combination of these

DRUJ

- Commonly affected early in the disease process with one-third developing caput ulnae
- The extensor tendons can, thus, be threatened along with the pain and loss of function from the arthritic DRUJ
- A synovectomy can be performed (rarely) in the presence of synovitis without arthritis
- Most surgeons would look rely on bony procedures to deal with a symptomatic DRUJ
- The options include – distal ulna resection, Suave–Kapandji and ulna head arthroplasty
- Distal ulna resection
  - Popularized by Darrach
  - Generally indicated for older sedentary patients
  - Dorsal approach through the floor of the fifth extensor compartment leaving a cuff of capsule on the radius for the repair
  - Osteotomy through the metaphysis conserving as much length as possible
  - Main complaint is painful impingement of the ulnar stump on the radius
  - Any pre-existing ulnar carpal translation is a contraindication as this may progress
- Suave–Kapandji
  - Fuses the DRUJ with excision of a section of ulna proximal to the DRUJ
  - Has the advantage of stabilising the ulnar side of the carpus and so preventing ulnar translocation that can occur with resection
  - However, stump instability remains a complication along with DRUJ non-union
- Arthroplasty – ulnar head replacement or DRUJ arthroplasty
  - Not commonly used
  - Typically used as a salvage procedure for failed resection or Suave–Kapandji
  - The ulnar head replacement requires good soft tissue for stability and so is not suitable in a significant proportion of RA patients

Partial arthrodesis

- Not rarely undertaken
- Useful in patients with isolated radiocarpal, midcarpal or to stabilise the wrist in ulnar translocation

Wrist arthrodesis

- A safe and reliable option that provides predictable pain relief (NB. Rule out DRUJ pain)
- Can be achieved with a wrist fusion plate, intramedullary pin (from third MCP joint through carpus and into distal radius) or K-wires
- The position of fusion is debatable but most surgeons prefer slight extension and ulnar deviation
- It is performed through a straight longitudinal dorsal skin incision and the dorsal halves of the carpal bones and the distal radius are fragmented with bone nibblers. The bone fragments are then packed into the wrist joint, which is stabilised with a Steinmann or Stanley intramedullary pin. This is normally inserted through the head of the third metacarpal and passed down the metacarpal shaft across the wrist joint and into the distal radius. Alternatively, damage to the MCP joint can be avoided by introducing the pin through the bases of the second and third metacarpals, but this gives less secure fixation
- In general, plate fixation is avoided in the rheumatoid patient. The porotic bone does not take screws well and there are concerns about wound healing
- Wrist arthrodesis is a good surgical procedure and remains the gold standard for salvage of the advanced RA wrist
- There is a high rate of fusion, with few operative or postoperative complications
- If a pseudoarthrosis develops it is rarely symptomatic
- If bilateral wrist fusion is performed, the dominant wrist should be fused in slight flexion to facilitate perineal care
- Ideally, both wrists should not be fused; if possible, one should be replaced to allow retention of some movement

Wrist arthroplasty

- The potential advantage over arthrodesis is preservation of motion
- The aim is for active wrist motion with an arc of movement ~30–40°
- Prerequisites for a wrist arthroplasty include good bone stock; the deformity must not be too severe (contraindicated if the wrist joint is subluxed or dislocated) and extensor tendons must be functional. A relative contraindication is previous sepsis
- A retrospective review by Murphy demonstrated similar complication rates between arthrodesis and arthroplasty but improved ability to undertake ADLs in the arthroplasty group[32]
- Patients who have had an arthrodesis on one side and an arthoplasty on the other side typically prefer the arthoplasty side
- Prostheses are inserted through a dorsal approach. It is generally only appropriate in the very low demand patient with a well-balanced wrist
- Salvage of failed arthroplasties remains difficult because of loss of bone stock. It typically requires conversion to an arthrodesis with a plate and bone grafting. The conversion arthrodesis confers significantly greater complications than primary fusion

MCP joint replacement

- This is the most commonly affected joint in RA
- Classic deformity of volar subluxation, flexion and ulnar deviation
- Synovitis weakens the radial collateral ligament and radial sagittal band leading to the ulnar extensor tendon subluxation and ulnar deviation of the digit
- Replacement aims to relieve pain and realign the joint
- Silastic replacements (Swanson® and NeuFlex®) are most commonly used
- Approach
  - Transverse or longitudinal skin incisions
  - Protect dorsal veins
  - Longitudinal capsulotomies to radial side of extensor tendons
  - Capsule and ulnar intrinsics released
  - Cut metacarpal heads just distal to the collateral ligaments
  - Ream and insert the implants
  - At closure, suture lax radial capsular flap under the extensor tendon to the ulnar capsule, to correct radial deviation
  - This can be carried out with or without intrinsic transfer
- Unconstrained pyrolytic implants are in use – But it is difficult to get good soft-tissue control in RA patients

PIPJ

- This can be treated with arthrodesis or arthroplasty (with a Silastic implant)
- Index finger is often more suited to arthrodesis as its role is primarily for pinching activities with the thumb (conferring a sideways force), whereas the ring and little fingers have a greater role in grip strength and so preservation of PIPJ motion as preferrable when possible
- Fusion is undertaken usually through a dorsal approach with the joint fused in flexion. The degree of flexion should reflect patient occupation, hobbies, normal cascade and cosmetic appearance. The fusion can be achieved with K-wires, cerclage wire or a headless compression screw
- Arthroplasty is mainly reserved for the older, less active patients though some surgeons are using the implant in younger patients to maintain function (accepting the risks of implant failure)
- Patients with pre-existing boutonnière or swan-neck deformities are associated with poorer results with arthroplasty and so arthrodesis may be preferrable

Examination corner

Hand oral 1: AP radiograph of a severely deformed rheumatoid wrist

- Description of radiographs
- Principles of management
  - Be careful when mentioning synovectomy in this particular instance – The bony changes are too advanced to preclude any useful benefit from synovectomy but, if the wrist was less severely affected, synovectomy may be useful
  - Discussion on the relative merits of arthrodesis vs arthroplasty

Hand oral 2

- Rheumatoid hand with caput ulnae
- **How would you manage this patient?**
  - All RA management is about managing the patient's pain and function not the appearance
  - The only exception is in cases where there may be a risk in leaving the condition such as persistent synovitis and caput ulnae can lead to tendon rupture
- Picture showing Vaughan–Jackson syndrome
- **What is the differential diagnosis and how would elicit which the diagnosis is?**
  - Drop fingers can be a result of PIN palsy, tendon rupture and extensor tendon subluxation ulnarwards
  - PIN palsy should affect all four fingers and thumb, and the tenodesis test will be intact. In tendon rupture, the tenodesis test will be positive. In sagittal band rupture, finger extension can be maintained the finger is extended passively (this relocated the extensor tendon over the metacarpal head)
- **How would you manage the drop fingers – If 2 affected? if 3? if 4?**
  - Firstly prevent further tendon rupture. Carry out a Darroch's procedure.
  - Tendons
- Just little – Buddy onto the ring finger
- Ring and little – Either buddy both to the middle or transfer EI to little and buddy ring to middle
- Middle, ring and little –EI to ring and little, with middle buddied to index
- FCR or FDS transfer if all digits affected

Adult and pathology oral 2: clinical photograph (Figure 21.10)
Clinical picture of rheumatoid hands.

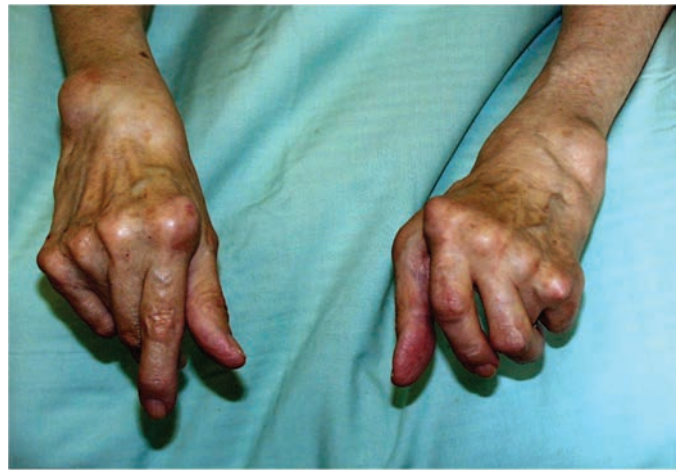

Figure 21.10 Clinical picture of rheumatoid hands

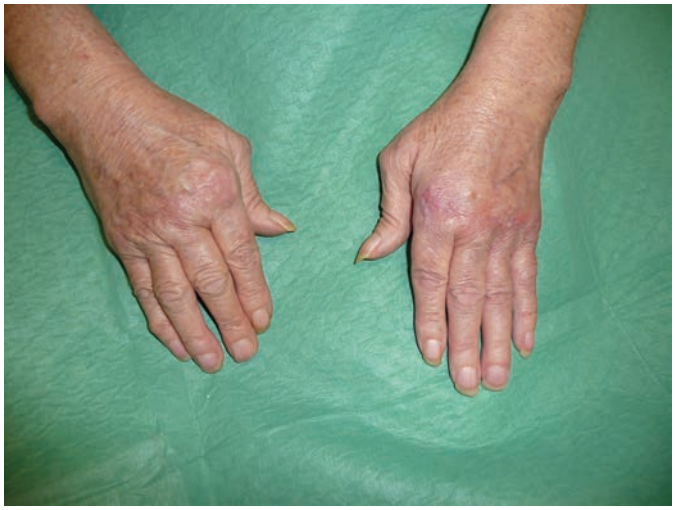

Figure 21.11 Clinical picture of hands of a rheumatoid patient

- **Describe these hands**
  - Always be systematic when describing any picture.
  - Start with deformity at wrist then digits.
- **Discussion on treatment of painful hands.**
  - Stepwise approach like any pain.
  - Analgesics
  - Splints
  - Steroid injections
  - Then surgically into arthrodesis or arthroplasty

Hand oral 3: clinical picture (Figure 21.11)
Clinical picture of hands of a rheumatoid patient.

- **Describe what you see.**
- **How do you do this operation (MCP joint replacements)?**
  - Either a single longitudinal incision to each digit or a single transverse incision over the MCP joints if doing multiple digits.
  - Separate the extensor from underlying capsule
  - Divide junctura if they are adding to the deformity
  - Release the ulna intrinsics and sagittal bands
  - Capsulotomy on the radial side (can be reefed on the way out to tighten the radial side)
  - Release of the ulnar collateral origin (unless minimal deformity)
  - Assess for bone loss
  - Metacarpal head resection
  - Thin layer of proximal phalanx resection
  - Preparation with awl and then broaches each side.
  - Trial (aim for ability to slightly hyperextend the MCP joint)
  - Closure 2–0 PDS.
  - Realign the extensor.
- **What is the chance of recurrence of the deformity?**
  - Most will gradually redevelop an ulnar drift but not to the extent of preop.

Flexor and extensor tendons

- Tenosynovitis in the rheumatoid hand is more obvious on the extensor surface of the wrist and hand but one should not neglect also to examine the flexor side of the hand and wrist for its presence

Symptoms

- If flexor tenosynovitis is present in the **carpal tunnel** it can cause
  - Carpal tunnel syndrome
  - Tendon rupture

Palm and fingers

- Triggering
- Loss of active finger flexion or passive finger extension

The consequences of flexor tenosynovitis are pain, stiffness (restricted active motion) and tendon rupture. Inevitably, flexor tenosynovitis can coexist with any related joint problems in the hand.

Examination

Examination for flexor tenosynovitis can be difficult as swelling is sometimes minimal, but restriction of active movement and crepitus as bulky tendons move beneath pulleys are common.

Look

- Overall posture of the hand
- For evidence of tendon ruptures, isolated swan-neck deformities (isolated rupture of FDS)
- Swelling is seldom visible (or palpable) beneath the flexor retinaculum, but appears in the palm, distal forearm and digits

Feel

- Puffy thick feel to the rheumatoid hand
- Pinch test
  - Normally you can pinch between your finger and thumb two thicknesses of skin in front of the proximal phalanx. Thickened tenosynovium bulges out through defects in the fibrous sheath and creates a wedge of tissue instead
  - A thickened sensation around the distal palmar crease area at the entrance to the A1 pulley may indicate the presence of synovitis
- Palpation of the fingers may indicate the presence of nodules or diffuse synovitis

Move

- Examination of tendon function for both FDS/FDP
- Crepitus over the tendons

Inability to flex the DIP joint

- Tendon rupture
- Adherence of FDP to FDS
- Triggering caused by nodules

Tendon rupture results from invasive synovitis, infarction secondary to vasculitis, attrition from bony prominences and pressure under the unyielding extensor or flexor retinaculum.

Management

Acute synovitis

Conservative

- Splintage and drugs
- Steroid injections into the carpal tunnel or tendon sheath (explain small risk of tendon rupture)

Surgery

- Surgery is indicated for
  - Failure of conservative treatment at 4 months and the presence of persistent and painful tenosynovitis
  - Median nerve compression in the carpal tunnel
  - Triggering
  - Tendon rupture

Timely tenosynovectomy is vital in preventing tendon rupture and preserving the function of the hand. When there is doubt it is better to perform a tenosynovectomy to prevent tendon rupture than to persist with medical treatment. The surgeon should adopt an aggressive approach towards rheumatoid tenosynovitis and be prepared to intervene surgically on a prophylactic basis.

Chronic synovitis

Synovectomy

There are three sites

- Carpal tunnel (floor of the carpal tunnel is inspected for bony spicules, which are excised if present)
- The palm at the level of the mouth of the A1 pulley
- Fingers at the level of the PIP joint just distal to the A2 pulley

Make a Brunner's incision. Remove diseased synovium and intertendinous nodules, and repair any tendon defects. Release of the A1 pulley in rheumatoid arthritis is controversial as it may allow ulnar migration of the flexor tendons and aggravate ulnar drift deformity at the MCP joint. The annular pulleys should be preserved (including the A1 pulley) and the tendon sheath is opened between the annular pulleys. Postoperative stiffness can be a problem and early mobility is essential.

Tendon rupture

- Primary tendon repair – Primary repair is generally not possible owing to poor tissue at the tendon ends
- Primary tendon graft – Fraught with difficulties and results are usually poor; in a young patient this should at least be considered
- Tendon transfer – Limited availability on flexor side (FDS ring finger, palmaris longus, brachioradialis)
- Side-to-side suture – Good in older patients, should be considered for ruptures at the wrist level
- Arthrodesis (DIPJ – Ruptured FDP but intact FDS)

Table 21.2 Options for reconstruction

Tendon ruptured	Salvage procedure
FDS	None
FDP – Wrist	Suture to adjacent FDP tendon or tendon graft
FDP – Finger	DIP joint fusion
FDP + FDS finger	Tendon graft or FDS transfer from another finger
FPL	Synovectomy and fusion IPJ

Full synovectomy should be performed simultaneously with any tendon reconstruction (Table 21.2), as re-ruptures are not uncommon.

Loss of both tendons within the digital sheath is disabling but reconstruction is difficult. Transfer of the FDS from another finger can be used if a healthy distal FDP stump is present, otherwise tendon grafting may be necessary despite its unpredictable outcome.

Vaughan–Jackson syndrome

Rupture of EDC of ring and little fingers caused by attrition from prominent ulna head (caput ulnae) and DRUJ synovitis.

Differential diagnosis of dropped fingers

- Ulnar subluxation of extensor tendons
- Volar subluxation of MCP joints
- PIN palsy
- Locked trigger finger

Management

- Synovectomy ± Darrach's for pre-rupture
- Tendon transfer
  - Little finger – EI to EDM transfer
  - Ring finger – Buddy ring EDC to middle EDC

Mannerfelt–Norman syndrome

The most common flexor tendon to rupture in rheumatoid arthritis is FPL rupture, caused by scaphotrapezial synovitis.

Management

- Prompt exploration of the carpal canal and removal of diseased synovium and osteophytes (scaphotrapezial joint)
- IPJ arthrodesis preferred option – Gives a stable thumb with good power transmitted from the short muscles
- Soft-tissue reconstruction
  - Transfer FDS of the ring finger – A possible option but the range of motion gained is small. It is difficult to get the tension right
  - A free tendon graft can be used to bridge the gap

Oral question

Describe the typical manifestations of rheumatoid disease at the hand and wrist.

Rheumatoid thumb

Introduction

More than two-thirds of rheumatoid patients have some involvement of the thumb. All three joints can be affected. Thumb deformities caused by RA can markedly impair function by limiting pinch, grip and motion.

Classification (Nalebuff)[29]

This classification is not sequential and only describes different patterns of deformity.

Type 1 – Boutonnière-like deformity

- MCP joint flexion and IP joint hyperextension
- Importantly the basal joint is not affected
- This is the most common pattern of deformity in the rheumatoid thumb
- The primary disease is at the MCP joint where synovitis bulging dorsally causes attrition of the EPB insertion and extensor hood damage with loss of MCP joint extension. The EPL subluxes ulnarly and in time starts to act as a flexor of the MCP joint. The IP joint gradually hyperextends because all the muscles are now extending it
- In the early stages both the MCP joint flexion and IP joint hyperextension are passively correctable
- However, relatively rapid fixed deformities develop, initially of the MCP joint and later of the IP joint as well
- Treatment
  - Synovectomy with EPL rerouting
  - MCP joint fusion

Type 2 – Boutonnière with CMC joint subluxation

- This is rare
- A combination of the type 1 boutonnière deformity with subluxation or dislocation of the CMC joint
- The treatment is similar to that for type 1 deformity, with the addition of trapeziectomy

Type 3 – Swan-neck deformity

- The reverse of the boutonnière type deformity
- This is the second most common rheumatoid thumb deformity
- The disease starts at the CMC joint and leads to subluxation of that joint
- Deformity is CMC joint subluxation/dislocation leading to an adduction contracture
- It is impossible to move the thumb out of the palm without hyperextending the MCP joint, which in turn causes IP joint flexion
- Mobility is needed at the basal thumb joint so that the thumb can be positioned appropriately and, therefore, this precludes fusion
- Management
  - CMCJ – trapeziectomy
  - MCP joint
    - Mild deformity – Temporary pinning and capsulodesis
    - Severe deformoty – MCP joint fusion

Type 4 – Gamekeeper's thumb at the UCL

- Disease is confined to the MCP joint and is similar to a UCL rupture
- The ligament is stretched rather than ruptured and this often results in a secondary adduction contracture of the web space
- The CMC joint and IP joint are usually normal
- Surgical treatment is aimed at stabilising the MCP joint
- Management
  - In the early stages this is achieved by synovectomy and repair or reconstruction of the UCL
  - In more advanced cases, where joint destruction is present, arthrodesis of the MCP joint with or without a web space release is indicated

Type 5 – Hyperextension of the MCP joint

- This is rare
- The deformity is caused by isolated hyperextension of the MCP joint owing to slackening and lengthening of the volar plate
- There is no adduction of the metacarpal, which distinguishes it from the type 3 deformity
- As this hyperextension increases there is compensatory flexion of the IP joint caused by FPL tightness
- Treatment aim is to provide stability to the MCP joint in extension
  - Capsulodesis
  - Tenodesis
  - Arthrodesis

Arthritis mutilans

- Severe destruction of all joints with gross instability and shortening of the thumb
- This is difficult to manage and treatment usually involves fusion to maintain or gain length

General rules

- Primary joint indicates the deformity and other joint collapses into a particular instability pattern
- Second joint deformity can become fixed and require treatment

- It is impossible to consider the primary joint in isolation and the effect of treatment on one joint must be considered in relation to its effect on other joints
- Is the joint deformity flexible or fixed?
- The thumb collapses into a zigzag pattern in both the flexion/extension and abduction/adduction planes
- Instability in the thumb, particularly at the MCP and IP joints, is more disabling than loss of flexion and extension

Examination corner

Hand oral 1: Clinical photograph of a rheumatoid hand

- **Describe the deformities present**
  - Best to say boutonnière or swan-neck and then describe the deformity – It allows the examiner to know you recognise it.
- **What classification systems are used for the deformities seen?**
  - Nalebuff
- **Describe the management of the boutonnière thumb.**
  - In the early stages synovectomy and possible soft-tissue but most patients will require fusion

Hand Oral 2: clinical photograph of a rheumatoid thumb

- **Describe the deformities seen**
  - As above – Be systematic
- **Discussion about the management options of the MCP joint**
  - Synovectomy in early cases
  - Most will be beyond this and require a bony procedure – Fusion
- **How would you fuse the joint – Approach, technique and position of fusion**
  - Dorsal approach
  - Care with the extensors
  - Multiple options – Decide which you will use
- Bony options –
  - Chevron is more stable but fiddly
  - Ball and socket gives more opportunity to adjust the fusion position
  - Straight cut is easiest but the fusion angle is the fixed with the cut
- Fixation options
  - K-wires – Cheap but can irriatate the skin, tendons and create a passage for infection, but are easily removed if infection occurs
  - Cerclage wire – Fiddly but good compression, the wires can irritate
  - Headless compression screws – Good compression, more costly, can be difficult in getting sufficient bone in the head of the metacarpal for the screw to purchase (given the angle of fixation)
- Angle of fixation best judged by resting the tip of the thumb on the radial aspect of the index fingertip – Know you can get good pinch
- **How would you manage the patient if the had associated CMCJ disease**
  - Consider steroid injections for pain
  - If deformity then trapeziectomy

Boutonnière deformity of the finger

Introduction

- **PIP joint flexion** and **DIP joint hyperextension**
- It can be further classified as to whether the deformity is fixed or flexible
- Boutonnière is the French word for buttonhole, and is used in this context because the head of the PP buttonholes through the extensor hood secondary to rupture of the central slip

Pathology

- The pathology starts in the PIPJ
- The central slip attenuates or ruptures due to synovitis
- Volar subluxation of the lateral bands occurs because of disruption of the triangular ligament
- The lateral bands become converted from an extensor to a flexor of the PIP joint
- The functional loss with a boutonnière deformity is a lot less than with the swan-neck deformity, especially if some flexion is possible at the distal joint

Acute injury

- This is usually traumatic and can be difficult to diagnose as the finger does not immediately adopt a Boutonnière position (not until the lateral bands have subluxed volarwards)
- Treatment is conservative, using a 'Capener splint' which allows active DIP joint flexion/extension

Elson's test for acute central slip disruption[32]

- The PIP joint of the finger is bent 90° over the edge of a table. With resisted middle phalanx extension, the DIP joint either
  - Goes into rigid extension (positive test – Disruption of the central slip) because all the forces in the finger are distributed to the terminal tendon through the intact lateral bands, or
  - Remains floppy (negative test)

Classification (Nalebuff and Millender)

Based on the degree of deformity, the presence of passive correctability and the state of the joint surfaces:

- **Mild**
  - Dynamic imbalance

  - Passively correctable PIP and DIP joints
  - PIP joint lag of 10–15° in extension
- **Moderate**
  - Established contracture (i.e. not passively correctable)
  - PIP joint lag of 30–40° in extension
  - Joint preserved
- **Severe**
  - PIP joint in fixed flexed position with joint involvement
  - Volar plate and collaterals also contracted

Management options for chronic deformity

- Many operations have been described for the management of this deformity but often the results from surgery can be highly variable and unpredictable
- A word of caution – Great care is needed when deciding to operate on the PIP joint as, although extension may be regained, one can easily lose flexion and end up either no better off or worse than before surgery
- Moreover, correction of a mild boutonnière deformity is often associated with minimal functional improvement and the recurrence rate is high
- The results of soft-tissue reconstruction of rheumatoid boutonnière deformity can be unsatisfactory and, if surgery is required, fusion of the PIP joint in a functional position may be a safer option

Terminal tendon release

- Release of the extensor mechanism at the junction of the middle and proximal thirds of the middle phalanx, which leaves the ORL intact
- The lateral bands slide proximally, increasing extensor tone at the PIP joint and the intact ORL provides extensor tone to the DIP joint
- More simply, there is less hyperextension stress on the DIP joint and the flexion of the PIP joint is lessened
- If a resultant mallet occurs in the DIPJ this is usually mild and better tolerated than the bouttoniere

Secondary tendon reconstruction

- Excision of scar tissue and direct repair of central slip
- Free tendon graft (central slip reconstruction)
- Lateral band transfer procedure
- This is only carried out after passive joint motion has been restored, using one lateral band as a form of reconstruction of the central slip
  - Littler – Ulnar lateral band through radial lateral band to P2
  - Matev – The ulnar lateral band is transferred to a distal stump of the radial lateral band. The proximal stump of the radial lateral band is brought through the central slip and anchored at the dorsal base P2

Arthrodesis

- A predictable form of relieving pain
- Function is best preserved by considering the role of the digit involved as varying degrees of flexion are required depending on which finger involved

Arthroplasty

- Not routinely used for grade 3
- The results of arthroplasty in pre-existing boutonnière fingers are poorer and less predictable

Classification-based approach

- Type 1
  - Trial of splinting
  - Terminal extensor release
- Type 2
  - Central slip reconstruction followed by static then dynamic splints
- Type 3
  - Arthrodesis

Examination corner

Hand oral 1: Clinical photograph of a rheumatoid boutonnière finger deformity

Spot diagnosis

- **What is a boutonnière deformity?**
  - Describe the deformity at each joint
- **How are boutonnière deformities classified?**
  - Nalebuff
  - Outline the classification
- **What are the management options for a boutonnière deformity?**
  - Depends on the pain and functional problems to the patient – It may require nothing
  - A classification-based approach:

- Type 1
  - Trial of splinting
  - Terminal extensor release
- Type 2
  - Central slip reconstruction followed by static then dynamic splints
- Type 3
  - Arthrodesis

Hand oral 2: candidate handed a Capener splint

- **What is this?**
  - Capener splint
- **Fit it to the examiner's hand**
  - Really simple – The key is to know which way round to place it!
  - Find one from a hand therapist and practice applying it

- **How does the splint work?**
  - Will maintain the PIPJ in extension at rest
- **When might you use it?**
  - Predominantly in acute central slip injuries.
- **Describe Elson's test**
  - The PIP joint of the finger is bent 90° over the edge of a table. With resisted middle phalanx extension, the DIP joint either
- Goes into rigid extension (positive test – Disruption of the central slip) because all the forces in the finger are distributed to the terminal tendon through the intact lateral bands, or
- Remains floppy (negative test)
- **Management of a chronic boutonnière.**
  - As above oral

Swan-neck deformity

Introduction

- PIP joint hyperextension and DIP joint flexion
- It is a common, progressive and disabling deformity that severely affects hand function due to loss of flexion at the PIP joint

Pathology

- The deformity is caused by an **imbalance of forces** at the PIP joint and a lax volar plate
- The deformity can only occur if hyperextension is possible at the PIP joint
- Unlike the boutonnière deformity the condition can be secondary to problems at either the MCP joint or DIP joint
- The condition most commonly occurs in rheumatoid disease although there are other rarer causes (mallet finger, laceration or transfer of FDS)
- Other causes
  - Intrinsic tightness secondary to MCP joint disease
  - Intrinsic contracture
  - FDS rupture
  - Volar plate insufficiency
  - Mallet deformity
  - Extrinsic spasticity

Classification (Nalebuff types 1–4)

1. Flexible hyperextension deformity of the PIP joint
2. PIP joint flexion is limited when the MCP joint is maintained in extension. i.e. Intrinsic muscle tightness is present
3. Limited PIP joint flexion in all MCP joint positions, but the PIP joint surface is still preserved
4. PIP joint is stiff and there is destruction of the articular surface of the joint

Management (based on the classification)

1. PIPJ correctable
   - Extension restriction splint, e.g. Murphy splint(if the PIP joint is the problem)
   - Fusion DIP joint (if the DIP joint is the problem)
2. Intrinsic release plus some form of tenodesis on the volar aspect of the PIP joint
   - Oblique retinacular ligament reconstruction
   - FDS tenodesis
3. Either a soft-tissue procedure or arthrodesis
   - Prerequisites for a soft-tissue procedure are full flexion of the PIP joint before soft-tissue reconstruction, which may require MUA, sometimes the release of the dorsally contracted skin and then intrinsic release, if appropriate, and some form of tenodesis
4. Arthrodesis is probably the procedure of choice in this advanced stage

FDS tenodesis

Through a volar incision one limb of the FDS tendon is divided proximally and passed dorsally through the middle phalanx to provide a volar tether to hyperextension. K-wires are placed across the joint for 4 weeks.

Examination corner

Hand oral 1: Clinical photograph of rheumatoid swan-neck finger deformity

- **What is the swan-neck deformity?**
  - Decribe the deformity at each joint
- **What is it caused by?**
  - Unlike boutonnière this is more complicated and can be caused by problems at MCP, PIP or DIP joints
  - The outline the problems that can be caused at each joint
- **How do you classify them?**
  - Nalebuff
  - Then outline the different classes
- **How do you manage them?**
  - Patient-centred treatment – May not require anything
  - Then talk through the management based on the classification (as above in the text)

Hand oral 2: Candidate handed a Murphy splint

- **What is this?**
  - Murphy ring
- **What is the classification of swan-neck deformity?**
  - Nalebuff then go through the stages
- **Which type/types would this be suitable for?**
  - Supple correctable swan-necks to prevent the hyperextension in the PIPJ
- **Asked to fit to the examiner's hand.**
  - Ensure you have seen one and fitted it – The important thing is to put it on the correct way around

Scaphoid fractures[33–39]

- The scaphoid is the most commonly fractured bone in the carpus
- Knowledge is required of the diagnosis and management of acute fractures and the complications of non-union and avascular necrosis (AVN)

Blood supply

- The blood supply of the waist and proximal pole of the scaphoid (70%) is derived from the dorsal branch of the radial artery entering distally on the dorsal ridge through ligamentous and capsular attachments
- The proximal pole is the region with the most tenuous blood supply, owing to the distal to proximal (retrograde) intraosseous supply
- The distal scaphoid and tuberosity (30%) are supplied by branches of the superficial palmar branch of the radial artery

Mechanism of injury

- Fall onto an outstretched hand resulting in forced dorsiflexion of the wrist

Examination

- Fullness in the ASB (effusion in wrist)
- Tenderness in the ASB and scaphoid tubercle
- Reduced range of motion (but not dramatically)
- Pain at extremes of motion
- Pronation followed by ulnar deviation will cause pain

Investigation

Radiographs (scaphoid series)

Four standard radiographs

- PA in ulnar deviation (extends scaphoid)
- Lateral
- Two oblique views

Sensitivity is only approximatley 70% at detecting acute scaphoid fractures. If radiographs are normal patients should be immobilized and re-examined and investigated approximately 2 weeks later.

Bone scanning

- Sensitivity 97% (93–99%) but specificity 89% (83–94%)
- May be positive in ST osteoarthritis

CT

- Sensitivity 93% (83–98%) and specificity 99% (96–100%)
- However, usually more accessible than MRI
- Also has a role in assessing displacement in radiographically proven fractures

MRI

- Sensitivity 96% (91–99%) and specificity 99% (96–100%)
- Fracture line will be visible on T2-weighted sequence as a line of high signal, which represents marrow oedema
- Changes present on MR scan after 12 hours
- MRI proven to have a greater diagnostic performance over other modalities and when can be undertaken offer cost-savings in terms of reduced use of immobilization and reduced time off work

Displacement

- This is important as displacement increase the risk of non-union
- Defined as
  - Displacement >1 mm
  - Angulation >10°
  - SL angle >45°
  - CL angle >15°

Fracture location

- Proximal pole (25%)
- Waist (65%)
- Distal pole and tuberosity (10%)

Classification (Herbert 1990)

- Interobserver reproducibility is fair
- Type A: Stable fractures
  - A1 Tuberosity fracture
  - A2 Incomplete waist fracture
- Type B: Unstable fractures
  - B1 Unstable oblique fracture
  - B2 Complete or displaced waist fracture
  - B3 Proximal pole fracture
  - B4 Transscaphoid perilunate dislocation
  - B5 Comminuted fracture
- Type C: Delayed union
- Type D: Established non-union
  - D1 – Fibrous union
  - D2 – Pseudarthrosis (sclerotic)

Management

Undisplaced

- Below elbow cast for 8 weeks
- Re-examine and x-ray at 8 weeks after removal of plaster
  - If still tender, then treat in cast for a further 4 weeks
- At 12 weeks leave free regardless of whether there is tenderness or not
- Percutaneous fixation
  - No current evidence for improving union rates over a cast assuming the fracture is truly undisplaced
  - However, an earlier return to work[34] (9.3 vs 13.2 weeks)
  - Suggested as over-treatment if used for all fractures[35]
- Cast choice – There is no proven benefit of plastering above or below the elbow, or of including the thumb or not

Displaced

- These require reduction and internal fixation with either
  - Headless compression screws (differential pitch on screw to provide compression), e.g. Herbert, Herbert–Whipple (cannulated), Acutrak (cannulated)
  - K-wires – Good ease of insertion but they do not provide compression. Use if there is marked comminution

Surgical technique

Volar

- Indicated for waist fractures as it does not damage the dorsal blood supply and can correct a humpback deformity
- Surface landmarks are the scaphoid tubercle and FCR tendon. Skin incision is longitudinal along the radial border of FCR, curving radially to the scaphoid tubercle at the distal wrist crease
- Divide the superficial branch of the radial artery and dissect through the bed of the FCR tendon sheath
- Incise and reflect the capsule and the radioscaphoid and radioscapholunate ligaments
- Screws are placed distal to proximal, 45° to the horizontal and 45° to the long axis of the forearm
- A piece of trapezium may need to be excised to gain access to the distal pole of the scaphoid

Dorsal

- Use for proximal pole fractures as it provides the best access when the wrist is hyperflexed
- Care is needed to avoid damage to the dorsal blood supply
- The incision is centred on Lister's tubercle. The approach is between the third and fourth extensor compartments (EPL and EDC). Transverse capsulotomy. Flex the wrist 90° to expose the proximal pole and to reduce the fracture
- The entry point for the wire for the screw is just radial to the scapholunate ligament and aim along the thumb metacarpal

Scaphoid non-union

Defined as fractures that have not united within 6 months of injury. The non-union is not necessarily painful to the patient but if left the patient will develop degenerative changes (SNAC – See later).

- Risk factors for the development of non-union include
  - Delay in diagnosis
  - Delay in treatment
  - Angulation (intrascaphoid angle >45°)
  - Displacement (>1 mm)
  - Location – The more proximal the greater risk
  - Smoking
- Incidence
  - Distal pole <10%
  - Waist 10–20%
  - Proximal third 30%
  - Proximal fifth 100%

Management

- Aims
  - Correct deformity
  - Restore alignment
  - Prevent development of a SNAC wrist
- Preoperative assessment
  - Position of non-union (proximal poles less likely to be successful than waist – 67% vs 85% respectively)
  - Time since original injury (The longer the time the less likely bone grafting will succeed)
  - Exclude evidence of degenerative changes
  - Vascularity of the proximal pole
  - Consider graft options and approach
- Graft options
  - Inlay (Russe) graft
    - Corticocancellous inlay graft set in a cavity made in the proximal and distal fragments of the scaphoid through a volar approach
    - The graft is slightly longer than the defect
    - The graft does not need internal fixation as the natural shape of the scaphoid clamps down on this graft and keeps it stable
  - Interposition (Fisk) graft
    - Corticocancellous opening wedge graft placed through a volar approach and designed to restore scaphoid length and correct angulation
    - This is the preferred option for a humpback deformity and carpal instability (DISI)
  - Vascularized bone graft
    - Reportedly higher union rates in displaced non-unions and non-unions with AVN
    - Huge number of grafts described but can be divided into
      - Pedicled
      - 1,2-intercompartmental supraretinacular artery (1,2-ISCRA) (aka Zaidemberg)
      - Pronator quadratus bone graft (aka Mathoulin)
      - Index finger metacarpal
      - Free vascularized bone grafts (NB. Microvascular skills and very time-consuming)
      - Iliac crest
      - Medial femoral condyle
      - Use of these free grafts is currently unclear and controversial

- Which graft?
  - Large systematic review[36]
    - Non-vascularized graft without internal fixation – Union rate 80%
    - Non-vascularized graft with internal fixation – Union rate 84%
    - Vascularized bone grafts – Union rate 91%
    - This study, however, failed to separate confounding variables such as fracture location and age
  - Meta-analysis[37]
    - AVN of the proximal pole on MRI
    - Vascularized graft – Union 88%
    - Non-vascularized graft – Union 47%
  - A suggested treatment algorithm by Gray and Shin[38]
    - If AVN or displaced, use a vascularized graft
    - If non-displaced and no evidence of AVN, use a non-vascularized graft
  - The choice of graft predominantly remains down to surgeons experience and preference

- Assessing for AVN
  - AVN of the proximal pole is an important predictive factor in the success of surgery to treat non-unions
  - The incidence varies widely, from 9% to 40%, following waist fractures
  - Radiographs may show increased density of the proximal scaphoid fragment (owing to decreased bone turnover)
  - Gadolinium-enhanced MRI may correlate with outcome, but the gold standard is punctate bleeding at surgery
- Fixation
  - None – Relies completely on the support from the graft wedged inside the bone
  - K-wires – Usually used in very proximal non-unions where there is insufficient bone proximally to take a screw. However, requires removal before wrist can be mobilized
  - Headless compression screw – Preferred option by most surgeons as provides compression, stabilises the graft and does not require routine removal
- Approach
  - Waist – Can be dorsal or volar though easier to correct a 'humpback' deformity through a volar approach
  - Proximal pole – Dorsal approach

Oral question

Hand oral

A set of scaphoid views radiographs – No obvious fracture. Patient present with acute pain following a fall.

- **How would you assess this patient?**
  - Careful history – Does the mechanism fit with a possible carpal injury
  - Examination – Presence of effusion, where are they tender, may have only minor limitation of movement.
  - Radiographs
  - Four views – May be negative
- **What treatment would you give this patient now?**
  - If clinically could be a scaphoid fracture treat as such
  - Cast for 2 weeks
  - Reasonable to state you would follow your hospital protocol (if you have one)
- **When would you review? How would you reassess?**
  - Two weeks clinically and radiologically
- **What would you do if repeat radiographs were normal?**
  - Need further imaging – Either CT, bone scan or MRI depending on local resources or protocol. Be aware of specificity and sensitivty rates (in above text)
- **What cast would you use? Why?**
  - Below elbow cast with the thumb not included
  - No evidence that above elbow or thumb inclusion makes any difference
  - Wrist position also makes no difference but most people use slight extension as this is a more useful position for the wrist
- **When would you get further radiographs?**
  - You can not diagnose union on one set of x-rays (Just as it can be difficult to ascertain the fracture on radiographs, union can be the same). It is a combination of clinical examination and radiographs that allow union to be stated
- **What would you do if the patient still had pain at 8 weeks? (If cast again – Then what at 12 weeks?) i.e. at what time would you mobilize?**
  - Get further x-rays.
  - Treat for upto 12 weeks.
  - No evidence that continued cast treatment beyond 12 weeks makes any difference to union rates
- **When do you discharge the patient from clinic?**
  - When clinically and radiologically united
  - Usually need at least one set of radiographs at least 6 months form injury to ensure the fracture united (or at least shows no evidence of non-union on the radiographs)

Hand oral 2: radiographs showing an obvious scaphoid fracture

- **What is going on here?**
- **Is it displaced? How else could you assess this?**
  - Displacement is difficult to adequately asssess on radiographs – Anything other than a simple hairline fracture may be displaced
  - Get a CT if in doubt
- **How would you treat this patient?**
  - CT to assess displacement
  - Closed reduction and percutaneous fixation with a headless compression screw
- **When would you let this patient return to work?**
  - Depends on the job
  - Desk based jobs can be returned to within 2 weeks if comfortable but must avoid heavy lifting
  - Heavy manual work may require 2–3 months

Hand oral 3: radiographs showing an established non-union

- **Why treat a scaphoid non-union?**
  - To decrease pain, reduce the risk of secondary OA, correct carpal kinematics and increase function
- **How would you treat the non-union?**
  - Preop work up with CT to assess size of the defect
  - MRI can be used to assess vascularity of the proximal pole
- **What type of bone graft would you use?**
  - Need a discussion of different forms of autograft available
  - Distal radius is within the operative field and avoid the co-morbidity of other donor sites
  - Vascularized 'vs' non-vascularized
  - Iliac crest – Morbidity associated with it.

Examination corner

Hand oral 1: Radiograph of a waist of scaphoid non-union

- Approaches to the scaphoid – Both dorsal and volar

Hand oral 2: Radiograph of a scaphoid non-union post screw fixation

- **Discuss your management now?**
  - Management based on patient symptoms and need
  - If the screw is loose this would need to be removed to prevent further damage to joint surfaces
  - Need to assess size of the defect and presence of arthritis
  - Discussion with the patient about possibly attempting bone grafting and further screw fixation

Hand oral 3: Radiograph of a complete waist of scaphoid fracture (Herbert B2)

- **How would you manage this fracture?**
  - This is a Herbert B2 fracture. Its management is controversial. Some surgeons would fix it and others would manage it conservatively. The advantage of percutaneous fixation is a faster return to work as suggested in the paper by McQueen et al. in the JBJS 2008[34] and I would discuss this with the patient

The present management for AVN is a vascularized bone graft.

Oral question

Discuss the nature and treatment of non-union and AVN.

SNAC wrist

- With a non-union of the scaphoid, arthritis is likely to develop at 5–10 years
- Patients commonly present after minor trauma with wrist pain, having been previously asymptomatic. Radiographs show a non-union with longstanding degenerative changes

Classification

1. Arthritic changes between the radial styloid and distal scaphoid
2. Degenerative process affecting the whole scaphoid fossa of the distal radius
3. Capitolunate arthritis (radiolunate joint spared)
4. Whole carpus involved

Management

Stage 1

- Radial styloidectomy and limited carpal fusion (scaphocapitate or scaphoid–lunate–capitate)
- If the scaphoid proximal pole is necrotic it may be removed after performing a limited arthrodesis between the distal scaphoid and capitate and styloidectomy

Stage 2

- Scaphoid excision and four-corner fusion
- Proximal row carpectomy (not in younger patients due to risk arthritis)

Stage 3

- Scaphoid excision plus four-corner fusion is probably the procedure of choice as the head of the capitate is involved

Stage 4

- Wrist arthrodesis

Four-corner fusion (FCF) vs proximal row carpectomy (PRC)[39]

- Decreased need for immobilization and earlier recovery with a PRC
- No concern with metalwork or risk of non-union with PRC
- Tendency towards greater grip strength with FCF
- No difference in ROM
- PRC produces an incongruent joint (capitate head has a smaller radius of curvature than the lunate fossa), and studies have shown degenerative change may occur. Therefore, not recommended for younger patients

Examination corner

Hand oral 1: SNAC wrist in a 35-year-old manual worker

- **What is the diagnosis?**
  - SNAC wrist
- **How do you classify this condition?**
  - Go through how the arthritis develops sequentially through each stage
- **What options could allow him to keep his manual job?**
  - Don't forget simple things – Analgesics, splints and steroid injections.
  - Can try a wrist denervation

- Limited arthrodesis such as a FCF
- Alternatively can consider a PRC

- **How would you perform a four-corner fusion?**
  - Dorsal approach to the wrist
  - Berger capsulotomy
  - Inspect the radiolunate joint surfaces to ensure a FCF is reasonable
  - Prepare the joint surfaces with osteotomes, curettage and rongeurs
  - Reduce the lunate angulation (usually extended in a DISI deformity) before fixation as this will affect the postop ROM
  - Fixation

- Headless compression screws – Main difficulty can be getting the correct angle
- Hubcap plate – Main difficulty is positioning the plate so it does not impinge on the dorsal surface of the radius

Hand oral 2: SNAC wrist grade I in an asymptomatic 25-year old man

- **How would you manage this patient?**
  - Advise this is a difficult problem and discuss carefully with the patient
  - As surgery can make him symptomatic and risk of complications advise non-operative management
  - It is likely his arthritis will progress on radiographs but he may remain asymptomatic
  - Expalin part of the joint is already damaged and surgery at this stage has not been proven to prevent progression
- **What if this man was 65?**
  - Most would agree to leave this until he became symptomatic

Carpal instability[40–44]

Definition

Carpal instability is a term used to describe abnormal carpal biomechanics under physiological loading owing to disruption of the complex ligament system that controls the relative motion of the bones that form the carpus.

Carpal anatomy

The biomechanics of the wrist joint are difficult to understand without first understanding some anatomy. The carpus is composed of two rows of bones: The proximal row and the distal row. There are four joints at the wrist: DRUJ, radiocarpal, midcarpal and carpometacarpal.

Distal row

The bones in the distal row are: Trapezium, trapezoid, capitate and hamate. They are bound together by strong interosseous (intrinsic) ligaments and move together as a single unit.

Proximal row

The bones in the proximal row are: Scaphoid, lunate and triquetrum. The proximal row moves as an intercalated segment. It has no direct muscle attachments and is linked by strong intrinsic ligaments. It moves as a result of the forces applied to the distal carpal row causing relative movement at the midcarpal and radiocarpal joints.

Intrinsic ligaments

- The intrinsic ligaments have their origin and insertion within the same carpal row
- They are short stout structures, which are not amenable to surgical repair
- The distal row firmly binds all the distal carpal bones together so that they move as one
- The most important proximal row ligaments are the scapholunate ligament (SLL) and the lunotriquetral ligament (LTL)
- Both these ligaments allow some (but not excessive) movement between the proximal carpal bones and transmit forces along the row to ensure adaptive motion
- Both these ligaments have three parts – Volar, proximal and dorsal portions. This leaves the distal portion of the articulation between the scaphoid and lunate, and between the lunate and triquetrum free
- Scapholunate ligament
  - The dorsal portion provides the greatest yield strength and constraint to rotation, translation and distraction
  - The volar portion provides additional rotational stability
  - Secondary stabilisers include the dorsal intercarpal (DIC), dorsal radiocarpal (DRC) and scaphotrapezial (ST) ligaments
- Lunotriquetral ligament
  - In contrast to the SLL, the volar portion is the strongest and thickest part of the ligament providing the greatest resitance to rotation, translation and distraction
  - Secondary stabilisers include the ulnar segment of the ulnar arcuate, radiotriquetral and DIC ligaments

Extrinsic ligaments

The extrinsic ligaments connect the carpal bones to the radius or metacarpals. They are stronger volarly. The dorsal aspect ligaments are weaker and consist of radiolunotriquetral (RLT) and transverse ligaments (basis for the Berger flap[42]).

Space of Poirier

There are no ligaments running from the centre of the distal end of the radius to the capitate and this leaves an area of weakness over the front of the lunocapitate joint. A lunate dislocation or perilunate fracture dislocation is associated with a transverse capsular rent through this inherently weak region.

Kinematics

- Kinematics involves the study of movements of a body without reference to the forces that are acting to cause that movement

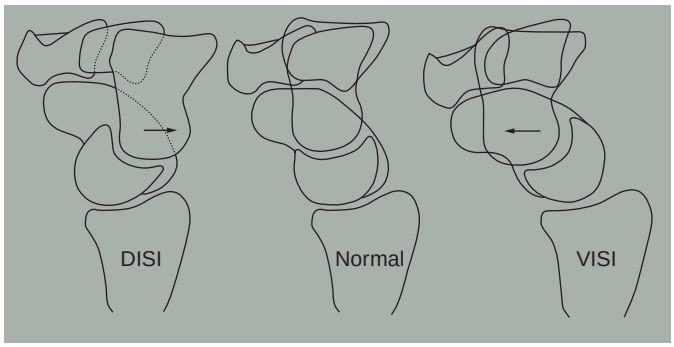

Figure 21.12 Dorsal intercalated segment instability (DISI) deformity – Dorsal angulation scaphoid with scapholunate angle >60°

- The wrist is a composite joint which requires movement in both the midcarpal and radiocarpal joints to achieve a full range of flexion and extension, with approximately half occuring in each of these joints
- The proximal carpal row flexes in radial deviation and extends in ulnar deviation
- The scaphoid has a naturally tendency to flex in contrast to the triquetrum which extends
- This leaves the lunate as the link between these two bones and so it essentially acts as a torque convertor
- If the LTL is disrupted the lunate would tend to flex with the scaphoid and create a VISI deformity (Figure 21.12)
- If the SLL is disrupted the lunate would tend to extend with the triqetrum and create a DISI deformity
- Isolated midcarpal joint motion occurs through a dart-throwing motion plane – Radial devation with extension to ulnar deviation with flexion. This *may* have a role in rehabilitation (e.g. following SLL reconstruction – Dart-throwing exercises will move the midcarpal joint without stressing the proximal row and, thus, the repair)
- In contrast, isolated radiocarpal motion occurs with a reverse dart-throwers action along the plane of extension with ulnar deviation to flexion with radial deviation

Row theory

The proximal carpal row is interlinked by the interosseous ligaments and moves independently of the distal carpal row.

Column theory

The wrist consists of three longitudinal columns: The lateral column (scaphoid), which is mobile; a central column (capitate, lunate), which provides flexion/extension; and a medial column (hamate, triquetrum), which allows carpal rotation. Each column provides a different type of wrist stability.

Oval ring theory

Compromise between the two above theories, in which the carpus is considered as a ring.

There has been disagreement between the various supporters of each theory but it would appear that some wrists function more like rows and others more like columns.

Terminology

Elements of the official terminology for instability are complex but it is critical to grasp them. They include:

- **Static** – Constant
- **Dynamic** – Intermittent
- **Dissociative** – Between bones of the same carpal row (e.g. DISI/VISI)
- **Non-dissociative** – Between the proximal and distal rows or between the proximal row and distal radius (e.g. midcarpal)

No universally accepted system exists for classifying carpal instabilities. Generally speaking, five patterns of instability are described.

Carpal instability dissociative (CID)

Relates to instability between (or through) carpal bones of the same row (either proximal or distal).

Carpal instability non-dissociative (CIND)

Relates to instability between carpal rows or transverse osseous segments and can be caused by ligament injury or bony fracture (or both).

Carpal instability complex (CIC)

Combination of CID and CIND lesions or defying other classifications that are called complex.

Most frequently represented by the perilunate fracture dislocation and the volar lunate dislocation. Mayfield et al. classified this injury into four stages, progressing from radial to ulnar[43]. They can be lesser arc (ligamentous) or greater arc (radial styloid, scaphoid or capitate fracture):

- Stage I – Rupture of the scapholunate and radioscaphocapitate ligaments
- Stage II – Dislocation of the capitolunate joint
- Stage III – Rupture of the lunotriquetral interosseous ligament
- Stage IV – Dislocation of the lunate

In 95% of cases the capitate dislocates dorsally off the lunate. In a volar lunate dislocation it passes through a weakness between the volar extrinsic ligaments – The space of Poirier.

Carpal injury adaptive (CIA)

Another type of carpal instability is called adaptive and is the consequence of deformity in the distal radius from a fracture. Therefore, it should correct with anatomical reduction of the fracture.

Clinical features

History

- Mechanism of any injury (e.g. history of a sprained wrist which fails to resolve)
- Aim to localize symptoms

- Pain with loading activities and weakness
- Click or clunk on wrist movement
- Swelling
- Loss of function

Examination

- Swelling
- Localized tenderness (scapholunate ligament found just distal to Lister's tubercle)
- Active and passive range of motion

Special tests

Specific provocative manoeuvres should be performed based on the patient's symptoms:

- Pseudostability test – For midcarpal instability
- Kirk–Watson's test – Assesses scapholunate ligament competence
- Reagan's ballottement test – For lunotriquetral instability. Trap the lunate between the thumb and index finger of one hand and the triquetrum in the other as the bones are moved independently and in opposite directions to each other
- Kleinman's shear test – The examiner's thumbs are placed on the dorsal aspect of the pisiform and lunate, and the bones are translated in an opposite direction with respect to each other

Investigations

X-rays

Carpal instability series:

- PA/lateral view of the wrist (wrist in neutral)
- Clenched fist PA view

Scapholunate instability (Figure 21.13):

- Scapholunate angle >60°
- Scapholunate gap >3 mm (Terry Thomas sign)
- Ring sign (end-on view of distal pole of flexed scaphoid)
- Step in Gilula's lines

Static instability:

- If present will show up on the x-ray

Dynamic instability:

- May not be seen even on the clenched fist view

If you clinically suspect a ligament injury but the Kirk–Watson's test and/or radiographs are negative, book the patient for either an MR arthrogram or an arthroscopy depending on your level of suspicion.

Arthroscopy

Allows direct visualisation of the radiocarpal and midcarpal joints, and intrinsic ligaments (Geissler classification for scapholunate instability). Dynamic stress tests can also be performed (although not with physiological loading). Arthroscopy allows the assessment of:

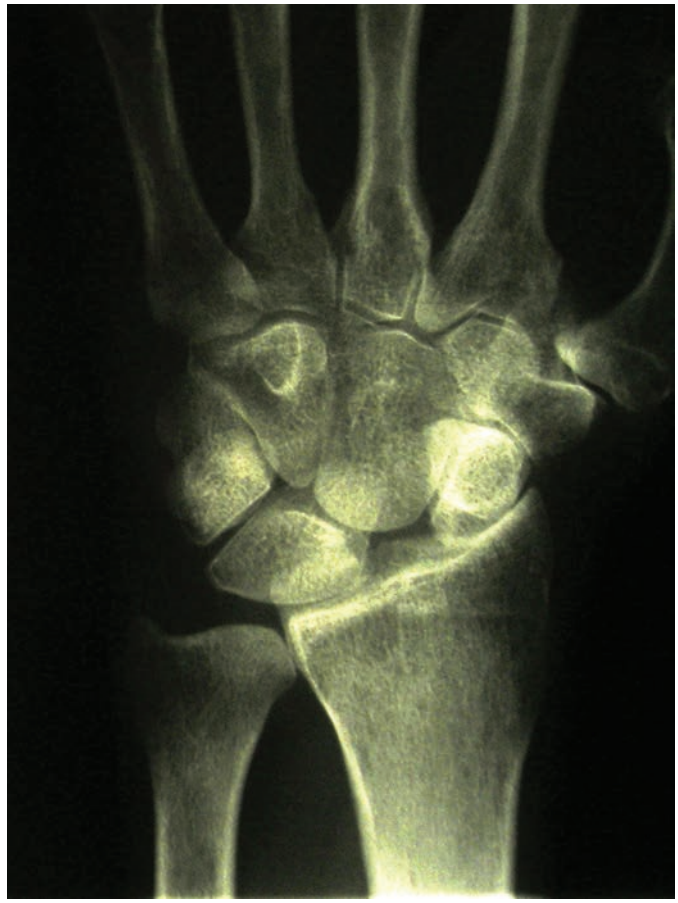

Figure 21.13 Radiograph showing marked scapholunate dissociation with flexed scaphoid (ring sign), loss of carpal height and loss of the radioscaphoid joint space (SLAC wrist)

- Cartilage surfaces
- Synovium
- Intrinsic and extrinsic ligaments
- Relative stability/motion of the carpal bones to each other
- Presence of anomalous structures, entrapped or mechanically interfering tissues, scar and/or other blocking tissue

The acronyms DISI and VISI refer to the static posture of the lunate seen on a true lateral radiograph of the wrist. A comparison of radiographic views of the contralateral wrist are essential because these findings may be noted in an asymptomatic wrist and may represent a normal variant.

Dorsal intercalated segment instability (DISI)

When the lunate is extended or rotated dorsally in relation to the long axis of the radius and capitate the situation is called DISI. The scapholunate angle is >60° (normal 30–60°, average 47°).

Causes include:

- SLL injury
- Scaphoid fracture
- Kienböck's
- Perilunate injury

Volar intercalated segment instability (VISI)

When the lunate is flexed and the scapholunate angle is <30° the situation is called VISI. It is much less common than DISI and is most commonly caused by lunotriquetral ligament injury.

Management

Scapholunate ligament injury

Acute

Early open repair through a dorsal approach by direct suture, pull-through sutures or suture anchors. Supplemented by K-wire stabilisation of the scapholunate and scaphocapitate articulations. Delayed open repair can be performed up to 6 months after acute injury.

Chronic

Surgical procedures are classified as either bony or soft tissue

- Soft tissue: Excellent results have been reported for a modified Brunelli procedure (FCR tenodesis)[44]. Dorsal capsulodesis (Blatt capsulodesis) has fallen out of favour
- Bony procedures: Limited wrist fusion, such as the scaphotrapeziotrapezoid fusion (STT), to correct DISI. These procedures can be technically difficult and demanding

Lunotriquetral ligament injury

Rarely recognised acutely but, if so, then perform acute open repair of the ligament. The scaphoid and lunate are both flexed, leaving the triquetrum extended. VISI due to CID. Chronic symptomatic lunotriquetral instability should be treated by either lunotriquetral fusion (high rate non-union), FCU tenodesis or an ulnar shortening.

Acute perilunate dislocation

Emergency treatment is closed reduction with open repair of the ligaments possibly when the swelling has settled.

Closed reduction

- Dorsal perilunate dislocation – Hyperdorsiflex the wrist, apply traction and try to hinge the capitate head on to the lunate before flexing the wrist. Likely to reduce because of extensive soft-tissue injury
- Volar lunate dislocation – Hyperdorsiflex the wrist, apply pressure on the lunate to reduce and flex the wrist. May not reduce in a closed procedure. If open reduction is required for volar lunate dislocation use an extended carpal tunnel incision

ORIF

- All injured structures (radial styloid/scaphoid fracture, scapholunate ligament injury) can be repaired through a dorsal approach, although a carpal tunnel release may be required
- K-wires are used for bony stabilisation

Examination corner

Hand oral: Radiographs demonstrating perilunate and lunate dislocations

NB. These are really quite common in either the hand or trauma oral. This is a spot diagnosis that you really must be able to recognise without any prompting from the examiners. Mention that you need to see two views – AP and lateral – And be prepared to discuss surgical approaches and treatment options.

- You are called down to the A&E department because a motorcyclist has come off his motorbike at high speed and landed on his right wrist. The casualty officer has asked you to look at this x-ray to see if there is a fracture present
  - This is a PA radiograph of the right wrist. The most obvious feature is a break in Gilula's lines about the wrist; the carpus is foreshortened with overlapping of the proximal capitate and distal lunate margins. There is radial displacement of the fractured scaphoid with the distal carpal row. I would want to get a lateral radiograph to confirm my suspicion
- This is his lateral x-ray
  - The lateral radiograph confirms a dorsal trans-scaphoid perilunate fracture dislocation of the carpus. The lunate lies in a neutral position within the lunate fossa, in line with the radius. The distal poles of the scaphoid and triquetrum have displaced dorsally. The longitudinal axis of the capitate lies dorsal to the longitudinal axis of the radius
  - (if the lunate is displaced there is a 'spilled teacup' sign – The lunate is volarly rotated and displaced resembling a spilled teacup.)

- **How will you manage this injury?**
  - After a thorough neurological examination the patient needs to be taken urgently to theatre for reduction of this injury. Internal fixation can be performed at a later stage when swelling has settled, using a dorsal approach to visualize the scapholunate ligament, but a volar incision may also be required. Plaster immobilization is necessary for 6 weeks while the ligaments heal. In the long term, I would be concerned about avascular necrosis of the proximal pole of the scaphoid.

Scapholunate advanced collapse (SLAC wrist)

Background

Progressive arthritis caused by scapholunate interval disruption with a flexion deformity of the scaphoid.

Pathology

SL ligament disruption allows the lunate to extend and the scaphoid to flex (a DISI deformity), thus, reducing carpal height. Arthritis develops initially at the radial styloid due to flexion of the distal scaphoid (see Figure 21.11). The proximal

pole behaves like a ball and socket joint and only develops degenerative change at a later stage. The capitate migrates proximally through the widened scapholunate interval (loss of carpal height), leading to capitolunate arthritis. The radiolunate joint is spared as it is a ball and socket joint, and lunate extension still allows concentric loading of the lunate fossa of the distal radius.

Scapholunate interval – >2 mm

Carpal height ratio – Used to assess carpal collapse. Ratio of carpal height to the length of the third metacarpal determined on a PA radiograph. The normal value for carpal height is >0.54 (0.46–0.61)

Ring sign – Cortical silhouette of the flexed scaphoid tuberosity seen on PA view

Scapholunate angle – Long axis of the scaphoid in relation to the long axis of the lunate. Average 47°

Capitolunate angle – 0–15°

Classification of SLAC wrist

Watson has classified SLAC wrist into four stages:

Stage 1: Arthritis between the scaphoid and radial styloid

Stage 2: Arthritis between the scaphoid and entire scaphoid facet of the radius

Stage 3: Stage 2 plus arthritis between the capitate and lunate

Stage 4: Generalized arthritis

Management of SLAC wrist

Non-operative

- Indicated if symptoms are minor/minimal. Advice, analgesia, wrist support, etc

Surgical

- Options for surgical treatment are based on the stage of the disease

Stage 1

- Radial styloidectomy ± scaphoid stabilisation (STT fusion) ± capitolunate fusion
  - Technically demanding; must adhere to strict surgical details (Kirk–Watson); results can be unpredictable; excessive resection can result in wrist instability and ulnar translocation

Stage 2

- Scaphoid excision and four-corner fusion
  - If it unites (non-union 7%) a good result is maintained in the long term and some wrist movement is preserved (50% normal). Performed through a midline dorsal incision and scaphoid used as the bone graft. Incomplete reduction of the dorsiflexed lunate may result in limitation of wrist extension. A spider plate is introduced to improve results seen with K-wires/staples. However, studies have not yet shown this
- Proximal row carpectomy
  - Best motion (60% normal), worst grip strength and pain relief. Technically less demanding

Stage 3

- Scaphoid excision and FCF

Stage 4

- Wrist fusion
  - Best pain relief, good grip strength but loss of motion. Immobilize the wrist for a couple of weeks in plaster beforehand to see how the patient gets on. Position of wrist fusion is slight)extension (20°)

Examination corner

Hand oral: PA and lateral radiographs demonstrating scapholunate dissociation with obvious Terry Thomas sign

- **What are the various radiographic features of carpal instability?**
  - Widening of the scapholunate interval (>3 mm Terry Thomas sign is so-called because of the comedy actor who had a large gap between his two front teeth). Increased scapholunate angle (>45°) in the lateral view. Cortical ring sign of the scaphoid in the PA view – Caused by a volarflexed scaphoid. V-sign of Taleisnik – Refers to the volar silhouette of the palmar flexed scaphoid and radius seen on the lateral view. Discussion on management followed.
- **Which ligaments give secondary support to the SLL?**
  - Dorsal intercarpal
  - Dorsal radiocarpal
  - ST ligament
- **How would you treat this if this was 4 weeks post injury?**
  - This is at a stage when repair may still be possible.
  - May arthroscope wrist first to assess
  - Open repair through a dorsal approach
- **How would you treat this if the patient was 6 months post injury?**
  - Likely beyond repair stage – Now a question of reconstruction – Depending on patient's symptoms.
  - Still do wrist arthroscopy first to assess and look for any degenerate changes.

Wrist arthroscopy

This is primarily a diagnostic modality that can help locate sources of wrist pain when other modalities have failed to do so. It can also be used for a range of therapeutic procedures such as debridement, washout for sepsis, TFCC repairs, arthroscopically assisted distal radius fracture fixation, ganglion excision, as well as carpal bone excision and ligament repairs. Most of the therapeutic treatments are still in the developmental stages and are not currently mainstream.

Setup

- Patient supine
- Hand suspended from finger traps (two to four fingers included)
- Elbow flexed at 90° with an inflated tourniquet
- traction

Portals

- Access is predominantly from the dorsal surfaces with the portals named after the relationship to the extensor compartments
- 3–4 portal is the main access point to the radiocarpal joint and can be found by palpating the 'soft spot' just distal to Lister's tubercle
- A 4–5 or 6R portal is often secondarily introduced for instruments. This is best done initially with a needle and checking with the camera from the 3–4 portal that the access point and angel are correct
- There are two 6 portals – 6R (radial) and 6U (ulnar) named in relation to the ECU tendon
- Midcarpal portal is usually a less well defined soft spot approximately 1 cm distal to the 3–4 portal. This is the midcarpal radial portal – A more ulnar portal can be created again initially with a needle
- A 2.7 mm 30°angled arthroscope is usually used for arthroscopic purposes

Flexor tendon injuries[45–47]

Types of injury

It is important to know whether the finger was flexed or extended at the time of injury as this the position of the hand at the time of injury determines tendon retraction[40].

- Flexed fingers – Distal tendon retracts
- Extended fingers – Proximal tendon retracts

Biology of healing

Intrinsic healing is the formation of collagen bundles directly across the injury site. Extrinsic healing involves the formation of a layer of scar tissue surrounding the injury site and can lead to adhesions. Aim for 'no-touch' technique to prevent damage to tendons and adhesion formation.

The repair is weakest at 6–12 days.

Stages of healing

- Inflammatory (cellular) 0–5 days
  - Haematoma formation at the site of injury
  - Platelets release vasodilatory chemokines and chemotactic factors
  - Migration of inflammatory cells particularly neutrophils and macrophages
  - Phagocytosis of necrotic tissue and clot
- Fibroblastic (collagen) 5–28 days
  - Fibrobasts begin to rapidly proliferate and produce collagen type 3, proteoglycans and components of the extracellular matrix
  - Random alignment
- Remodelling (cross-linking) >28 days
  - Cellularity begins to decrease
  - Decrease in collagen type 3 production
  - Collagen fibres begin to orientate along the tendon parallel to the direction of stress
  - Connections between collagen fibres to increase strength

Contraindications to repair

- Wounds liable to infection
- Uncooperative patient

Zones

Kleinert and Verdan classified flexor tendon injuries according to the anatomical zone of injury:

Zone 1: Distal to FDS insertion

Zone 2: Bunnell's 'no man's land'. From A1 pulley to FDS insertion. FDS and FDP tendons are enclosed in a flexor sheath

Zone 3: Distal edge of flexor retinaculum to A1 pulley

Zone 4: Within the carpal tunnel

Zone 5: Proximal to the carpal tunnel

There are similar but less specifically described zones for the thumb, prefixed by T:

Thumb T1: FPL insertion to A2 pulley

Thumb T2: Zone 1 to distal part of the A1 pulley

Thumb T3: Zone 2 to carpal tunnel

Management

- The optimal suture material, number of strands and repair technique are still debated amongst hand surgeons
- The aim is to provide a repair that provides sufficient strength to allow early postoperative mobilization and minimize gap formation whilst trying to prevent further damage to the tendon and adhesion formation
- Technical aspects
  - Number of strands
    - Increasing the number of strands across the repair site increases the strength of the repair
    - The more strands means a bulkier repair which can inhibit gliding
    - The more strands means potential further tendon damage from increased handling
    - Currently most repairs provides a minimum of four strands

- Position of core suture
  - Position dorsally within the tendon increases strength possibly as this is the tension surface of the tendon when the finger is flexed
  - The core suture should pass 1 cm from the repair site. This has shown increased strength in contrast to shorter distances. This is probably due to the increased risk of suture pull-through form shorter distances
- Epitendinous suture
  - Helps to tidy the repair site and improve gliding
  - Improves the overall strangth of the repair
- Locking loops rather than grasping loops have increased breaking strength, reduced gapping and fewer incidents of suture pullout
- Minimal handling
  - The less the tendon is handled and further damaged the less risk there is of adhesion formation
- Actual repair techniques
  - There are multiple ones described (make a decision as to the one you will use, why and be able to draw it)
  - Examples are Kessler, modified Kessler, Strickland, Bunnell and Adelaide

- Technique
  - The repair should be carried out as early as possible after the injury
  - Apply a tourniquet and regional anaesthesia or a general anaesthetic
  - Make a Brunner or midaxial approach and use windows between pulleys
  - For retracted tendons try to milk the tendon with the wrist/digits flexed
  - If this fails, make a small transverse incision at the level of the distal palmar crease just proximal to the A1 pulley. Pass a Silastic cannula from the distal wound through the sheath to the proximal wound. Attach a catheter to the proximal end of the tendon and pull through to the distal wound
  - Use 3/0 non-absorbable sutures for core suture and a 6/0 monofilament for a circumferential epitenon suture
  - Close the sheath, if possible
  - For zone 2 injuries it is recommended that both tendons are repaired

Rehabilitation

- There are many variations on the rehabilitation and it is worth spending a couple of hours with a therapist to see what regimen they use
- Aim to mobilize early (48 h postoperatively) to prevent adhesions
- As little as 1.6 mm of tendon excursion can prevent clinically important adhesion formation
- Most patients will be put into and active 'place and hold' regimen rather than a traditional passive ROM regimen
- A randomised control trial by Trumble in 2010 found that patients that underwent active motion had significantly fewer flexion contractures, better satisfaction scores and improved ROM than passively rehabilitated patients. Importantly there was no difference in the tendon re-rupture rate[47]

Complications

- Re-rupture
- Infection
- Adhesions
- Joint contractures – Too tight a repair or from prolonged splintage
- Bowstringing – From damaged pulleys

Reconstruction

Defined as a delayed primary repair performed >3 weeks after injury. Contracture of the muscle–tendon unit has usually occurred and a tendon graft is often required. It can be carried out as a one-stage or two-stage procedure.

Prerequisites for tendon reconstruction:

- Motivated patient
- Adequate skin and soft-tissue cover
- Full passive range of movement of joints
- Adequate sensation and circulation of finger

Methods

- Delayed direct repair
- Single-stage flexor tendon grafting
- Two-stage grafting
- Tenodesis or arthrodesis
- Amputation

Two-stage flexor tendon reconstruction

First stage

Aims:

- Tenolysis and release of joint contractures
- Digital nerve repair or grafting
- Provide healthy skin (may require a flap)
- Full flexion on traction of the Silastic rod at the wrist
- Preserve A1, A2 and A4 pulleys

Second stage

Carried out 2–3 months after first stage. Tendon graft options include:

- Palmaris longus
- Plantaris – Medial to tendo-Achilles

- Long toe extensors – second, third or fourth toes
- EIP
- Fascia lata

Flexor pulley reconstruction

- Indicated if bowstringing of flexor tendons causes a functional problem with loss of flexion of digit
- This is required after rupture or iatrogenic damage to A2 or A4 pulleys
- Can use a free tendon graft of FDS passed under extensor tendon and neurovascular bundles at the level of the proximal and middle phalanges

FDP avulsion injuries

Caused by forced extension of a flexed DIPJ

Classification (Leddy)

I – Tendon end in palm. Rupture of vinculae
II – Tendon held at level of PIPJ by long vinculus
III – Held at A4 pulley by avulsed bony fragment
IV – Profundus avulsed off bony fragment

Management

In types I and II, extensive trauma and complications of adhesions and the quadriga effect are seen. DIP joint fusion should be considered. Type III requires ORIF and the fragment can be held with sutures that are passed through the distal phalanx and then tied on to a button on the nail.

Examination corner

Hand oral 1: Clinical photograph of a hand with a 3- to 4-cm horizontal laceration over the ulnar side of the distal palm

- **What structures could be damaged?**
  - Be systematic. State this is a laceration over the volar aspect of the distal palm
  - You would be concerned about the flexor tendons (both FDS and FDP at this level), the ulnar digital nerve and the radial digital/common digital nerve, the radial and ulnar digital arteries.
- **How would you extend the wound?**
  - The wound needs to be extended in a way that allows assessment of all these structures AND the potential need for repair as such they will require longituinal extensions probably in a Brunner type fashion
- **What are the principles of tendon repair?**
  - Minimal further damage to the tendon or sheath
  - Need to create windows within the sheath for repair (as needed) without risking bowstringing
  - Grasp the tendon in one spot only.
  - The need for both core and epitendinous sutures (have decided which and why you will use the one you do)
- **What are principles of nerve repair?**
  - Debride devitalized tissue
  - Mobilize each end of the nerve
  - Tension free repair
  - Repair under magnification/microsope
  - Fine suture, e.g. 9–0
- **What are the clinical signs of re-innervation of a nerve?**
  - Tinel's sign
  - Improving sensation
  - Sweating

Hand oral 2: flexor tendon injuries

- **Show me how you examine a injured hand?**
- **What are the results of flexor tendon repairs?**
- **Discuss the rehabilitation regimes: Kleinert, Duran and Belfast**
- **Are there any particular problems with Kleinert rehabilitation (PIP joint stiffness)?**

Hand oral 3: clinical picture of a hand with all fingers in resting posture except the middle finger in full extension

- **What is the diagnosis?**
  - FDP rupture
- **What other structures would you check?**
  - FDS
  - Digital nerve and artery
- **Check the digital artery supply on the examiner's fingers.**
  - Perform an Allen's type test on the digit
  - Also be prepared to demonstrate assessing for the digital nerve
- Pin-prick/2-point discrimination are fine
- Slide a pen down the side of the digit (**absence** of sweating in a cut nerve)
- **What repair would you use?**
  - Decide beforehand and be able to justify (see above text)
- **Draw the repair on a piece of paper.**
  - This is a must - amazing how many candidates can't draw it
- **What evidence have you for that repair?**
  - See above notes
- **How would you rehabilitate the patient afterwards?**
  - There are so many regimens – The key is to whether you are going for early active mobilization. That relies on a co-operative patient. Otherwise can do delayed mobilization but early active yield better results. Best to know what your therapists do and go through that – Most are variations on the Belfast

Hand oral 4: oatient presents with an inability to flex the thumb several months following volar plating for a distal radius fracture

- **What is the diagnosis?**
  - FPL rupture
- **Why has this happened?**
  - Usually attritional rupture over the plate or screw
- **What are the treatment options?**
  - Removal of the plate
  - Tendon reconstruction/transfer (using FDS ring finger)
  - or IPJ fusion

Thumb amputation

Examination corner

Hand oral: Clinical photograph of thumb amputation

A patient arrives in casualty with this injury. What is your management?

History

- Age, occupation, hand dominance
- Mechanism of injury
- Pre-existing hand problems
- Ischaemia time (warm – 6-h hand/12-h digit, cold – 12-h hand/24-h digit)
- Current medication
- Previous medical history
- Hobbies
- Smoking
- Tetanus status

Examination

- Level of injury
- Amputated part
- Degree of crush/avulsion
- Quality of skin and soft tissues
- Degree of contamination

Investigations

- Radiographs of the hand and injured part

Management

Absolute contraindication for reimplantation include:

- Life-threatening concomitant trauma
- Severe premorbid disease
- Severe injury to the digit – Extensive degloving, gross contamination

Relative contraindications:

- Lengthy warm ischaemia time
- Elderly with microvascular disease
- Uncooperative patient

Surgical options

Refer to experienced hand surgeon or the plastic surgeons if not experienced in dealing with this type of injury[41,42]

- Primary closure
- Reimplantation
- Thumb reconstruction
  - Wrap around procedure
  - Great toe transfer
  - Second toe transfer procedure

Ring avulsion injuries[48]

This is a topic that under normal circumstances one would briefly skip over during the course of preparation for the FRCS (Tr & Orth) exam. You need to be reasonably familiar with this one as it tends to be asked more often than you would normally expect.

Background

- Underestimated injury

Urbaniak classification

Class 1

- Circulation adequate, circumferential laceration
- Standard bone and soft-tissue management

Class 2

- Circulation inadequate
  - A – No additional bone, tendon or nerve injury
  - B – There are additional injuries present
  - Vessel repair (microvascular) is required
- In general, class 2B injuries may be better treated with amputation

Class 3

- Complete degloving of skin ± amputation through the DIPJ. These injuries are unlikely to gain adequate function, and amputation is usually required
- The most common complication following surgical reconstruction is cold intolerance

Examination corner

Hand oral 1: Clinical photograph of ring finger ring avulsion injury

- **Classification?**
  - Urbaniak and then outline the different classes
- **When would you consider an amputation?**
  - Class 2B and 3
- **What is the prognosis?**
  - Depends on the severity
  - Worsens in the greater classes
  - Cold intolerance, stiffness and swelling are not uncommon

Mallet finger[49,50]

This is another favourite FRCS (Tr & Orth) question in either the hand or trauma oral.

Definition

- A mallet finger deformity is characterized by loss of extension at the DIPJ
- They are typically closed injuries but can be caused by a laceration
- The mallet can be either a tendon tear or bony from an avulsion fracture
- Mechanism of injury is thought to be forced flexion of the DIPJ (e.g. a ball striking the tip of an extended finger)

Management

- Tendinous mallets
  - treat non-operatively in an extension splint
  - A wide variety of splints are available – Stack splints, aluminium and thermoplastic splints. A randomized control trial[50] found no difference in final extension lag following treatment. However, care has to be taken over skin maceration from sweating and often a custom-made splint may help to reduce this
  - Treat in the splint for 6–8 weeks
  - If there are soft-tissue complications from the splint or non-compliance is a problem then a percutaneous K-wire can be put across the DIPJ
- Bony mallet
  - most can be managed non-operatively unless the joint is subluxed or possibly a displaced fragment involving >1/3 of the joint
  - For those with joint subluxation attempt a closed reduction and percutaneous K-wire. If that fails, be prepared to perform an ORIF with either dorsal blocking wire and K-wire across the DIPJ or a suture anchor
- The results of both non-operative and operative treatment are not always satisfactory, with only 30–40% of patients regaining full extension the DIPJ
- However, the residual extansion lag in most patients is a cosmetic rather than a functional problem

Chronic mallet finger

- Initially treat with mallet splint for 6 weeks
- If this produces no benefit and the patient is requesting treatment, book for DIPJ fusion

Fingertip injuries[51]

- Either soft tissue or bony
- The principle is to achieve a well-healed fingertip and to preserve length
- If there is no exposed bone, treat non-operatively with Tegaderm dressings, especially in children
- If there is exposed bone this needs soft-tissue coverage. Options
  - Shorten the bone end and Tegaderm dressings or primary closure
  - Local flap coverage (Atasoy, Cutler, cross-finger)
  - Terminalisation – Indicated for a severely crushed distal phalanx, and is made through the DIPJ with trimming of the condyles of the head of the middle phalanx
- The nail plate lies beneath the nail and is responsible for its growth. If there is a crush injury possibly involving the nail bed, the nail should be removed and the nail bed explored and repaired as appropriate

High-pressure injection injuries[52]

- These are rare but severe injuries that should be treated as a surgical emergency
- The non-dominant hand is more commonly affected
- They occur when equipment, capable of reaching pressures greater than the skin can withstand, injects its contents into the human body
- The skin can withstand 7 bar whereas some of the equipment causing these problems can exceed 2500 bar
- Paint is thought to be more toxic than grease. Extensive soft-tissue necrosis occurs despite an often small entry wound. The extent of spread depends on the site of entry, anatomical barriers and the volume and pressure of the substance injected. Material readily passes along tendon sheaths as these offer the pathway of least resistance
- Clinical evaluation
  - History
    - Presure of the gun
    - Material injected
    - Volume injected
    - Distance from gun to extremity
    - Pain may not be present intially
  - Physical examination
    - The whole extremity
    - Look for entry and, if present, exit wounds
    - Careful N/V assessment of the digits and hand
    - Tendon involvement
    - Presence and location of crepitus
  - Radiological assessment
    - Radiodense materials can easily be seen and the extent located on plain radiographs
- Management
  - Broad spectrum antibiotics
  - Tetanus
  - Urgent surgical exploration, debridement and fasciotomy
  - The wound is excised and extended proximally and distally until the full extent of spread is revealed and as much material and non-viable tissue as possible is removed
  - Repeat debridement at 48 hours should be done
  - The hand is splinted and elevated in the position of safety
- Aggressive treatment has reduced the amputation rate, with most patients returning to their original work
- Digital injections are, however, followed by high rates of necrosis and amputation, and there is long-term morbidity in surviving digits

Examination corner

Hand oral 1: Clinical slide of a pressure injection

- **How would you assess this injury?**
  - History and physical assessment as detailed above
- **What are the principles of management?**
  - Emphasize surgical emergency
  - Broad spectrum antibitoics and tetanus
  - Exploration and debridement

Compartment syndrome

- Decreased tissue perfusion in a myofascial compartment leading to muscle necrosis and contractures
- This condition presents with severe pain and can be caused by any injury that either decreases compartment volume or increases compartment content such as:
  - Tight casts and dressings
  - Burns
  - Trauma
  - Reperfusion after replantation
- Hand – Treat by prompt decompression through two dorsal incisions (over the second and fourth metacarpals for interossei), carpal tunnel release ± midlateral incisions over the hypothenar and thenar eminences, and midlateral release to digits

Tendon transfers[53]

In the oral just relax, take a deep breath in and answer the question.

Definition

- A tendon transfer is the use of a functioning muscle–tendon unit to restore function in a non-functioning muscle or tendon

Indications

- Permanent nerve injury
- Ruptured or avulsed tendon or muscle
- Neuromuscular disease
- The options of nerve reconstruction, tenodesis and arthrodesis have been considered

Principles of tendon transfers

Surgeons planning tendon transfers need to adhere to the following principles whilst tailoring the surgery to the needs and requirements of the indiviudal patient.

- Motivated patient
- Full passive range of movement of joints
- Adequate tissue bed – The soft tissues must be adequate to allow tendon gliding, ideally on a fascial plane. Tendons do not glide well through scar tissue or under skin grafts
- Function – Sacrificing existing function should be kept to a minimum or the function to be gained must be significantly more desirable than that lost
- Sufficient amplitude – The transferred muscle–tendon unit needs amplitude (i.e. excursion) to produce the movement required. Tendon excursion is propotional to the muscle fibre length. It can determined by the amount a muscle can be stretched from its resting position, plus the amount it contracts. The amplitude is typically 33 mm for wrist flexors/extensors; 50 mm for finger extensors and 70 mm for finger flexors
- Adequate strength – The muscle-tendon unit must be able to generate sufficient strength to undertake its action. The force is proportional to the cross-sectional area of the muscle. Power is force × distance. The power of a donor muscle will reduce by one MRC grade after transfer and, if a weak donor is chosen, it may be too weak for useful function after transfer. Therefore, only grade 4 or 5 muscles should be used
- Synergistic action – The transferred tendon works best if the preoperative function is synergistic with its desired action after the transfer, as it will be more readily integrated into normal hand use and easier to rehabilitate
- Direction – Best if the tendon runs in a straight line, which permits it to exert its maximal effect. This is not always possible and some transfers need to go through a pulley or through the interosseous membrane in the forearm. This, however, will weaken the action of a tendon transfer
- Only one transfer for one function per motor unit

Operative technique

- Multiple short transverse incisions
- Careful tendon handling
- Correct tension

Joining the tendons

There are a number of options – Any can be used

- End-to-end anastomosis (allows for a good line of pull for the donor)
- End-to-side (allows for recovery of the recipient muscle-tendon unit – When this may be anticipated)
- Side-to-side
- Tendon weave procedures
  - Pulvertaft weave (concerns over propogation of slits and adjusting tension)
  - Spiral linking
  - Lasso
  - Loop-tendon suture

Transfers for specific lesions

There are numerous transfers described in the literature. It is unnecessary to know them all. We present classic transfers for median, ulna and radial nerve palsies

Median nerve – Low lesion (wrist)

- A low median nerve palsy results in loss of thumb abduction and opposition
- Many patients may have a function thumb with residual function provided by the dual innervation of FPB and continued function of adductor pollicis and flexor pollicis longus
- Each patient should be assessed on an individual basis to see if transfer is of benefit
- To get appropriate line of pull for the transferred tendon a pulley is often required on the ulna side of the wrist
  - Extensor indicis to APB which is divided at the MCP joint, passed proximal to the extensor retinaculum and rerouted around the ulnar side of the wrist and then subcutaneously onto the APB
  - Ring finger FDS is transferred to APB via pulley in the FCU tendon
  - Abductor digiti minimi to APB insertion (Huber transfer). Care must be taken not to damage the tenuous blood supply and innervation during this transfer
  - Transfer palmaris longus and a strip of palmar aponeurosis to APB (Camitz)
- ± MCP or IP fusion
- These tendon transfers allow the thumb to be placed in a more functional position with some dynamic control

Median nerve – High lesion (elbow)

- The deficit is as for low lesion plus loss of flexion of the index and middle fingers
- Thumb flexion
  - Brachioradialis is the transfer of choice
  - ECRL and ECU are alternative options
- Index ± middle finger DIPJ flexion
  - Most common is to suture all four FDP tendons together in the forearm (this does not increase grip strength)
  - An alternative is to transfer ECRL around the radial side of the forearm onto the index FDP and possibly also onto the middle finger FDP
  - The ECRB more centrally based than ECRL so produces wrist extension without radial deviation
- Thumb extension
  - Palmaris longus to EPL
  - If palmaris longus is absent then use FDS from middle or ring finger
- Finger extension
  - FCR/FCU (through interosseous membrane) to EDC
  - Avoid using FCU when ECRL is still functional (as in a PIN palsy) as this can cause excessive radial deviation in the wrist
  - An alternative option is the FDS from middle and ring fingers using a slip of each tendon to a single digit

Examination corner

Hand oral 1

- **What are the indications for a tendon transfer?**
  - Permanent nerve injury
  - Ruptured or avulsed tendon or muscle
  - Neuromuscular disease.
  - The options of nerve reconstruction, tenodesis and arthrodesis have been considered
- **What are the prerequisites for a tendon transfer?**
  - Motivated patient
  - Scar free bed
  - No infection
  - Function to be gained is more useful than that lost
  - Full joint ROM preop
  - Amplitude
  - Strength
  - Synergistic tendon where possible
  - Minimal change in direction
  - One transfer for one function
- **Are there any tendon transfers you are familiar with?**
  - Start simple
  - EI to EPL
  - Radial nerve palsy – Easier in terms of loss of function and the transfers done

Hand oral 2

- **A picture of a patient unable to give a 'thumb's up'**
- **What is happening here?**
  - EPL rupture
- **How does the EPL rupture?**
  - Most common following distal radius fractures
- **Why is it more common in undisplaced distal radius fractures?**
  - Increased pressure within the intact third extensor compartment which causes ischaemia to the tendon. Classically ruptures several weeks after injury.
  - Displaced fractures are thought to cause damage to the third compartment reducing pressure on the tendon so ischaemia does not occur.
- **What are the treatment options?**
  - Leave it alone
  - EI to EPL tendon transfer
  - Arthrodesis.
- **How can you tell preoperatively the patient has an EI to transfer**
- **Pointing with the index finger and the other three digits flexed means EDC is not working and has isolated EI**
- **Describe EI to EPL indicating on the examiner's hand where you would make your incisions?**
  - Transverse over neck of index metacarpal and fourth extensor compartment at the wrist crease and then a

longitudinal incision over the EPL tendon along the thumb metacarpal.
- **How would you joint the two tendons together?**
  - Pulvertaft weave using a 4–0 PDS to hold the two ends together once correctly tensioned.
- **How would you rehabilitate the patient?**
  - 3–4 weeks in a splint preventing tension on the repair then gradual ROM and then strengthing

Hand oral 3

- **What deformities and patient difficulties are seen with a high radial nerve palsy?**
  - Wrist drop and inability to extend fingers or thumb
- **What are the typical causes of a high radial nerve palsy?**
  - Humeral shaft fracture
  - Saturday night palsy
- **What are management options for this palsy?**
  - Splints
  - Nerve repair ± grafting (where indicated)
  - Tendon transfers
- **What tendon transfers are you aware of for this palsy?**
  - Pronator teres for wrist extension
  - Palmaris longus to EPL
  - FCR to Finger extensors

Trigger finger[54]

Background

- Also known as digital tenovaginitis or stenosing tenosynovitis
- Swelling of the superficial and deep flexor tendons at the entrance to the A1 pulley
- The ring finger is most commonly affected followed by the thumb, middle, index and then little finger
- It is associated with underlying conditions such as diabetes, rheumatoid arthritis and gout
- Idiopathic trigger finger is up to 6 times more common in women than men, and is most prevalent between ages 40 and 60

Clinical features

- Clicking
- Pain can be on both active and passive movement
- May demonstrate triggering
- A tender nodule may be palpable at the level of the A1 pulley
- May have a secondary PIPJ contracture

Classification

1. Mild crepitus
2. Abnormal movement
3. Clicking/triggering but not locking
4. Locked but passively correctable
5. Locked and not passively correctable

Aetiology

- Exact aetiology is unclear
- Whether tightness of the fibrous tendon sheath results in inflammation and narrowing of the sheath, or degeneration of the tendon results in tendon enlargement and nodule formation remains unclear
- Indeed, both may occur in response to inflammation
- Often follows trauma or unaccustomed activity

Management

- Steroid injection
  - Place the needle into the tendon and then withdraw slowly until the injection can be easily placed as this will be within the sheath and not the tendon
  - Multiple injections into the same digit are a risk factor for tendon rupture
  - Success rate is about 80% in idiopathic cases and 60% in diabetics
  - Less likely to be effective if symptoms have been present for >6 months
  - Patients should be warned about flare reaction, fat atrophy and skin depigmentation
  - Diabetics may observe a transient rise in serum glucose levels
- Open surgical release
  - Indications
    - Locked digit
    - Failure to respond to two injections
  - Performed as a day case under LA and tourniquet
  - Warn about digital nerve injury (particularly the thumb radial digital nerve) and recurrence
  - Technique
    - Oblique or longitudinal (over the metacarpal head) or transverse incision over the distal palmar flexor crease
    - Blunt dissection
    - Identify the proximal edge of the A1 pulley and release the proximal 1 cm of the pulley
    - If the A2 pulley is released this can result in bowstringing
  - The most common complication of open release is digital nerve injury
  - Diabetes is a poor prognostic indicator, they typically have multiple digitis involved and are especially prone to develop stiffness following surgical release

Oral question

How does the management of a trigger finger differ in a rheumatoid patient?

Causes of a flexion contracture

- Congenital – Camptodactyly
- Skin – Scar contracture
- Fascia – Dupuytren's disease
- Flexor tendon sheath – Fibrous contracture
- Tendon – Subluxation from sagittal band rupture, extensor tendon rupture, locked trigger finger
- Capsular structures – Volar plate shortening
- Block to extension – Arthritis, osteophytes

If the finger is locked in flexion exclude:

- Infection – Kanavel's signs
- Arthritis

Examination corner

Hand oral 1: Clinical photograph of a hand with one of the fingers flexed into the palm (ring or middle)
Spot diagnosis

- **What is the diagnosis?**
  - Most likely to be trigger finger
- **What is the differential diagnosis?**
  - Consider Dupuytren's, PIPJ contracture, trauma
  - Also scar contracture, congenital, arthritic
- **What conditions are associated with trigger finger?**
  - Diabetes, RA, gout
- **What is the management?**
  - Many respond to steroid injections though this is lower in diabetics
- **When would you perform open release?**
  - Failure to respond to injection

Tumours of the hand

Differential

- Ganglion (see earlier)
- Giant cell tumour of tendon sheath (pigmented villonodular synovitis, xanthoma)
  - Second commonest soft-tissue tumour of the hand
  - Firm swelling on volar aspect of the digits
  - 20% arise from joints, and bony erosions are seen in 10%
  - Treat by excision, with a recurrence rate of 10%
- Epidermal inclusion cyst
  - Generally seen on the volar aspect with a small wound on close inspection
  - Painless
- Enchondroma
  - Benign hyaline cartilage tumour found in medullary bone
  - Forty per cent of enchondromas occur in the hand
  - Cause of pathological fracture and bone graft once fracture is united
  - Most occur sporadically although multiple enchondromas are seen with Ollier's disease and in Maffucci's syndrome occur with haemangiomata
- Lipoma
- Neurilemmoma
  - Schwann cell origin and encapsulated so can be shelled out of nerve
- Glomus
  - Pain, tender and cold intolerance
  - Tumour of perivascular temperature-regulating bodies
  - Fifty per cent subungual. MR can be useful. Treat by excision
- Sarcoma
- Acrometastasis – Metastasis to a digit. Very rare. When metastases are seen below the elbow they are mainly lung with renal and breast accounting for most of the rest

Squamous cell carcinoma of the skin over a finger digit

This is one of those esoteric questions that may appear in the FRCS (Tr & Orth) hand oral. They always seem so straightforward afterwards (when discussing them with colleagues) but, for the unprepared candidate in the heat of the exam, you can certainly struggle with them.

A clinical photograph in the hand oral is essentially a spot diagnosis. You may be asked a list of likely causes, investigations and management.

- Risk factors for SCC
  - UV
  - Ionizing radiation
  - Genetic predisposition (e.g. Xeroderma pigmentation)
  - HPV infection
  - Chronic inflammatory disorders
  - Chronic scarring or ulcers
- Diagnosis
  - An erythematous scaly plaque or nodule that may be ulcerated or exophytic
  - Usually itchy and bleeds easily
  - Usually on the dorsal surface of the hand (rare on the palm)
  - Subungual SCCs are uncommon but a paronychia failing to respond should be biopsied
- Management
  - MDT with a dermatologist
  - Cryotherapy
    - Can be used on small (<1 cm) superficial lesions
    - 94% 5-year cure rate

- Wide surgical excision
  - Mainstay of management
  - 95% cure rate

Examination corner

Hand oral: Clinical photograph of squamous cell carcinoma at the tip of a finger with ulceration, necrosis and skin breakdown

- **An elderly gentleman is seen in your clinic with the lesion on his finger. How will you manage it?**
  - Ulcerating lesion with skin breakdown and necrosis over the DP of the ring finger.
  - History and clinical examination looking for involvement of structures and presence of lymph adenopathy
  - Further imaging such as radiograph
- **The bone is not involved. The radiograph is normal. What do you think the diagnosis is?**
  - Main concern is malignancy, with benign lesions .
  - Probably SCC
- **How are you going to manage it?**
  - This needs to be staged
  - Involvement of skin cancer MDT
  - Role of incisional biopsy to gain histological diagnosis – Don't forget to send a sample for microbiology.
  - Once staged and have the biopsy result may require amputation of the part or all the digit

Congenital hand deformities

The examining board is very conscious of its responsibility to provide a fair examination as well as a rigorous one.

Introduction

- In the FRCS (Tr & Orth) examination hand oral any congenital abnormality is fair game for the examiners to show
- There is an opportunity to discuss Swanson's classification of congenital deformities
- If you are very unlucky there will be an opportunity to discuss the development and function of the hand
- Likewise, any congenital abnormality may also appear in the short cases (cleft hand, syndactyly, polydactyly)

Background

About 1 in 600 children is born with a congenital upper limb deformity.

Swanson's classification

1. Failure of formation
   a. Transverse arrest – Amelia
   b. Longitudinal arrest – Radial club hand, cleft hand
2. Failure of differentiation
   a. Soft-tissue involvement
   b. Skeletal involvement
3. Duplication (polydactyly)
4. Overgrowth (macrodactyly)
5. Undergrowth
6. Congenital constriction hand
7. Generalized skeletal anomalies – Madelung's deformity

Radial longitudinal deficiency

- Represents a spectrum of developmental deformities on the radial side of the arm
- Bilateral in up to 75% of cases
- In unilateral cases the opposite thumb is hypoplastic
- The classification focuses mainly on the radiological appearance of the radius; however, the abnormalities can extend well beyond the radius
  - Shortening and bowing of the ulna
  - Absence or hypoplasia of scaphoid and other carpal bones
  - Thumb hypoplasia/absence
  - Thenar muscle hypoplasia/absence
- The abnormalities result in significant functional difficulties
- The forearm is frequently shortened with loss of elbow extension

Classification (Bayne and Klug with additions from James and Goldfarb)

- Type 0
  - Radius normal length with proximal and distal physes
  - Hypoplasia or absence of scaphoid and other carpal bones
  - Can result in radial angulation of the hand and carpus
  - May not require surgery
  - Some require release of radial wrist extensors and release of tight dorsal, volar and radial capsule
- Type 1
  - Distal radius physis is deficient
  - Normal proximal radius
  - Radioulnar synostosis or congenital dislocation of the radial head
  - Lengthening with a frame
- Type 2
  - 'Radius in minature'
  - Entire radius is hypoplastic but both physes are present
  - Bowing of the ulna
  - Distraction lengthening
- Type 3
  - Distal portion including the physis is absent

  - Usually treated with centralisation or microvascular metatarsophalangeal free tissue transfer
- Type 4
  - Absent radius
  - Usually treated with centralisation or microvascular metatarsophalangeal free tissue transfer
- Type 5
  - Abnormal glenoid
  - Absence of proximal humerus

Radiographs

- Important to assess the whole limb from shoulder to fingertip
- Look for short humerus
- Radius hypoplasia or absence
- Ulna bowing
- Absence of carpal bones

Associated conditions with radial club hand

- Fanconi's syndrome
  - Autosomal recessive
  - Aplastic anaemia
  - Need bone marrow transplant
- TAR
  - Autosomal recessive
  - Thrombocytopenia and absent radius
  - Thumb usually present
- Holt–Oram
  - Autosomal dominant
  - Cardiac abnormalities
- VATER
  - **V**ertebral anomalies
  - Imperforate **A**nus
  - **T**racheo-o**E**sophageal aplasia
  - **R**enal anomalies

Management

- Counselling
- Search for associated congenital abnormalities
  - FBC
  - Renal USS
  - Echo
- Surgery usually undertaken at 6–9months
- Centralisation procedure
  - Most popular treatment for types 3 and 4
  - Requires pre-centralisation stretching with a cast or with an external fixator
  - Align carpus on ulna
  - Soft-tissue balancing
- Ulnocarpal arthrodesis
  - Prevents the problems of gradual recurrence of deformity with the centralisation procedure
  - A number of surgeons perform this for a failed centralisation
- Vasularized second MTP joint transfer
  - Designed to reconstruct the osseous support on the radial side of the wrist
- Address the thumb – May require pollicisation

Contraindications for surgery

- Severe neurovascular anomalies
- Stiff elbow
- Good function
- Surgery can be dangerous if there are other congenital conditions

Ulnar club hand

- Less common than radial club hand
- No associated cardiac or haematological problems
- Wrist is stable but the elbow is a problem
- Ulnar digits are often absent and, if present, syndactylized

Swanson's classification of ulnar club hand

1. Hypoplastic ulna
2. Total absence of the ulna
3. Humeroradial synostosis (congenital fusion of the elbow joint)
4. Deficient ulna and absent wrist

Thumb duplication

- Occurs in 1 per 3000 live births
- Approximately 50% are Wassel type 4
- Usually sporadic and unilateral and not associated with syndromes
- Unless Wassel 7 which is associated with
  - Holt–Oram
  - Fanconi's syndrome
  - Blackfan–Diamond syndrome

Classification (Wassel)

- Based on the complete or incomplete duplication of each phalanx
- Uneven numbers are incomplete duplications (bifid) and even numbers are compete bony duplications
- Numbers rise with sequential number of bones involved, starting distally and working proximally

1. Bifid DP
2. Duplicate DP

3. Bifid PP
4. Duplicate PP (most common)
5. Bifid metacarpal
6. Duplicate metacarpal
7. Triphalangism

Principles of management

Forming one thumb out of two

1. Preserving the skeleton of one thumb and augmenting this with soft tissue from the second thumb. Nearly all of one digit is retained and augmented with tissues from the other digit. Duplicate tissues from the 'spare part', which are not used, are excised. This allows for obtaining a good size match and tendon and ligament balance. This is the favoured option. For Wassel 4–6 – Retain ulnar thumb so that integrity of the UCL is maintained
2. The other option is removing the central composite tissue segments from each thumb and combining the two into one (Bilhaut–Cloquet procedure). There are significant problems with stiffness, size, angular deformities, nail scarring and function. This procedure is generally avoided unless there is no other way to obtain a thumb of sufficient size
3. Segmental digital transfer. This is occasionally performed when there is a clearly superior proximal segment on one digit and a clearly superior distal segment on the other digit. Bring the best distal segment of one duplicate on to the best proximal segment of the other
4. Excision. Appropriate where duplication is rudimentary without skeletal elements or the accessory thumb is widely separated from a normal thumb

Examination corner

Hand oral: Picture of a Wassel IV thumb

- **What is this?**
- **How do you classify thumb duplication?**
  - Wassel classification
- **What type is this?**
  - IV
- **Is this associated with any syndromes?**
  - Wassel IV is usually sporadic
  - Wassel VII are associated with conditions
- **What are the principles of surgical management?**
  - Preserve the ulnar thumb but use parts of the radial thumb for reconstruction.

Post-axial polydactyly

This is 10 times more common in African Americans. If it occurs in Caucasians there may be serious associated abnormalities.

Hypoplasia of the thumb

Commonly requires pollicisation of the index finger in the first year of life.

Blauth's classification of thumb hypoplasia (five types)

- Thumb hypoplasia has also been classified by Blauth
- **Type I:** Short thumb, hypoplastic thenar muscle
- **Type II:** Adduction contracture, UCL instability, normal skeleton with respect to articulations
- **Type IIIA:** Extensive intrinsic and extrinsic musculotendinous deficiencies, intact CMC join
- **Type IIIB:** CMC joint not intact
- **Type IV:** Floating thumb
- **Type V:** Complete absence of thumb

Management

- Type I requires no treatment
- Types IIIB–V are treated with pollicisation
- Types II and IIIA are treated with reconstruction, addressing the following issues
  - Stabilisation of the MCP joint
  - Ulnar collateral ligament
  - Web deepening
  - Opponens transfer if opposition is insufficient
  - Extrinsic flexor and extensor exploration with correction of any anomalies

Principles of thumb reconstruction

- Allow opposition
- Must be sensate
- Must have good circumduction at the CMC joint
- Joints must be stable to allow pinch grip

Syndactyly

- Congenital fusion of digits
- Most common congenital malformation of the upper limb
- Affects 1 in 2000
- 50% bilateral
- More common in males
- Third web most common
- Classification
  - Simple
    - Only soft tissue – No bony connections
  - Complex
    - Side to side fusion of adjacent phalanges
  - Complicated
    - Accessory phalanges

- Complete or incomplete
  - Complete extend to the fingertips
- Timing of surgery
  - Usually left until 1 year of age
  - Deal with digits that have biggest length disparity first
  - If multiple digits do a staged approach to avoid compromising vasculature

Cleft hand

- Central deficiency
- At least one digit absent
- Sporadic are usually unilateral, U-shaped and affect the ulnar side
- Familial tend to get worse with each generation and are V-shaped with radial side involvement
- Surgery is usually only required when there is first webspace involvement

Camptodactyly

- Congenital digital flexion deformity
- Usually occurs at the PIP joint of the little finger
- Affects <1% of the population
- Can be familial
- No functional significance in the majority
- Can be static or progressive
- There are two types
  - Type 1 infantile type
    - Seen in infancy
    - M = F
  - Type 2 adolescent type
    - F > M
    - Frequently bilateral but not symmetrical
    - Familial deformity
    - Increases during adolescent growth spurt

Aetiology

- The deformity has been attributed to the following abnormalities
  - General absence of development of all tissues of the digit
  - Abnormal lumbrical origin
  - Contracture of the collateral ligaments of the digits
  - Flexor and extensor tendon imbalance
  - Abnormal FDS origin or insertion

Management

- Reassurance and stretching
- Avoid surgery if at all possible
- Surgery may be indicated for a flexion contracture of >60°
  - Release skin, fascia, tendon sheaths, intrinsics, collateral ligaments and volar plate
  - Lengthen the FDS tendon

Clinodactyly (lateral plane deformity)[a]

- Radioulnar curvature of the little finger
- More common in males
- Usually bilateral
- There are three types
  - I – Minor angulation, normal length (very common)
  - II – Minor angulation, short phalanx, associated with Down's syndrome
  - III – Marked deformity, associated with delta phalanx. A delta phalanx is a wedge-shaped phalanx with a C-shaped physis

Management

- If a delta phalanx replaces a normal bone, manage by an opening reverse osteotomy
- If delta phalanx is an extra bone, then excise it (Figure 21.14).

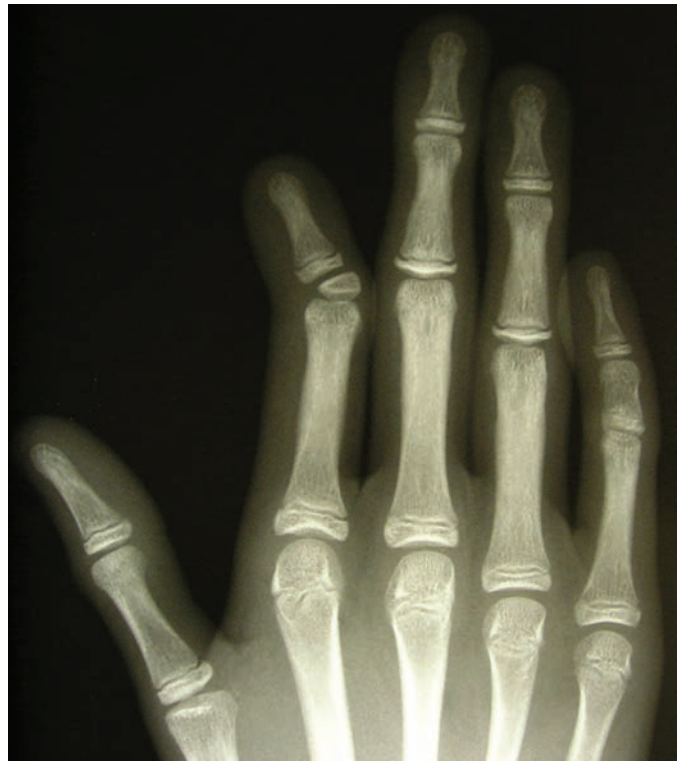

Figure 21.14 Delta phalanx

[a] Clino has 'C' and 'L' = lateral plane; Campto has 'C' and 'A' = AP deformity.

Kirner's deformity

May be mistaken for clinodactyly. Volar and radial curvature at the distal phalanx of the little finger. Usually autosomal dominant. Frequently bilateral.

Management

Surgery is generally for cosmetic reasons only. Avoid when the growth plate is open – Corrective osteotomy.

Examination corner

Hand oral 1: Clinical photograph of duplicated thumb

- **Diagnosis**
- **How do you classify duplicated thumb (Wassel)?**
- **Principles of treatment and treatment of type shown**

Hand oral 2: Clinical photograph of syndactyly

- **Asked for a classification system for congenital hand deformities (Swanson's)**

Hand oral 3: Clinical photograph of camptodactyly (usual questions)

- **What is this deformity?**
- **What causes the deformity?**
- **How is it managed?**

Hand oral 4: Clinical photograph of syndactyly

- **Asked for classification of congenital hand deformities.**

Hand oral 5: Clinical photograph of a radial club hand

EXAMINER: You are called to the paediatric ward because a newborn baby has the above condition. What is this deformity? *(Spot diagnosis.)*

EXAMINER: OK – How do you manage it?

CANDIDATE: I thought I answered the questions fairly well but the discussion seemed to be going around in a bit of a circle. The examiner seemed a bit unhappy and eventually came out with what they really wanted. Somewhere along the way I should have mentioned that there was a high incidence of other congenital deformities with this condition and I might want to consider arranging a renal and cardiac ultrasound!

(Candidate pass.)

Hand oral 6: Clinical photograph of radial club hand

EXAMINER: What is the diagnosis and how do you manage it?

CANDIDATE: This is a clinical picture, which is suggestive of bilateral radial hemimelia. Both forearms are short; there is radial and volar deviation at the wrist and hypoplastic thumbs. The fingers also appear poorly developed.

There is no point in beating around the bush as the diagnosis is obvious. However, after you have given the diagnosis continue to describe the clinical features on the photo.

Work through an answer of the management options beforehand rather than jumping about with this one as I did: I first mentioned that it will be very upsetting to the parents and one would need to spend time with them.

I did not mention a search for associated congenital abnormalities, which the examiners pressed me about. I suggested observing the condition initially.

For mild cases manipulation and control with strapping may be all that is required.

I mentioned the words *'centralisation of the wrist/forearm deformity'* and *'thumb reconstruction'* and the examiners were happy with this and were not interested in any further details whatsoever.

When discussing management options try to avoid jumping straight in with surgery.

(Candidate pass.)

Hand oral 7: Clinical photograph of radial club hand

- **What is the diagnosis and how do you manage it?**
  - Spot diagnosis and very quick. The examiners were not looking for a detailed answer. The whole thing took <30 seconds maximum before we moved on to another clinical photograph.

Hand oral 8: Radiograph of obvious radial club hand

Spot diagnosis.

Constriction bands

Streeter's dysplasia. More common in fingers and toes. May be deep with distal oedema. Congenital amputation. Treat by Z-plasties.

Miscellaneous

- Arthrogryposis – Stiff joints. Absence of skin creases
- Symphalangism – Stiffness of PIP joints ± ankylosis
- Congenital trigger thumb – Common, palpable swelling – Notta node. Most resolve by 1 year – Surgical release if continues
- Madelung's deformity – Growth arrest of the volar-ulnar distal radius

Hand infections[55–57]

Recognition and prompt intiation of appropriate treatment is required to prevent permenant impairment.

Aetiology

- Most commonly occur following trauma
- Human bites
- Animal bites
- Illicit drug use
- Post surgery

Pathogens

The history is useful in helping to elicit the most likely organism and, thus, the most appropriate antibiotic treatment. The vast majority of hand infections are bacterial in origin with *Staphylococcus aureus* the most common pathogen. Contaminated wounds from agriculture injuries often are affected by multiple organsisms including anaerobic and gram negative bacteria. Children can also present with unusual pathogens including oral flora, *Haemophilus influenzae* and *Pseudomonas sp*. Bites by human often involve *Eikenella corrodens*. Immunocompromised patients are predisoposed to opportunistic infections from mycobacteria, fungi and viruses.

History

- Penetrating injury, fight bite – Note where
- When
- Pain
- Loss of function
- Medical history – Diabetes
- Tetanus status
- Consider HIV and hepatitis status

Examination

- Temperature, pulse and respiration
- Examine for puncture wounds
- Swelling
- Posture of the hand
- Warmth
- Tenderness
- Test motor and sensory function
- Examine the arm for spreading lymphangitis
- Epitrochlear lymph nodes drain the ring and little finger, axillary nodes drain the radial digits
- Cellulitis resolves with antibiotics only and elevation

Investigations

- FBC, ESR, CRP
- Blood cultures
- Wound swab/pus sample
- Radiographs

Specific infections

Paronychia/eponychia

- Infection of the nail fold bordering the nail plate
- Usually occurs followig disruption of the seal between the nail fold and nail plate folowing trauma such as nail biting or manicure. This allows entry of bacteria, usually *S. aureus,* though mixed flora can be seen with nail biters
- Early presentation before the onset of fluctuance may be managable by elevation and oral antibiotics
- The presence of fluctuance necessitates incision and drainage which may require temporary nail removal

Felon

- This is a localized compartment syndrome contained by the fibrous septae connecting the pulp to the distal phalanx
- Usually occurs following penetrating trauma
- Present as a tense, tender, swollen and erthythematous pulp
- Urgent decompression through a vertical midline incision distal to the skin crease, taking care to break down all containing septa is required to stop the vicious cycle of inflammation, veous congestion, venous compromise, pulp necrosis and abscess formation

Deep space infection

There are several deep anatomical spaces within the hand and wrist that can contain infection following trauma: Webspaces, thenar, mid palmar, hypothenar, radial and ulnar bursae, space of Parona and dorsal subcutaneous and subaponeurotic spaces.

Thenar space

- Thumb typically held abducted away from the palm with pain over the adductors on extension or opposition
- Drainage requires incisions to volar thenar crease and drosal webspace to drain both the retro-adductor and thenar spaces

Mid palmar space infections

- Space lies between the metacarpals and palmar aponeurosis
- Seperated form the thenar space by an oblique septum that connects the third metacarpal with the palmar aponeurosis
- Clinically this presents with tense and painful erythematous swellings on both palmar and dorsal surfaces of the hand
- Loss of active motion of the middle and ring fingers
- Drain through a curved incision beginning at the distal palmar crease, extending ulnar-ward to just inside the hypothenar eminence

Hypothenar space

- Infection confined to the hypothenar eminence muscles
- Contained in the space by the septum connecting the palmar aponeurosis to the fifth metacarpal
- Little finger typically held in maximal abduction
- Drainage is by a longitudinal incision down the ulnar border between glabrous and non-glabrous skin

Radial and ulnar bursae including space of Parona

- The radial and ulnar bursae are proximal continuations of the flexor sheaths to the thumb and little finger respectively
- These two bursae can communicate with each other through the space of Parona which lies between pronator quadratus and FPL

- Classically present with a 'horseshoe abscess'
- Requires drainage of both digits flexor sheaths and Parona's space
- To drain the radial bursa, make a lateral incision over the proximal phalanx of the thumb and enter the sheath. Introduce a probe and push it towards the wrist
- Make a second incision where the probe is palpable just proximal to the wrist. Irrigate with a cannula
- To drain the ulnar bursa, open it distally on the ulnar side of the little finger, and through a transverse incision just proximal to the wrist and lateral to FCU

Web space infection

- The subfascial web space is a fat-filled space situated on the palmar surface of the hand and interdigital area
- The limits of the web space are the natatory ligaments distally, the deep attachment of the palmar fascia proximally and its attachment to the tendon sheath laterally
- The infection usually arises from a wound to the skin between the fingers though can occur from contiguous spread from a pulp infection or via the lumbrical from a deep palmar space infection
- Web space infections may lead to a collar stud abscess
- Drainage is via two longitudinal incisions, one dorsally and one ventrally, but the web should not be incised

Dorsal subcutaneous and subaponeurotic spaces

- Dorsal swelling from infection can occur either primarily from infection in the dorsal space (superficial or deep to the extensors) or from a palmar infection
- The strong fascial components on the palmar side in comparison with the loose mobile dorsal skin favour the palmar infection to balloon dorsally and so careful examination of the palmar surface should be undertaken when assessing any dorsal infection
- Dorsal spaces are best drained via longitudinal incisions centred over second and fourth metacarpals
- Subaponeurotic space collections require incision of the dorsal fascia between the extensor tendons down to the interosseous fascia

Flexor sheath infection

Suspicion of this infection mandates immediate surgical drainage as infection within this space destroys the synovial gliding surface resulting in adhesions or tendon rupture.

Anatomy

The flexor sheaths of the index to the ring fingers start from the proximal edge of the A1 pulley. The little finger flexor sheath connects to the ulnar bursa (contains flexor tendons 2–5 deep to the flexor retinaculum).

Clinical features

Typically occurs following penetrating trauma though haematogenous spread can occur and gonococcal infections should be considered in young patients without a history of penetrating trauma.

Kanavel's four cardinal signs:

- Finger held in a flexed position
- Sausage digit (symmetrical swelling)
- Severe tenderness along the tendon sheath
- Pain on passive extension of the finger

Management

- Intravenous antibiotics and prompt surgical drainage
- Make a transverse incision over the distal finger crease or a midlateral incision at the level of the middle phalanx, and open the tendon sheath and pass a catheter
- Make a second transverse incision at the level of the distal palmar crease and the sheath just proximal to the A1 pulley and flush through until clear
- Some advocate continuous catheter irrigation on the ward following surgery. The evidence that is beneficial is inconclusive and there are reports that the catheter blocks and/or the skin becomes macerated from the continual irrigation
- Severe cass with associated necrosis require an open exploration to debride devitalized tissue. This can be done either through a midaxial or Brunner incision. The Brunner incision is more straightforward with lower risk to the neurovascular bundles though the midaxial gives a more reliable flap for later closure

Osteomyelitis

- This is an infrequent complication if hand infections are treated appropriately
- More common from direct innoculation or spread from local infection, particularly in diabetics
- Typically it is only suspected after failure of antibiotic therapy or repeat infections
- Requires surgical debridement of all affected bone and sequestra and prolonged antibiotic therapy
- If amputation is necessary, it should be done at the joint proximal to the infected bone or the infection will not clear
- NB. Infection of the finger pulp may erode the distal phalanx, but may improve when the overlying abscess is drained

Human bite ('fight bite') injuries

- These are common with the patient presenting with a history of punching someone and a wound over the MCP joint
- Radiographs should be taken to exclude fracture or the presence of a fragment of tooth
- Patients may deny the mechanism of injury but wound over the MCP joint should be assumed to have penetrated the joint and mandate a formal arthrotomy and washout under general anaesthetic
- Pathogens include the normal flora of the mouth, which includes 42–190 different organisms. The most

common infecting organism is still *S. aureus*; other common organisms include streptococci, *Eikenella corrodens, Enterobacter, Proteus* and *Serratia* spp

Septic arthritis

- As with any other joint this is a surgical emergency, as untreated the cartilage is destroyed by the lysosomal activity of the bacteria
- It can occur from direct innoculation, contiguous or haematogenous spread
- Most valuable sign if pain on passive or active motion of the affected joint from its position of maximal volume (MCP joint extension, IP joint 30° flexion)
- Any suspicion is better managed with surgical exploration rather than expectantly
- MCP joint should be approached with a dorsal longitudinal incision over the joint and a longitudinal split between the extensor tendon and saggital band
- PIP joint can be approached via a dorsolateral incision with the joint entered between the central slip and lateral bands

Examination corner

Hand oral 1: Clinical photograph of paronychia?

- **What are the common causative organisms?**
  - Often *S. aureus* though mixed flora in nail biters is not uncommon
- **How would you manage this patient?**
  - If there is pus – Let it out
  - Antibiotic therapy
  - If no pus warm soaks may help witht the antibiotics.

Hand oral 2: Clinical photograph of fight bite injury to the MCP joint of the index finger

- **How would you assess this injury?**
  - History. Wounds over the MCP joint should raise the suspicion of a fight bite until proven otherwise. Must specifically ask the patient.
  - Wounds – Depth and exact location, tendon function
- **What organsisms so you need to cover?**
  - The most common infecting organism is still *S. aureus*
  - Other common organisms include streptococci, *Eikenella corrodens, Enterobacter, Proteus* and *Serratia* spp.
- **What is the role or surgery?**
  - Hard to disagree with stating you would explore, debride and washout all these wounds in theatre – Not infrequently can see bits of tooth in the joint that are not visible on radiographs.

Tuberculous dactylitis

Inflammation of the phalanges or the metacarpals. The bone becomes enlarged, spindle-shaped and, in the case of tuberculous dactylitis, is painful. The skin overlying the affected bone appears smooth and shiny. With further progress the skin may become red, tender and frequently an abscess forms.

In syphilitic dactylitis the swelling is painless. Sickle cell disease causes dactylitis because of infarction of bone secondary to thrombosis of the nutrient artery.

Radiographs in tuberculous dactylitis

- Soft-tissue swelling
- Cortical thinning
- Medullary destruction
- Periosteal reaction

Management

Curettage for culture material followed by antituberculous chemotherapy and splinting.

Differential diagnosis

- Pyogenic infection
- Syphilis
- Enchondroma
- Mycetoma (Madura hand)
- Multiple xanthomatosis
- Sickle cell disease

Diagnosis can be confirmed by biopsy. In spina ventosa there is grossly swollen, spindle-shaped bone.

Triangular fibrocartilage complex (TFCC) lesions

There are several topics that you are extremely unlikely to be asked about in the FRCS (Tr & Orth) exam. It is always risky saying 'never' and that's why one learns these topics on the off chance that they in fact turn up. Although I doubt very much that you will be asked about TFCC lesions, one should know about the anatomy of the TFCC, the two subgroups of TFCC lesions (Table 21.3) and the various management options available.

Table 21.3 Classification of TFCC lesions

Class 1: Traumatic injuries	
1A	Central perforation or tear
1B	Ulnar avulsion with or without ulnar styloid fracture
1C	Distal avulsion (origins of ulnolunate and ulnotriquetral ligaments)
1D	Radial avulsion (involving the dorsal and/or volar radioulnar ligaments)
Class 2: Degenerate TFCC tears	
2A	TFCC wear (thinning)
2B	2A plus lunate and/or ulnar chondromalacia
2C	TFCC perforation plus lunate and/or ulnar chondromalacia
2D	2C plus lunotriquetral ligament disruption
2E	2D plus ulnocarpal and DRUJ arthritis

Introduction

TFCC is important in loading and stabilising the DRUJ. Tears are a cause of ulnar-sided wrist pain and are classified according to their location.

Anatomy of TFCC

- Dorsal and volar radioulnar ligament
- Articular disc
- Meniscus homologue
- Ulnar collateral ligament
- ECU subsheath
- Origins of the ulnolunate and lunotriquetral ligaments

The periphery is well vascularized, whereas the radial central portion is relatively avascular, thin and prone to degenerative changes. Peripheral tears are usually traumatic, whilst central tears are generally degenerative and are often found in association with ulnar-positive variance.

Clinical presentation

- Ulnar wrist pain and restriction of forearm rotation
- Tenderness over TFCC
- Pain with ulnar deviation of carpus and compression

Investigations

Radiographs

- TFCC is not visualized on plain radiographs
- May show ulnar-positive variance
- Localized subchondral defect of the lunate caused by impaction on the distal ulna

Arthrography

- Leakage of dye distally

MRI

- Offers improved accuracy in the diagnosis of TFCC tears

Diagnostic arthroscopy

- Gold standard

Management

Traumatic injuries

All acute traumatic lesions of the TFCC are initially managed non-operatively with immobilization and NSAIDs.

In general:

1A – Arthroscopic resection of the torn portion (Figure 21.15 a–c)
1B – Arthroscopic repair
1C – Arthroscopically assisted limited open repair
1D – Partial excision or direct repair with ulnar shortening

Degenerative tears

The abnormalities involve a pathological progression of disease associated with ulnar-positive variance and impaction between the ulnar head and the proximal pole of the lunate. Non-operative treatment is tried first with rest, immobilization and steroid injections.

Surgical treatment is aimed at decompressing the ulnocarpal articulation. Traditionally, surgery involved diaphyseal ulnar shortening with the added advantage of tightening the ulnocarpal ligaments and is particularly recommended when concomitant LT instability is present.

Other options include the wafer (2A–2C) or arthroscopic wafer (2C) resection of the ulnar head. Positive ulnar variance >2 mm is a contraindication to wafer resection and is best managed with diaphyseal shortening. Class (2E) lesions are managed with either a limited ulnar head resection such as a Sauve–Kapandji procedure (arthrodesis of the DRUJ and creation of a pseudoarthrosis at the level of the ulnar neck).

Darrach's resection of the distal ulna is considered a salvage procedure because of the concerns regarding impingement and instability of the residual ulnar stump.

Examination corner

Hand oral 1: Management of TFCC lesions

Most TFCC tears respond to conservative management – Splintage, steroid injections and restriction of activities.

Peripheral tears may be repaired arthroscopically or at open operation, although this procedure is not easy as the TFCC is small and exposure is limited.

Large central flap tears will not heal; they may be debrided arthroscopically but this is ineffective if there is ulnar impaction as the ulna will still abut on the lunate.

Ulnar impaction is treated with ulnar shortening, either a shaft osteotomy or trimming of the distal end of the ulna beneath the TFCC (wafer procedure of Feldon). The ulna needs only to be shortened by 1–2 mm and it is not necessary to repair or resect the TFCC tear.

Hand oral 2: arthroscopic picture of a central TFCC tear

- **What is the diagnosis?**
- **How are these tears classified?**
  - Traumatic and Degenerative
  - Then detail then actual parts of each
- **Which tears would you repair?**
  - Peripheral tears that lead to an unstable DRUJ
- **How would you repair a tear?**
  - Arthroscopic or open
  - Open allows the knot to be placed whilst ensuring the dorsal sensory branch of the ulnar nerve is not going to be injured
- **How would you treat this patient?**
  - Debridement to of the central flap to a stable rim
  - Assess the distal ulna – Consider either a wafer resection or an ulnar shortening

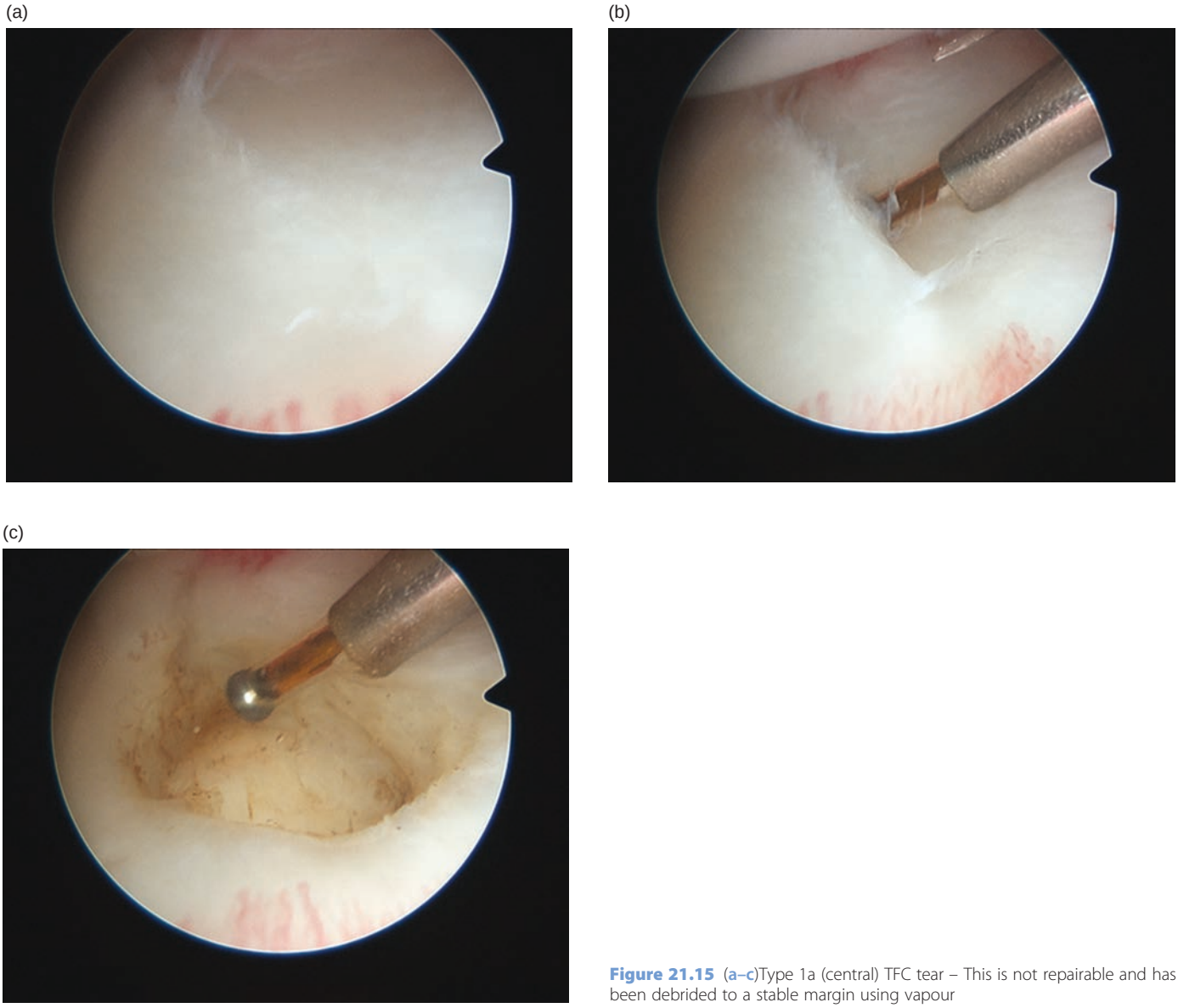

Figure 21.15 (a–c)Type 1a (central) TFC tear – This is not repairable and has been debrided to a stable margin using vapour

Wrist arthrodesis

Indications

A painful or unstable wrist joint with advanced destruction caused by:

- Osteoarthritis
- Rheumatoid disease
- SNAC/SLAC wrist
- Salvage of failed wrist arthroplasty
- Salvage in Kienböck's disease

Contraindications

- Infection
- Lack of soft-tissue coverage

This procedure is more beneficial for young, active patients or middle-aged patients but is not for elderly patients.

Preoperative considerations

In the rheumatoid wrist the application of a dorsal plate increases the chances of a dorsal wound dehiscence.

Range of movement of other joints

Remember that the elbow and shoulder joints will have to compensate for loss of wrist motion.

Surgical details

Dorsal approach in the wrist

With a severe deformity, consider a wider exposure to the first dorsal compartment to allow excision of the radial styloid. The

individual carpal bones and distal radius are exposed with the wrist in hyperflexion. Articular cartilage is removed with a Rongeurs. It is important to treat the long finger CMC joint in the arthrodesis or a painful non-union may occur, whereas most surgeons usually prefer to spare the index CMC joint to allow its participation in power grip.

Ulnar head

In RA consider resection of the ulnar head, and then using it for a bone graft.

Position of arthrodesis

With the non-RA wrist

Place in 20° of dorsiflexion because this position allows for power gripping. Maximum grip is generated in 35° of dorsiflexion but this interferes with ADLs.

In the rheumatoid wrist

A neutral or a flexed position is more desirable. In the frontal plane a position of 5–10° of ulnar deviation is preferred to counterbalance the zig-zag collapse and ulnar drift. Despite the usual recommendations, some patients will prefer slightly more flexion or extension in the wrist. If possible, consider casting the wrist before surgery in extension and the neutral position to determine which position is more comfortable for the patient.

Methods of fixation

Steinmann pin fixation

Through the third metacarpal into the radius or via the second or third web space of the hand. Plaster for 8 weeks to prevent rotation.

AO wrist fusion plate

This is an 8-hole titanium plate with 2.7-mm screws inserted into the distal four holes and 3.5-mm screws in the proximal four holes. To have the wrist in 20° of dorsiflexion, a contoured plate is necessary. Lister's tubercle will have to be removed to achieve a flat bed for plate application and use as a bone graft. Excise all cartilage and insert bone graft (do not forget the third carpometacarpal joint). Most often the plate is applied to the long metacarpal so that three cortical screws can be inserted into the metacarpal and four screws into the radius (often a screw will also be inserted into the capitate).

Postoperative routine

- Volar splint for 6 weeks
- Union is usually achieved by 3 months
- Plate is not removed unless it causes symptoms

Complications

- Extensor tenosynovitis is the most common complication and is related to a prominent dorsal plate and screws
- Skin necrosis
- Infection
- Transient nerve palsy
- Persistent pain (exclude non-union)

Complex regional pain syndrome (CRPS)[58]

Definition

CRPS is not a disease, but a collection of symptoms without a known cause, without a clear pathophysiology and without a cure. The condition comprises of four cardinal features: Pain out of proportion to the degree of injury, swelling, stiffness and vasomotor instability. It is divided into two types: type 1 where there is no obvious nerve damage and type II where there is identifiable nerve damage.

Diagnostic criteria

The following 'Budapest Criteria' should be present at the time of evaluation to make the diagnosis:

1. Continuing pain disproportionate to the inciting event
2. Symptoms (at least 1 in 3 of the following 4 categories)
   I. Sensory (hyperaesthesia/allodynia)
   II. Vasomotor (temperature/colour changes – Asymmetrical)
   III. Sudomotor (oedema/sweating – Asymmetrical)
   IV. Motor/trophic (Decreased ROM, weakness tremor/dystonia, trophic changes in skin, hair or nails
3. Signs (at least 1 in 2 of the following categories)
   I. Sensory (hyperaesthesia to pin prink, allodynia to light touch)
   II. Vasomotor (evidence of temperature or colour asymmetry)
   III. Sudomotor (evidence of oedema/sweating asymmetry)
   IV. Motor/trophic (evidence of decreased ROM, weakness, tremor, dystonia, trophic changes to skin, hair or nails)
4. No other diagnosis explaining the signs or symptoms

Investigations

Primarily this is a clinical diagnosis.

Radiographs can show diffuse osteopenia but this is not a sensitive test and, thus, along with other imaging modalities play no role in the diagnostic criteria.

A bone scan may show increased uptake of isotope in early CRPS but later on the bone scan returns to normal.

- Diagnostic sympathetic block (stellate ganglion)

Aetiology

- Trauma is the most common, particularly wrist fractures
- Iatrogenic such as carpal tunnel decomrpession and dupuytens surgery
- Ischaemic heart disease and myocardial infarction
- Cervical spine or spinal cord disorders

- Cerebral lesions
- Infections
- In some patients a definite precipitating event cannot be identified

Theories

The true cause of CRPS is controversial but the evidence revolves around two theories or what the intiating event causes.

1. Inflammatory. The early phase of the condition is that of an inflammatory response and some believe that CRPS is an exaggerated local inflammatory response to trauma
2. Neurological. Neuralgic pain associated with CRPS may be explained by damage to small fibre neurons. A-delta and C-fibres transmit information from mechanical, thermal and chemical stiumli. They release vasoactive substances such as substance P which is found in CRPS

Natural history

- 90% of CRPS following distal radius fractures resolved within 2 years
- Some, however, do have persistent signs and symptoms
- It is believed to be reactivated or recur following further trauma or surgery. This remains unproven

Prevention

- Two RCTs that have found a decrease in CRPS of 8–15% with distal radius fractures by administration of 500 mg of vitamin C per day
- Some studies have suggested a decreased risk with regional rather than general anaesthesia but this is not proven

Management

- Good analgesia including centrally acting medication such as gabapentin
- Physiotherapy
  - Hand therapy is the mainstay of treatment
  - Should continue until symptoms have resolved
  - Aim is to prevent late contractures, secondary weakness and reduce cortical remodelling
- Mirror visual therapy
  - Patient exercises than normal and affected hand along but sees the affected hand moving normally due to the mirror image of the normal one
- Regional blocks with sympathetic blockade are often used but no strong evidence that they work

Splinting of the hand and wrist

A splint may appear in the clinicals or be used as a prop in the orals. Therefore, a basic understanding of principles of use, materials and indications is needed.

Definition

- Type of orthosis (external device to support a body part)

Uses

- Immobilize, protect, controlled mobilization, prevent deformity

Types

- Static
- Dynamic

Materials

- Plaster/synthetic
- Thermoplastic – Become malleable with heating (water, bath)
- Examples:
  - Capener – Provides passive extension force (three-point loading) to PIPJ for boutonnière, but allows active flexion against resistance
  - Murphy rings – Statically limit PIPJ hyperextension and allow flexion for swan-neck deformities
  - Stack – Static extension to DIPJ for mallet finger
  - Dynamic outrigger – For extensor tendon repair/MCP joint replacements; allows passive extension/active flexion

Miscellaneous oral questions

It is impossible to cover every possible hand oral topic that could be asked in the FRCS (Tr & Orth) exam in detail. Below, however, are some less known questions that candidates may be asked.

Examination corner

Hand oral 1

Clinical photograph of a large, well-circumscribed, lobulated, firm mass situated over the volar surface of the long finger (Figure 21.16). Painless but slowly enlarging. Radiographs show no bony abnormality.

- **What is the diagnosis and management?**
  - Spot diagnosis of pigmented villonodular synovitis (giant cell tumour of tendon sheath).

After a ganglion, this is the second most common tumour found in the hand and a classic favourite with examiners.

A benign lesion, radiographs are usually normal but may show soft-tissue swelling or may show local pressure effects on bone.

Localized form PVNS that arises from a tendon sheath or adjacent joint. A diffuse form of giant cell tumour not seen in the hand that occurs in areas adjacent to large weight-bearing joints. This is synonymous with the extra-articular form of PVNS. Local complete excision is recommended but the condition has capacity for local reoccurrence (10–20%).

Must discuss differential diagnosis of a soft-tissue swelling of the digit.

Hand oral 2: clinical photograph of subungual exostosis

A spot diagnosis – Move on to it if you know it; if not, a safe approach is to describe what you see and then go through a differential diagnosis.

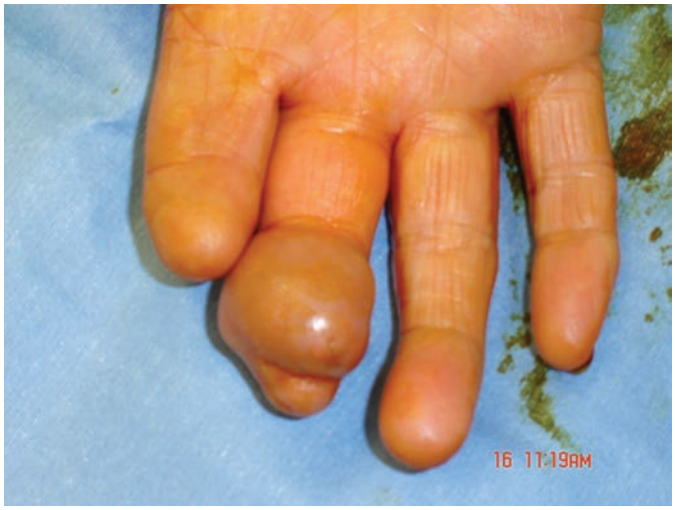

Figure 21.16 Clinical photograph of hand swelling

This is a bony diverticulum from the terminal phalanx and has the typical cartilaginous cap seen with exostosis elsewhere in the body. This elevates the nail plate, with subsequent ridging if the germinal matrix is involved. It is thought the aetiology is probably traumatic but this is in dispute. The treatment is surgical excision. It is necessary to first remove the nail, then the nail bed is split and elevated, the exostosis removed and the nail bed can be sutured back into place.

Differential diagnosis includes:

- Glomus tumour (Masson's tumour) – Tumour of glomus body which regulates blood flow and temperature; 50% subungual. Triad of pain, exquisite tenderness and cold intolerance
- Enchondroma
- Subungual inclusion dermoid cyst following an old penetrating injury (look for an overlying scar, which may be very small)
- Amelanotic melanoma, which usually presents as a granulation in the centre of the nail, but this diagnosis must be considered if there is swelling beneath the nail. The diagnosis may be delayed as it may mimic infection. Nodal involvement is present in 40% of patients at first presentation. A biopsy should be taken, including sampling of the lymph nodes and then the tumour can be staged before definitive treatment, which usually consists of ray amputation and chemotherapy. A clinical picture of subungual melanoma is sometimes shown in the hand oral

Hand oral 3: Pigmented lesion under nail bed

Differential diagnosis: Melanoma, subungual haematoma, glomus tumour.

Hand oral 4: Fingertip injury

- Fingertip injuries of the distal phalanx
- Management in a young, female non-smoker, dominant hand, tip available

Hand oral 5: De Quervain's disease. Clinical photo of swelling over radial styloid

- What is the diagnosis?

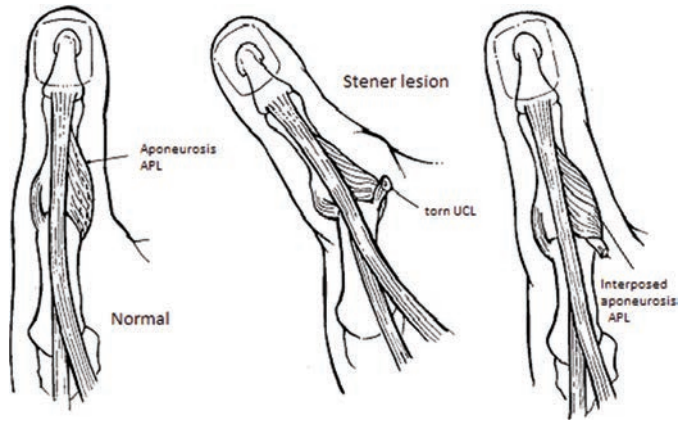

Figure 21.17 Stener lesion – Clinically the displaced proximal end of the UCL can be felt over the metacarpal head

- What clinical tests can be undertaken?
- What are the other causes of radial-sided wrist pain?
- What are the treatment options?
- Which tendons are involved?

Hand oral 6: Bennett's fracture

- Why is it unstable (i.e. what is the deforming tendon)?
- Management
- What size of K-wire is used for fixation?

Trauma oral 1: UCL injury of the thumb

- Stener lesion (Figures 21.17, 21.18)
- Approach
- Complications

Trauma oral 2: rugger jersey finger (Figure 21.19)

- Classification
- Discussion of Brunner incisions
- Pull-out suture

Trauma oral 3: clinical photograph of a hand after a crush injury (Figure 21.20)

- Differential diagnosis

This includes compartment syndrome and infection

- **How assess?**
  - Look for any breaks in the skin or discharge. Check if hand is perfused – Skin colour/warmth, radial pulse, capillary refill time. Feel if swelling soft and fluctuant, or hard. Ask patient to move their fingers. Check passive stretch of digits. Request urgent radiographs to look for any fractures
- **Management?**
  - If compartment syndrome of the hand is suspected urgent fasciotomies are required. This is performed under a general anaesthetic. An arterial pressure transducer can be set up in theatre and compartment pressures in the interosseii, thenar and hypothenar muscles recorded. A pressure >30 mmHg confirms the diagnosis but you will proceed to fasciotomies based on the clinical suspicion. A full release can be achieved using dorsal incisions over the second and fourth metacarpals, a lateral incision to the thenar eminence and a medial incision to the

Figure 21.18 Testing the integrity of the UCL. (Picture Courtesy of ©DonaldSammut 2014)

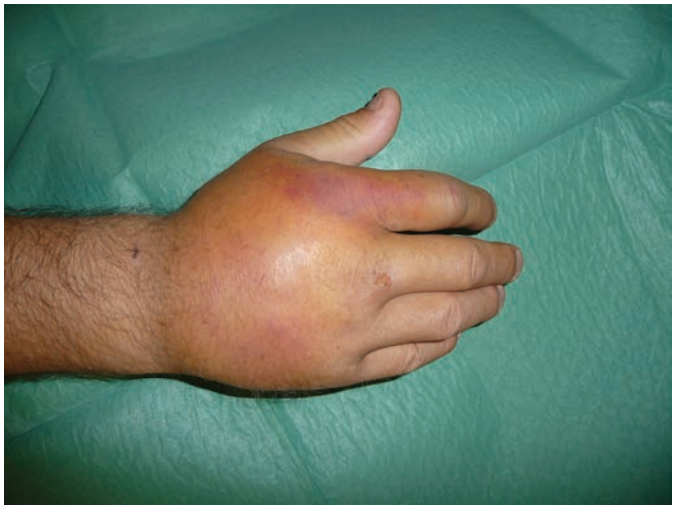

Figure 21.20 Clinical photograph of a hand after a crush injury

hypothenar eminence. If the digits are grossly swollen these can be released through midlateral incisions.

Trauma oral 4: Radiograph of a motor cyclist's wrist after an accident (Figure 21.21)

- Pathogenesis

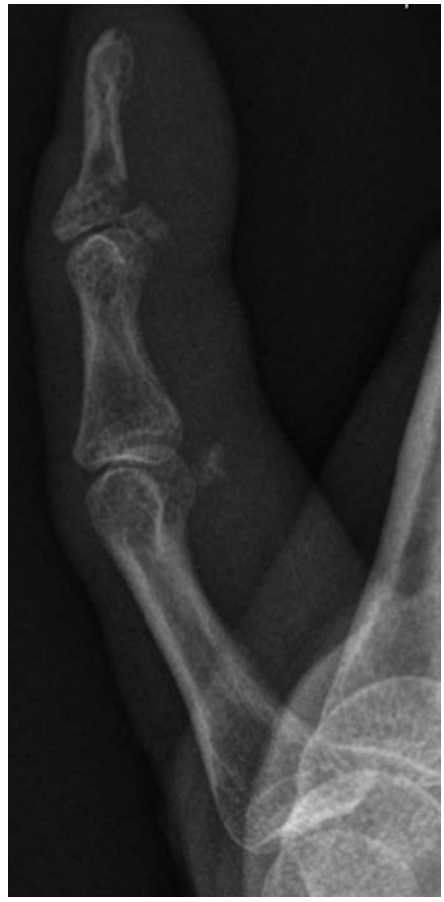

Figure 21.19 Radiograph of little finger of patient following a fall onto her hand

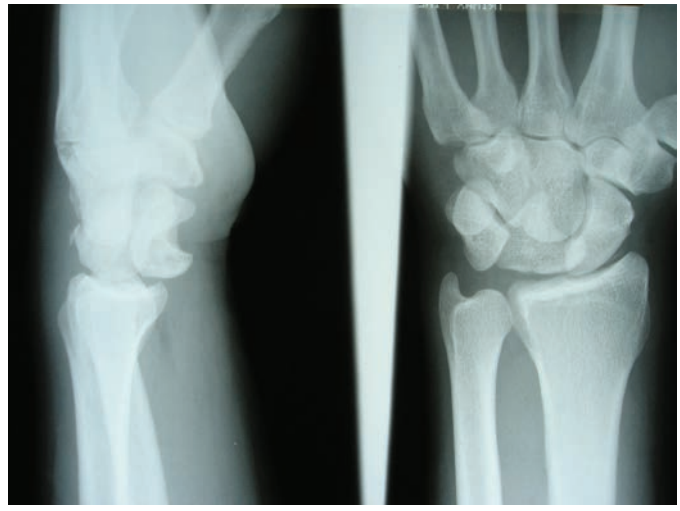

Figure 21.21 Radiograph of a motor cyclist's wrist after an accident

These show a lunate dislocation. This can occur with hyperextension of the wrist leading to tearing of the perilunate intrinsic carpal ligaments starting on the radial side. The most severe injury can lead to extrusion of the lunate through the Space of Poirier – A weak area in the volar extrinsic carpal ligaments. This is a Mayfield grade IV injury, the most severe degree of injury[59]. Mayfield described the pathomechanics of perilunate injuries with grade I is disruption of the scapholunate ligamentous

complex, grade II disruption of the lunocapitate connection and grade III the lunotriquetral connection is violated.

- **How manage?**
  Check for and document any median nerve symptoms. This injury requires prompt reduction in theatre. This can be achieved closed by hyperextension of the wrist and pressure over the lunate. The patient can be referred to a hand surgeon for repair of the intercarpal ligaments through a dorsal approach and temporary K-wire fixation in the next few days.

Further questions

- **Q. Presentation?**
  - A.
- Significant wrist pain
- Swelling hand
- Limited hand and wrist movement
- Tingling and numbness of the fingers (acute onset carpal tunnel syndrome)
- Digits are often held in a semiflexed position and passive extension causes pain
- There is abnormal carpal alignment and crepitus may be felt is there is an associated fracture

- **Q. Timing of intervention?**
  - A. The scenario would be the injury occurring in the middle of the night and you on call as the orthopaedic consultant and what to do with the injury.

The dilemma is do you take the patient to theatre and perform emergency reduction or wait until the morning and get the hand surgeons involved for more definitive management (repair of carpal ligaments)?

A closed reduction should generally be performed to re-establish overall alignment followed by delayed surgery. The majority of time you will get a closed reduction although the injury itself may be unstable.

A worry is that the injury may be irreducible as interposed capsule may prevent reduction and you are stuck with a patient in theatre with an unreduced lunate dislocation at 4 am.

It is unlikely you will be experienced enough to perform an open reduction, emergency repair of intercarpal ligamants and K-wire stabilisation. You may or may not be familiar with the technique for open reduction of this injury.

If you can't get a reduction and are unfamiliar with the surgery for open procedure discuss with your hand colleague(recommended you pre-warn him/her before taking the patient to theatre) who will then need to perform an open reduction with removal of the obstructing factor, usually interposed capsule.

- **Q. Specifics of operative management (scoring 7 or 8)[60]?**
  - A. Volar, dorsal and combined dorsal–volar approaches

The volar approach is required to repair the tear in the palmer capsule ligament at the lunocapitate joint as well as carpal tunnel release.

The dorsal approach gives the best exposure of the carpus for restoration of alignment and interosseous ligament repair. In addition, fractures of the scaphoid and capitate can be secured with antegrade fixation devices.

The combined dorsal-volar approach offers the advantages of both approaches, but increases surgical time and dissection.

Dorsal approach

Incision

- A standard straight midline longitudinal incision in line with the third metacarpal extending into the distal forearm
- The incision is made though the third compartment

Extensor retinaculum

- The extensor retinaculum between the third and fourth extensor compartments is reflected off of the wrist capsule with care to avoid any damage to the capsule itself
- The EPL is mobilized out of its sheath and is reflected radially
- Subperiosteally elevate the fourth compartment, without disrupting the tendon sheath
- Homan retractors are placed on either side of the radius

Capsular incision

- Longitudinally incise through the dorsal capsule in line with Lister's tubercle, and then elevate the wrist capsule off of the dorsal rim of the distal radius including the dorsal radiotriquetral ligament
- Preserve the radiotriquetral ligament
- The dorsal capsule is usually opened along its origins from dorsal rim and longitudinally in space between second and fourth extensor compartments

- **Q. Operative repair?**
  - A.

  K-wire fixation
- Temporary lunate fixation to the radius
  - Scapholunate fixation
  - Before the scapho-lunate joint is pinned, pass the ligamentous repair sutures, but do not tie them together until all of the pins have been inserted and the reduction is optimal
  - Once k-wire fixation has been performed, repair the scapholunate interosseous ligament and augment this with bone anchors placed in the scaphoid. Reinforce the repair with a dorsal capsulodesis
- Scapho-capitate fixation
  - An additional K-wire is often across the scapholunate joint
  - With optimal reduction, the lunate should cover the head of the capitate
- Lunotriquetral fixation

- **Q. Complications?**
  - A.

- Chondrolysis
- Persistent wrist pain
- Traumatic osteoarthritis
- Carpal instability

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Chapter 22

Elbow oral core topics

Matthew Jones and Asir Aster

Anatomy of the elbow

The elbow is a complex joint consisting of ulnohumeral, radiocapitellar and proximal radioulnar joints. The distal articular part of the humerus is angled anteriorly by 30° to the axis of the humeral shaft. As the medial ridge of the trochlea is larger than the lateral ridge and the capitellum, the distal humerus has a valgus angle of 6° compared to the epicondylar axis. The coronoid and olecranon fossae are separated by a thin section of bone accommodating the coronoid and olecranon in extreme flexion and extension respectively. The radial fossa accommodates the radial head in full flexion. The medial epicondyle gives the origin to the common flexors/pronator muscles and the medial collateral ligament (MCL). The lateral epicondyle gives the origin to the common extensors/supinator muscles and the lateral collateral ligament (LCL). The greater sigmoid notch, the articulating part of the proximal ulna, is not covered with articular cartilage centrally. The lesser sigmoid notch, articulating with the radial head, is on the lateral aspect of the coronoid process.

The elbow capsule allows a maximum distension between 70° and 80° of flexion. This is the position of relative comfort for patients with a tense effusion.

The MCL has three bundles: Anterior, posterior and transverse bands. The anterior is the strongest of the three and is taut from full extension to 60° of flexion. It resists valgus stress in pronation. The radiocapitellar articulation is the secondary constraint to valgus stress. The posterior band is taut between 60° and full flexion.

The LCL has the lateral ulnar collateral ligament (LUCL), the annular ligament, the radial collateral ligament and the accessory collateral ligament. The LCL complex is taut throughout the elbow motion owing to its isometric position, with the exception of the LUCL, which is taut in flexion beyond 110°.

The radial nerve spirals medial to lateral posteriorly (13 cm from the trochlea) and then pierces the lateral intermuscular septum (7.5 cm from the trochlea) to lie between brachialis and brachioradialis, and passes distally anterior to the lateral epicondyle. The ulnar nerve is medial to the brachial artery in the arm prior to piercing the medial intermuscular septum to pass posterior to the medial epicondyle. The median nerve crosses the brachial artery lateral to medial in the arm and enters the cubital fossa, which is formed by the distal humerus proximally, the pronator teres medially and the brachioradialis laterally.

Surgical approach

- Posterior: Various modifications (the method of triceps split/olecranon osteotomy)
- Anterolateral (Henry's): Brachialis splitting (dual innervated muscle: Between radial and musculocutaneous nerves) followed by dissecting between pronator teres (median nerve) and brachioradialis (radial nerve)
- Posterolateral (Kocher): Between anconeus (radial nerve) and extensor carpi ulnaris (posterior interosseus nerve)
- Lateral column approach: Lateral supracondylar ridge proximally into 'Y'-shaped distal extension. The first limb of the 'Y' is between anconeus and the extensor carpi ulnaris to expose the posterior joint. The second limb of 'Y' is between extensor carpi radialis longus and brevis to expose the anterior joint
- Medial column approach: Medial incision. Free the ulnar nerve. Expose the medial intermuscular septum and flexor/pronator muscle group. Detach the intermuscular septum and reflect triceps posteriorly to expose the posterior joint. Split the flexor/pronator distally to expose the anterior joint

Arthroscopic portals

- Direct lateral portal: At the centre of a triangle defined by the lateral epicondyle, the radial head and the olecranon. This is frequently used as the initial entry portal to inflate the joint with saline
- Anterolateral portal: 1 cm distal and 1 cm anterior to the lateral epicondyle, between the radial head and the capitellum. This gives good access to the anterior aspect of the joint
- Anteromedial portal: 2 cm distal and 2 cm anterior to the medial epicondyle. This is often created using an 'inside out' technique by cutting down onto the tip of the arthroscope inserted using the anterolateral portal

- Proximal medial portal: 2 cm proximal to the medial epicondyle along the anterior surface of the humerus towards the radial head
- Direct posterior portal: 2 cm proximal to the tip of the olecranon. Access to olecranon fossae
- Posterolateral portal: 2–3 cm proximal to tip of olecranon, lateral border triceps. Access to the radiocapitellar joint

Examination corner

Basic science oral

- Identification of different bands of the medial and lateral collateral ligaments and their function
- Identification of neurovascular bundles around the elbow
- Surgical approaches, internervous and intermuscular planes

Tendinopathies

Repetitive tensile overload which exceeds tissue stress tolerance causes tissue damage. If it occurs at a rate that exceeds a tissue's ability to heal, tissue degeneration is caused. Histologically, there are no acute inflammatory cells. There is granulation-like tissue which consists of immature fibroblasts and disorganized non-functional vascular elements termed **angiofibroblastic hyperplasia**[1]. It is thought to result from an aborted healing response to microtrauma. Pain arises possibly from tissue ischaemia. Electron microscopy has shown that these vascular elements do not have lumen[2].

A 'mesenchymal syndrome' is described, whereby patients appear to develop multiple, related conditions including lateral and medial epicondylitis, achilles tendinopathy, rotator cuff pathology and carpal tunnel syndrome.

Tendinopathies around the elbow are tennis elbow, golfer's elbow and posterior tennis elbow.

Tennis elbow

This is degenerative tendinosis of extensor carpi radialis brevis (ECRB) and extensor digitorum communis (EDC).

The point of maximum tenderness is just anterior and distal to the lateral epicondyle.

Provocative test

- ECRB – Elbow extension/forearm pronation/fingers in flexion/wrist extended against resistance
- EDC – Elbow extension/forearm pronation/wrist neutral/fingers in extension/long finger extended against resistance. (Pain over EDC origin – Tendinosis. Pain over radial tunnel – Radial tunnel syndrome.)

Investigation

- MRI in acute post-traumatic conditions, where avulsion of the extensor origin may be diagnosed, which may need reattachment

Treatment

Non-operative management

The aim is to decrease the stress applied to the injured tendons/increase the stress tolerance. Approximately 75% of patients improve.

Rest, NSAIDs and steroid injections (injected under the muscle – Not subcutaneously – To prevent depigmentation of skin and fat atrophy and not into the tendon to avoid tendon weakening and rupture) may reduce the pain temporarily but do not necessarily improve healing. Counterforce bracing reduces the force transmitted to the tendon origin/prevents full expansion of the muscle; therefore, preventing maximal contraction. There are six key forearm exercises: Wrist flexion/extension, forearm supination/pronation and finger extension/flexion with gradual increase in the resistance, repetitions and gradual decrease in forearm support during the exercises.

Operative management

ECRB/EDC release by arthroscopy, percutaneous or open procedure, when non-operative treatment has made no improvement. Good results have been reported in up to 85%. This may be secondary to tendon lengthening/defunctioning/denervation or the introduction of an acute inflammatory response.

Differential diagnosis

Radiocapitellar degeneration (tender 2 cm distal to the lateral epicondyle) and radial tunnel syndrome (tender 4 cm distal to the lateral epicondyle).

Golfer's elbow

This is degenerative tendinosis of pronator teres (PT) and flexor carpi radialis (FCR).

The point of maximum tenderness for PT is proximal to the medial epicondyle and for FCR is just distal to the medial epicondyle.

Provocative test

- FCR – Elbow flexion/forearm supination/resisted wrist flexion and forearm pronation
- PT – Resisted forearm pronation

Surgical release is not as satisfactory as for tennis elbow.

Differential diagnosis

Injury to MCL, ulnar neuritis/cubital tunnel syndrome and ulnohumeral arthritis.

Posterior tennis elbow

This is degenerative tendinosis of the triceps tendon insertion. Maximum tenderness is at the insertion. It is provoked by resisted elbow extension. Surgically treatment is by debridement (not >50% tendon excision) and direct side-to-side repair.

Differential diagnosis

Posterior impingement, olecranon periostitis/bursitis and ulnohumeral arthritis.

Examination corner

Upper limb short case

EXAMINER: This gentleman is complaining of numbness in the ulnar nerve distribution of his hand. What is the cutaneous distribution of the ulnar nerve in the hand?

CANDIDATE: There is some anatomical variation, but in most people it is the little finger and ulnar border of the ring finger. There is also a dorsal branch which supplies the dorsum of the hand on the ulnar side.

EXAMINER: The symptoms have been present for 4 weeks since a posterior decompression and instrumented fusion of his lumbar spine. Why do you think he has developed these symptoms?

CANDIDATE: The procedure will have taken place under general anaesthetic and he would have been positioned prone with his arms resting on boards. He would have been in that position for some time and the nerve may have been compressed during this period.

EXAMINER: Why is it only unilateral?

CANDIDATE: It could be the way that the arm was positioned on that side, or he had some subclinical compression on that side preoperatively which was exacerbated.

EXAMINER: After clinical examination, what test would you request?

CANDIDATE: Nerve conduction studies.

EXAMINER: And if these confirmed severe slowing of conduction at the cubital tunnel, what would you recommend?

CANDIDATE: If symptoms were persisting and significant enough for surgery, I would offer surgical decompression. I would be more likely to offer surgery if there were loss of motor function or constant sensory dysfunction.

EXAMINER: He has no weakness or wasting and in fact his symptoms are slowly resolving.

CANDIDATE: In that case I would treat non-operatively with night splintage and review in a few weeks to re-examine him and confirm resolution of symptoms.

Tendon ruptures

Distal biceps rupture

A predisposing factor for rupture is degeneration in the hypovascular zone close to the radial tuberosity insertion. Risk factors are smoking (7.5 times) and anabolic steroid use. Mechanical irritation also plays a role – There is 50% reduction in the interosseous space in pronation. This is common in the dominant arm, in males of 40–60 years and in weightlifters.

The tendon ruptures with a painful pop following the arm being forced from a flexed position into extension – Eccentric tensile overload.

Apart from swelling, ecchymosis and tenderness (in both partial and complete ruptures) it is vital to examine for proximal migration of the muscle belly and loss of proximal to distal tracking of the tendon on passive forearm rotation (complete rupture). Do not be deceived by palpating the intact bicipital aponeurosis.

MRI or USS will be useful in doubtful cases and in partial ruptures.

Treatment

Non-operative

Analgesia and early range of motion exercise, when able. Leads to 30% loss of forearm flexion power and 40% loss of supination power[3].

Operative management

- Acute rupture: Best done within the first 2 weeks prior to the obliteration of the tunnel of the tendon. A single-incision technique using anchors or a two-incision technique using bone trough can be used. The two-incision technique reduces the risk of injury to the radial nerve. The dominant arm usually achieves a better result than the non-dominant arm
- Chronic rupture: Needs allograft or autograft to regain the length. Results are not as good as acute repair
- Partial tear: Splinting and decreased activity. Failing this management, surgical completion of tear, debridement and repair may be necessary

Distal triceps rupture

This is the rarest of all tendon ruptures. Risk factors are renal insufficiency with secondary hyperparathyroidism, systemic or local steroid use and previous surgery using a posterior approach.

The mechanism of rupture is similar to olecranon fracture – Sudden forced flexion of the extended elbow (eccentric tensile loading). A common site is at the insertion enthesis.

Modified Thompson test[4]: Forearm hanging free from bed – Elbow 90° flexed. Triceps muscle belly is squeezed. Absence of elbow extension suggests complete triceps rupture. If elbow extension is present but painful, suspect a partial tear.

Partial tear should be further investigated with MRI as >50% tear needs surgical repair. Repair is done through a posterior approach and gives good results.

Olecranon bursitis

The olecranon bursa is the most commonly affected bursa around the elbow. The bursa is a discrete structure, which does not usually communicate with the elbow joint, although in rheumatoid arthritis it may do so.

Causes

- Infection: 25% of bursitis is due to infection. *Staphylococcus aureus* is responsible for 90% of these infections

- Inflammatory
- Traumatic: Acute or repetitive trauma ('student's elbow')
- Haemodialysis
- Idiopathic

Assessment of systemic signs, haematological and biochemical markers help to identify infection. Aspiration may be beneficial. Organisms may not always be seen on Gram staining. A high WCC of $>1000/mm^3$ in the aspirate suggests infection.

Treatment

Infected bursitis

- Stage of initial cellulitis with no collection – Rest and antibiotics
- Presence of collection of pus – Aspiration and maybe drainage (may cause chronic sinus formation)

Non-infected bursitis

- NSAIDs and compressive bandages
- Steroid injections have been used, but may lead to dermal atrophy
- Excision

Examination corner

Adult pathology oral

EXAMINER: A 45-year-old patient in the ED with a painful, swollen left elbow and evidence of cellulitis covering most of the skin, worse on the posterior aspect. His temperature is 38.4°C and he keeps the elbow in 30° of flexion and does not like any further flexion. What is your differential diagnosis, please?

CANDIDATE: Septic arthritis of the elbow or infected olecranon bursitis.

EXAMINER: Which do you think is more likely based on what you have heard so far?

CANDIDATE: Infected olecranon bursitis. I would expect the elbow to be held nearer to 80° of flexion if there were a painful joint effusion.

EXAMINER: What will you do clinically to confirm your diagnosis?

CANDIDATE: I will test passive supination and pronation. In septic arthritis the patient will not allow any rotation of the elbow.

Elbow instability

Instability of the elbow may follow bony or ligamentous injuries. The outcome of ligamentous injuries is worse than bony injuries[5].

Elbow stabilisers

- MCL (anterior bundle) and LCL complex (especially lateral ulnar collateral ligament)
- Coronoid (50% loss of height – Instability)[6] and radiocapitellar joint (secondary valgus restraint)
- Varus/valgus – Common extensor and flexor/pronator muscles
- Anteroposterior – Brachialis, biceps and triceps

Instability – Types

- Acute – Following fracture dislocation or dislocation
- Chronic – Repetitive microtrauma, usually affecting MCL
- Longitudinal – Injury to radial head, central band of interosseous membrane and ligamentous injury to radioulnar joints (Essex–Lopresti fracture/dislocation)
- Rotatory – Posterolateral rotatory instability (lateral ulnar collateral ligament)[7]

Acute instability

Acute instability presents as fracture dislocation or pure ligamentous injury. The terrible triad comprises:

- Posterolateral dislocation or LCL injury
- Coronoid fracture involving >50% of its height
- Radial head fracture

After reduction of the dislocation, assess the congruity of reduction. If the reduction is incongruent, a bony fragment or soft tissue is interposed. If no clear bony fragment on plain x-ray, consider a CT and/or MRI. Check the stability of the joint. If there is loss of stability when extending beyond 45° of flexion, immobilize at 90° of flexion and pronation. If it is not possible to maintain stability between 45° and 60° of flexion in pronation, surgical exploration and repair/reconstruction of the ligaments is indicated. Postoperatively the elbow is immobilized in flexion with an extension block; the extension is increased by 30° every week for 3 weeks to allow a full arc of movement. In fracture dislocations anatomical reconstruction of the fractures is essential with or without ligament repair (depending on assessment of stability after fracture fixation).

Chronic instability

Chronic valgus instability results from repetitive microtrauma to the anterior bundle of MCL in athletes involved in over head throwing. It presents with pain at the ulnar attachment of MCL. Valgus laxity allows abutment of the olecranon against the medial aspect of the humerus, resulting in the formation of an osteophyte. This posteromedial osteophyte gives rise to posterior impingement and loss of full extension. Direct ligament repair is less successful than reconstruction. Reconstruction is performed using autograft (eg palmaris longus tendon). Success depends on correct tensioning of graft (the ulnohumeral joint medial opening should be closed) and placement of the graft in the position that allows isometric tension during the full arc of motion.

Longitudinal instability

Longitudinal instability arises from an Essex–Lopresti fracture/dislocation owing to fracture of the radial head, rupture of the interosseous membrane and the radioulnar joint ligaments. If excising the radial head is an option, the longitudinal stability must be tested under fluoroscopy by

proximally pulling the radius and assessing the distal radio-ulnar joint (DRUJ). If proximal migration of the radius is >6 mm at the DRUJ, the radial head must be replaced.

Failure to replace the radial head causes proximal migration of the radius, giving symptoms from lateral elbow instability as well as DRUJ/triangular fibrocartilage complex (TFCC) symptoms. Treatment options are ulnar shortening, interosseous membrane reconstruction with patellar tendon graft (as the modulus of elasticity and ultimate tensile strength are closer to the patellar tendon), radial head reconstruction and, lastly, in patients with limited forearm rotation and painful forearm, creation of a one-bone forearm.

Rotatory instability

Posterolateral rotatory instability arises from damage to the lateral ulnar collateral ligament (LUCL), which normally provides a sling support to the radial head posterolaterally. In the absence of an intact LUCL, the radial head subluxes laterally and the ulna pivots on the MCL and rotates off the trochlea in a valgus/externally rotated position during flexion. Acute avulsion injuries are treated with reattachment to bone. Acute intrasubstance injury and chronic injury are treated with reconstruction using tendon graft.

Examination corner

Trauma viva

EXAMINER: You are called to see a 30-year-old man in Resus who has come off his motorbike at high speed and collided with a tree. What are your priorities?

CANDIDATE: ABC. I would initially manage the patient according to ATLS® principles.

EXAMINER: OK. We'll assume that has been done and he is cardiovascularly stable with an isolated injury to the left elbow. What can you see from this photograph?

CANDIDATE: This is a clinical photograph of this gentleman's left elbow, forearm and hand. The elbow is grossly swollen, bruised and deformed. I cannot see any wounds on this picture but would examine him closely for evidence of an open injury.

EXAMINER: It is open. There is a wound on the medial side of the elbow.

CANDIDATE: I would also check his neurological and vascular status and for evidence of compartment syndrome.

EXAMINER: The injury is neurovascularly intact and he is comfortable when the limb is splinted. What investigation will you do?

CANDIDATE: He should have had a chest and pelvis x-ray as part of his ATLS® management. I would also request plain radiographs of the elbow.

EXAMINER: Here they are.

CANDIDATE: These are AP and lateral radiographs of the left elbow of this gentleman. There is an intra-articular, comminuted fracture of the distal humerus. The upper humerus is not visible, I would want x-rays of this too.

EXAMINER: There fracture does not extend beyond what you can see. It is now midnight. Are you going to take him to theatre tonight?

CANDIDATE: Unless the wound is grossly contaminated I will not operate on him tonight. I will ensure he has had antibiotics, a photograph of the wound, a sterile dressing and a splint. I will plan to operate on him in the morning with a full complement of staff.

EXAMINER: You are in theatre with him the next morning. How will you position him and what kind of anaesthetic will he require?

CANDIDATE: This operation may take >2 hours and ,therefore, he is probably better with a general anaesthetic, but I will liaise with the anaesthetist regarding this. I will position him laterally with the arm over a bar. I will use a tourniquet as the fracture is low enough to accommodate this.

EXAMINER: What approach will you use?

CANDIDATE: A posterior approach with olecranon osteotomy. I will make a chevron osteotomy using an oscillating saw followed by an osteotome to crack through to the joint. I will pre-drill the olecranon for later repair.

EXAMINER: Where will you put your plates?

CANDIDATE: (Discussion regarding 90/90 vs parallel plating)

Elbow arthritis

The elbow helps to position the hand in the spherical space for which the shoulder is the centre of rotation. Its **functional** range of movement is a 100° arc of flexion (30–130°) and pro-supination (50°/50°). The symptoms of elbow arthritis include pain, stiffness, swelling (effusion and synovitis), neurological symptoms (mostly ulnar nerve) and instability.

Causes

- Inflammatory
- Post-traumatic
- Primary osteoarthritis
- Neuropathic

Inflammatory

Rheumatoid arthritis is a common inflammatory arthritis affecting the elbow. Up to 50% of patients with rheumatoid arthritis have elbow involvement. Women are affected three times more commonly than men. Medical anti-rheumatoid therapy has reduced the need for surgery, but this is still required in selected cases.

Clinical findings

- Joint contractures (especially juvenile idiopathic arthritis)
- Ligamentous instability secondary to attenuation

  MCL – Valgus ulnohumeral instability
  LUCL – Posterolateral rotatory instability
  Annular ligament – Radial head subluxation
  Combined – Multidirectional instability

- Neurological symptoms

  Ulnar nerve – Secondary to valgus instability
  Posterior interosseous nerve – Synovitis/pannus around the radiocapitellar joint
- Subarticular fractures

  Subchondral cyst – Pannus induced
  Periarticular osteopenia

Radiological findings

- Panarticular (ulnohumeral and radiocapitellar) loss of joint space
- Subchondral cysts
- Periarticular osteopenia
- Absence of subchondral sclerosis and osteophytes, except when secondary osteoarthritis present
- Subluxed/dislocated radiocapitellar joint

Post-traumatic

The elbow is most intolerant to trauma. Immobilization predisposes it to stiffness. Intra-articular injuries predispose to post-traumatic arthritis. It affects all age groups, mainly young/middle-aged individuals.

Clinical findings

- Pain at the end range of movements
- Stiffness
- Locking or clicking – Loose bodies
- Ulnar nerve dysfunction – Medial osteophytes from the ulnohumeral joint

Radiological findings

- Joint space irregularity/narrowing of the affected compartment
- Subchondral sclerosis
- Subchondral cysts
- Osteophytes

Primary osteoarthritis

This type of arthritis is common in middle- to old-age males performing repetitive manual labour.

Clinical findings

- Pain at the end range of motion
- Mechanical block to flexion and extension
- Pain on carrying weight in extension
- Locking and clicking – Loose bodies

Radiological findings

- Subchondral sclerosis of the radiocapitellar joint with preserved joint space
- Progressing to radiocapitellar narrowing producing valgus tilt, with involvement of the lateral facets of the ulnohumeral joint
- Osteophytes at the tip of the olecranon and coronoid
- It is not common to have mal-alignment or subluxation of radiocapitellar joint

Neuropathic

The neuropathic joint is painless with severe loss of bone and joint architecture and loss of joint stability.

The causes include surgical denervation, diabetes, syringomyelia and syphilis.

Treatment of elbow arthritis

Non-operative

- Activity modification/splinting/intra-articular steroid injection/analgesia
- Disease modifying agents for inflammatory arthritis

Operative

- Debridement – Arthroscopic/open. The Outerbridge–Kashiwagi (OK) procedure involves opening the olecranon fossa, drilling through the floor of the olecranon fossa to the coronoid fossa and debriding osteophytes and removing loose bodies from both fossae
- Synovectomy with or without radial head excision – Arthroscopic/open
- Ulnohumeral interposition arthroplasty – For non-inflammatory conditions. Resurfacing materials used include fascia lata and Achilles tendon. Rarely done
- Resurfacing – Radiocapitellar joint (metaphyseal fixation by pegs). Lateral resurfacing of the elbow – In the early stages of osteoarthritis involving the radiocapitellar joint prior to the progression into valgus tilt. Advantages are – Minimal bone loss during bone preparation (3 mm loss from either surface), maintains normal anatomy and alignment and the ability to regain full rotational arc (as replaced radiocapitellar joint) as well as flexion/extension arc (as resurfacing gives ideal surface and room for radial head). As the lost cartilage in the lateral elbow is being replaced, theoretically the forces are redistributed anatomically and, therefore, it could prevent the usual progression of the arthritis to the medial joint
- Replacement – Unlinked. Greater risk of instability especially in rheumatoid patients as the unlinked prosthesis relies on ligamentous stability
- Replacement – Linked/semi-constrained. 'Sloppy hinge' such as the Coonrad-Morrey and Discovery prostheses. Ideal for unstable rheumatoid elbow
- Replacement – Linked/constrained. It is historical. There is excessive load transfer to stem/cement/bone interfaces and high (25% in 4 years) evidence of loosening
- Arthrodesis – unilateral arthrodesis is at 110° of flexion to help reaching the face. If bilateral arthrodesis is needed, the second elbow should be at a greater degree of extension to reach perineum for personal hygiene

Examination corner

Adult pathology viva

EXAMINER: This 40-year-old delivery driver with rheumatoid arthritis is sent by his rheumatologist. He has failed conservative management and the rheumatologist is asking whether surgery is an option. What do you think of his x-rays?

CANDIDATE: These are AP and lateral plain radiographs of this gentleman's left elbow. There is a marked loss of joint space in the ulnohumeral joint and radiocapitellar joint. There are extensive osteophytes implying secondary osteoarthritis. There are some cysts in the distal humerus.

EXAMINER: What are the surgical options?

CANDIDATE: This is a difficult problem as this is a young patient. The options are debridement, partial replacement, total replacement and arthrodesis. Based on his x-rays, I would not expect him to benefit much from debridement or partial replacement. Does he need to lift for his work?

EXAMINER: Yes.

CANDIDATE: The problem is that a total elbow arthroplasty would not be strong enough for him to use the arm for heavy physical activity. He is only 40 and has a physical job. The alternative of elbow arthrodesis would obliterate elbow movement and may also prevent him from doing his job.

EXAMINER: So what will you do?

CANDIDATE: I would discuss the options with the patient so that he is fully informed about the advantages and disadvantages of each option. My recommendation would be to try and persist with non-operative treatment for as long as possible.

EXAMINER: He has exhausted all non-surgical therapy and is interested in the arthrodesis. What position would you fuse him at?

CANDIDATE: I would tailor the angle to the patient. Essentially he needs to get his hand to his mouth, and this would usually involve fusion at about 110° of flexion.

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Chapter 23

Shoulder oral core topics

Matthew Jones and Asir Aster

Anatomy of shoulder

The shoulder comprises three joints: The glenohumeral, sternoclavicular and acromioclavicular. The scapula in turn articulates with the chest wall to confer additional range of movement. Two-thirds of shoulder abduction occurs at the glenohumeral joint and one third at the scapulothoracic articulation.

The glenohumeral joint is extremely mobile, but this mobility comes at a cost to stability. The joint has both **static** and **dynamic** stabilisers to prevent dislocation. The static stabilisers are:

- Bony congruity between the humeral head and glenoid
- The glenoid labrum
- Negative pressure
- Ligaments (Table 23.1)

The dynamic stabilisers are the **rotator cuff** and **extrinsic muscles** of the shoulder including the pectotalis major, latissimus dorsi, deltoid, coracobrachialis, pec minor, biceps and triceps.

In functional ranges of movement, the dynamic stabilisers are the principle stabilisers. They centre the humeral head in the glenoid by **concavity compression**. The capsular ligaments act as check reins at extremes of range.

Cuff function is compared to a **suspension bridge** model and cable/crescent. The subscapularis tendon forms the anterior pillar and the infraspinatus and teres minor form the posterior pillar. Even if there is a tear in the supraspinatus tendon, as long as the pillars of the suspension bridge or the cable are intact the cuff function is maintained. When there is discontinuity of either end of the pillar or cable, there will be anterior or posterior translation and elevation will be compromised. The rotator interval (triangular) is formed superiorly by the anterior border of supraspinatus, inferiorly by the superior border of subscapularis and medially by the base of the coracoid. The apex is formed by the transverse humeral ligament. It contains the coracohumeral ligament.

Table 23.1 Shoulder ligaments and action

Ligament	Action
Superior glenohumeral	Opposes inferior translation in adduction
Middle glenohumeral	Opposes anteroinferior translation in the midrange
Inferior glenohumeral	Opposes anterior translation abduction
Anterior ligaments collectively (superior, middle and inferior)	Oppose anterior humeral translation in external rotation
Posterior glenohumeral	Oppose posterior translation in internal rotation

The sternoclavicular (SC) joint and acromioclavicular (AC) joint are gliding joints. In the SC joint, the anterior and posterior SC ligaments prevent superoinferior translation and the interclavicular and costoclavicular ligaments prevent anteroposterior translation. In the AC joint, the superior and inferior acromioclavicular (AC) ligaments prevent anteroposterior translations and the coracoclavicular ligaments (CC; trapezoid and conoid) prevent superior translation of the clavicle. The clavicle is the first bone to ossify (5 weeks of fetal development) and it is the last one to fuse (25 years – Medial epiphysis), and although it is a long bone it ossifies by intramembranous ossification.

The scapula spans ribs 2 to 7. It has three processes: The spine, coracoid and acromion. Glenoid orientation ranges from 7° of retroversion to 10° of anteversion and has 5° of superior tilt. The humeral head is in 20–30° of retroversion and has a 130° superior inclination relative to the shaft. The coracoid process provides attachment to three ligaments (coracohumeral, coracoacromial and coracoclavicular) and three muscles (pectoralis minor, coracobrachialis and short head of biceps). The suprascapular **artery** is superior to the superior transverse ligament and inferior to the inferior transverse ligament. The suprascapular **nerve** is inferior to both ligaments.

The scapula can wing medially or laterally. In medial winging, trapezius is unopposed by the weak **serratus anterior** and the medial border of the scapula becomes prominent. In lateral winging, the **trapezius** is weak and, therefore, it is the lateral border that protrudes.

Pectoralis minor divides the axillary artery into three parts. The first part has one branch, the second has two branches and the third has three branches.

Parts of the axillary artery (Table 23.2)

Spaces of the shoulder (Table 23.3 and Figure 23.1)

Surgical approaches

- Deltopectoral approach: Between deltoid (axillary nerve) and pectoralis major (medial and lateral pectoral nerves). The cephalic vein lies between them: Retract it either way, usually laterally due to multiple deltoid branches. Place stay sutures in subscapularis and incise tendon leaving a cuff to repair. Incise capsule. Beware of brachial plexus and particularly the musculocutaneous nerve medial to the coracoid, at risk from excessive retraction. Lateral to the coracoid is the 'safe side', medial is the 'suicide'!
- Lateral approach: Deltoid splitting (axillary nerve). When split extends beyond 5 cm inferior to acromion; axillary nerve is at risk. Rotator cuff tendons are beneath deltoid so one can use this approach for access to the cuff. Can also be used to nail the humerus, but if doing this, split the muscle, not the tendon, and repair it afterwards
- Posterior approach: Between infraspinatus (suprascapular nerve) and teres minor (axillary nerve). Inferior retraction of teres minor risks damaging the axillary nerve and the posterior humeral circumflex artery (quadrilateral space). Medial retraction of infraspinatus can injure the suprascapular nerve

Table 23.2 Axillary artery anatomy

Part	Location	Branches
First	Medial to pectoralis minor	Supreme thoracic
Second	Posterior to pectoralis minor	Thoracoacromial Lateral thoracic
Third	Lateral to pectoralis minor	Subscapular Anterior humeral circumflex Posterior humeral circumflex

Table 23.3 Anatomical spaces of the shoulder

Space	Borders	Contents
Quadrilateral space (rule of fours)	Four borders: Teres minor, teres major, long head of triceps, humerus	Four words: Posterior humeral circumflex artery Four syllable nerve: Axillary
Triangular space	Teres minor, teres major, long head triceps	Circumflex scapular artery
Triangular interval	Long head of triceps, humerus (plus lateral head of triceps), teres major	Profunda brachii Radial nerve

Arthroscopic portals

- Posterior: (viewing) 2 cm medial and inferior to the posterolateral corner of the acromion
- Anterior: Lateral and inferior to the coracoid process. This portal should be made under direct vision using a needle as a guide. Aim for the rotator interval between supraspinatus and subscapularis
- Lateral: Junction of anterior one-third and posterior two-thirds of the lateral border of the acromion and 2 cm inferior. Provides a view of/access to the subacromial space for cuff inspection/repair
- Additional arthroscopic portals – Nevasier, anterolateral, posterolateral, anteroinferior and posteroinferior

Examination corner

Basic science oral

- Identification of structures from pictures, especially axial CT images or cross-sectional cadaveric photographs – Muscles, tendons, ligaments, intermuscular spaces and intervals, nerves and blood vessels
- Explain – Static and dynamic stabilisers of the shoulder
- Surgical approaches – Surface markings, internervous planes, intermuscular planes and structures at risk
- Mark the standard arthroscopic portals on the picture and mention the use of these portals
- Cuff – Identification of individual muscles, innervation, function and rotator interval

Impingement syndrome

Types of impingement

- Subacromial impingement

  Primary – Intrinsic (degenerative tendonopathy) or extrinsic (coracoacromial arch)

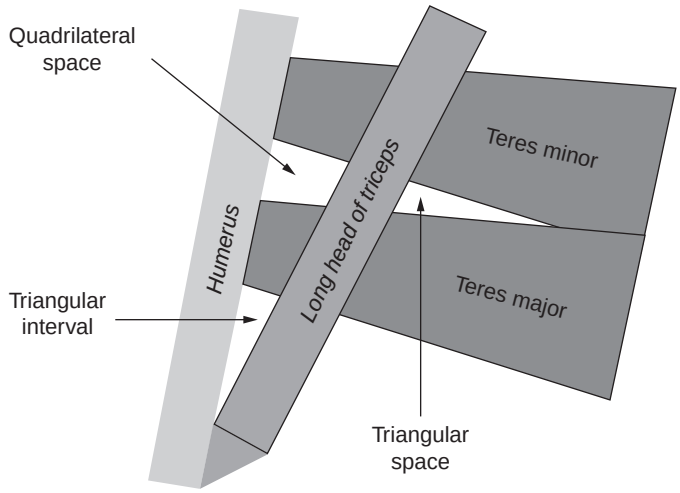

Figure 23.1 Shoulder spaces and intervals

Secondary – Owing to glenohumeral instability
- Subcoracoid impingement
- Internal impingement

Subacromial impingement

Subacromial impingement relates to the symptoms of pain and dysfunction from either a reduction in the volume of the subacromial space, or an increase in the contents. Rotator cuff damage occurs with excess stress, repetitive tensile loading, or inadequate healing. Subacromial impingement is an overuse phenomenon, which lacks acute inflammatory cells and should appropriately be called tendinosis. The affected swollen tendon has less space under the acromion and causes intrinsic impingement.

Extrinsic impingement occurs when the space available for the rotator cuff is diminished because of:

- subacromial spurring
- acromial fracture or os acromiale
- osteophytes from the under-surface of the acromioclavicular joint
- exostoses at the greater tuberosity

The subacromial spurring is caused by enthesophyte formation at the coracoacromial ligament's acromial insertion, secondary to dynamic loading. In 1986, Bigliani introduced a morphological classification of the acromion[1]

- Type I: Flat acromion
- Type II: Curved acromion (parallel to the humeral head)
- Type III: Hooked (converging on the humeral head) – Commonest association with subacromial impingement

Chronic degeneration of the cuff affects its optimum function and allows the deltoid to overcome the cuff's concavity compression and pulls the humeral head upwards. The deltoid's upward pull causes the cuff to impinge on the coracoacromial arch, causing secondary subacromial impingement.

Stages of impingement[2]

- Stage 1: Oedema and haemorrhage <25 years old
- Stage 2: Fibrosis and tendonitis 25–40 years old
- Stage 3: Bone spurs and tendon rupture >40 years old

The symptoms include anterolateral shoulder pain over the shoulder with aggravation by overhead activities. Examination reveals a painful mid-abduction arc from 60° to 120°.

Provocative tests

- **Neer's impingement sign**: Pain on forward elevation of the arm
- **Neer's impingement test**: Temporary relief of pain with subacromial injection of lidocaine
- **Hawkins' sign**: Pain on internal rotation of the arm in 90° of forward elevation, as this brings the greater tuberosity under the acromion and further exacerbates any compression of the supraspinatus tendon

Treatment

Non-operative management

Anti-inflammatory medications and steroid injections for symptom control. Physiotherapy aiming at stretching for full shoulder motion and strengthening the rotator cuff, deltoid and scapulothoracic muscles.

Operative management

Subacromial decompression: Arthroscopic or open release of the coracoacromial ligament and removal of the anterolateral lip and undersurface of the acromion. Failure to respond to conservative therapy is an indication for operative intervention.

Subcoracoid impingement

This presents with pain in the anterior shoulder caused by contact between the subscapularis and the coracoid process, and is caused by a prominent coracoid in forward flexion and internal rotation of shoulder.

Provocative tests

- Coracoid impingement sign: This is performed in a similar way to Hawkins' sign, apart from the arm being adducted 10–20° to bring the lesser tuberosity in contact with the coracoid
- Coracoid impingement test: Pain relief with local anaesthetic in the subcoracoid region

Treatment

Open or arthroscopic coracoplasty.

Internal impingement

This is caused by internal contact of the posterior rotator cuff with the posterosuperior aspect of the glenoid when the arm is abducted, extended and externally rotated. It often occurs in throwers and they demonstrate lost internal rotation of the affected shoulder in comparison to the unaffected side[3].

Treatment

Physiotherapy is used to regain internal rotation and rotator cuff strengthening. Surgical treatment is by removal of posterosuperior glenoid osteophytes and posterior release.

Rotator cuff tears

Rotator cuff tears range from partial tears to complete irreparable tears. The cuff fails because of a repetitive tensile load which exceeds its ability to heal. Greater than 90% of partial tears occur on the articular side away from the acromion secondary to poor blood supply[4]. The natural history of rotator cuff tears cannot reliably be predicted.

Classifications

- Partial-thickness tears based on location

  Bursal side tear
  Intratendonous tear

Articular side tear (includes PASTA lesion: Partial articular supraspinatus tendon avulsion)

- Partial-thickness tears based on depth of involvement: Ellman's classification
  - Grade I: Tears with a depth of <3 mm
  - Grade II: Depth of 3–6 mm, but always less than half tendon thickness
  - Grade III: Depth >6 mm or more than half thickness of the tendon
- Full-thickness tears: Cofield's classification

  Small (<1 cm)
  Moderate (1–3 cm)
  Large (3–5 cm)
  Massive (>5 cm)
- Goutallier staging of fatty degeneration of the rotator cuff muscle[5]
  - Stage 0: Normal muscle without any fatty streak
  - Stage 1: Muscle contains some fatty streaks
  - Stage 2: There is still more muscle than fat
  - Stage 3: There is as much fat as muscle
  - Stage 4: More fat than muscle
- Anatomical classification
  - Longitudinal tear: Longitudinal split
  - Crescent shaped: Minimally retracted
  - U-shaped: Medially retracted
  - L-shaped: A combination of a longitudinal tear with a transverse limb

Clinical features

- Referred pain at deltoid insertion site
- Loss of range of motion: Initially affecting active movements, but later passive movements are restricted due to progressive capsular contracture
- Weakness and wasting of the involved cuff muscle
- Instability – Especially when the cuff tear was secondary to dislocation
- Mechanical – Snapping or catching during movements owing to lack of concentric concavity compression

Assessment

- Wasting – Mainly seen in the supraspinous and infraspinous fossae
- Range of motion: Active followed by passive movements. Examine from the front and from the back with your hands on the scapulae to differentiate glenohumeral from scapulothoracic movement
- Power
  - Supraspinatus: Jobe's test (empty can test)
  - Infraspinatus and teres minor: External rotation strength
  - Subscapularis: Gerber's lift-off test if sufficient range of internal rotation to get the hand to the lumbar spine. Beware of triceps recruitment giving a false negative test. Alternatively, the belly press (Napoleon) test
- Lag tests for each cuff muscle
  - Supraspinatus: passively place the limb in the position for Jobe's test and see if the patient can maintain it
  - Infraspinatus: passively place the limb in external rotation and see if the patient can maintain it
  - Teres minor: passively place the shoulder in abduction, forward elevation and external rotation. If the patient cannot maintain this actively, the hand will drop in front of the face (hornblower's sign)
  - Subscapularis: if sufficient range of internal rotation, passively place the patient's hand off their lumbar spine and see if they can maintain it

Investigations

- Plain radiographs
  - Sclerosis of undersurface of the acromion
  - Traction spurs in the coracoacromial ligament
  - Upward displacement of the humeral head
  - Acetabularized coracoid, acromion and glenoid
  - Acromioclavicular joint arthritis – Inferior osteophytes
  - Degenerative calcification of the cuff
- Cuff tendon imaging
  - Ultrasonography: Dynamic evaluation of cuff
  - MR arthrogram: Detects cuff tears and also assesses cuff musculature

Treatment

Partial-thickness rotator cuff tears

Non-operative management includes activity modification, stretching followed by strengthening exercises and anti-inflammatory medication. Surgery is indicated for failure of conservative treatment. If the shoulder demonstrates stiffness, a shoulder mobilization programme is instituted before surgery.

Principles of partial cuff tear surgery include:

- Assessment of the cuff tear – Mainly the thickness of the tear and its quality
- Release of capsular contractures
- Reintroduce healing biology – By debridement of the degenerated cuff and by performing simultaneous subacromial decompression
- Reattach the tendon to its anatomical footprint
- Protect the repair until it heals
- Regain movement and its control postoperatively by a physiotherapy programme

Tears involving <50% are treated with debridement with or without acromioplasty. A tear depth of >50% is an indication for completion of tear and repair of the cuff with acromioplasty.

Full-thickness rotator cuff tears

Non-operative management consists of physiotherapy, NSAIDs, rest, avoidance of aggravating factors and judicious steroid injections.

The principles of physiotherapy include four-quadrant stretching, strengthening exercises of the rotator cuff, deltoid and surrounding musculature.

Operative management is by open, mini-open or arthroscopic approaches

- Longitudinal tear: Side-to-side repair
- Crescent-shaped: Repaired back to greater tuberosity
- U-shaped: Margin convergence to create crescent-shaped tear and repaired back to bone
- L-shaped: Mobile torn leaf to other leaf by side-to-side repair and repair back to bone

Irreparable rotator cuff tears

Non-operative treatment includes NSAIDs, steroid injections and local therapeutic modalities to relieve pain. Early restoration of the passive range of motion and activity modification is followed by muscle strengthening exercises.

Surgical options in this difficult scenario include

- Subscapularis tendon transposition is used to fill large gap in the supraspinatus insertion
- Latissimus dorsi muscle transfer for posterosuperior defects
- Pectoralis and teres major transfer for anterosuperior defects
- Glenohumeral arthrodesis when the deltoid and rotator cuff muscles are not functional
- Human-derived allograft (e.g. Graft Jacket)
- Porcine xenograft

Arthritis

Glenohumeral arthritis

This is the end result of loss of articular cartilage in the shoulder. Clinical features include muscle wasting, painful limitation of range of movement and palpable crepitus. Radiographically, the usual findings of loss of joint space, osteophytes, subchondral sclerosis and bone cysts can be seen.

Types

- Primary degenerative: Triad of anterior capsular contracture, posterior glenoid wear with posterior humeral subluxation
- Secondary degenerative: Secondary to trauma, infection or avascular necrosis
- Inflammatory arthritis: Glenoid erosion is predominantly medial with frequent bilateral symmetrical involvement
- Cuff tear arthropathy: A deficient cuff leads to a lack of centralisation of the humeral head in the glenoid. Deltoid and other extrinsic muscles pull the humeral head upwards. Progressive incompetence of the coraco-acromial ligament leads to anterosuperior escape of the humeral head. The glenoid and coracoacromial arch becomes 'acetabularised'
- Capsulorrhaphy arthropathy: The posterior glenoid is eroded with anterior capsular tightening, and anterior erosion of the glenoid with anterior translation of humerus occurs with posterior capsulorrhaphy
- Neuropathic arthritis

Table 23.4 Walsh classification of glenoid wear

A1	Minor **concentric** wear, no subluxation
A2	Major **concentric** wear, no subluxation
B1	Posterior **eccentric** wear, posterior subluxation
B2	**Biconcave** glenoid, posterior subluxation
C	Retroverted glenoid >25°

Investigations

- Standard radiographs include AP views in the plane of the glenoid and axillary views
- CT scan to assess glenoid bone stock, zone of wear and orientation
- US scan or MRI is used to evaluate the quality of the rotator cuff for preoperative planning

Table 23.4 shows Walsh classification of glenoid wear[6].

Treatment

- Non-operative treatment: range of movement and strengthening exercises with NSAIDs, analgesics and steroid injections
- Operative management: Arthroplasty for pain relief and improved function

Types of shoulder arthroplasty

- Anatomic total shoulder replacement
- Hemi-arthroplasty
- Reverse total shoulder replacement
  - Indicated when the cuff is deficient
  - Not recommended in younger, higher demand patients due to loosening of glenoid component
  - Relies on a functional deltoid
  - Medialises the centre of rotation of the shoulder and thereby increases the length of the lever arm for the deltoid

Examination corner

Upper limb intermediate case

EXAMINER: This 70-year-old gentleman has been referred by his GP with swelling and reduced function of the right shoulder. What can you see on inspection?

CANDIDATE: This gentleman's right shoulder is grossly swollen over the AC joint and anterior glenohumeral joint. There are no scars or erythema present.

EXAMINER: OK. Here are his x-rays.

CANDIDATE: These are AP and axillary radiographs of this gentleman's right shoulder demonstrating cuff arthropathy. The humeral head is superiorly displaced and there is acetabularisation of the glenoid and coracoacromial arch. There is secondary osteoarthritis as evidenced by loss of joint space, subchondral sclerosis and cysts, and osteophytes.

EXAMINER: What do you expect to find when you examine his rotator cuff?

CANDIDATE: I expect to find weakness in abduction and external rotation.

EXAMINER: This gentleman has exhausted non-operative management. What surgery would you propose?

CANDIDATE: In view of his deficient rotator cuff, I would suggest a reverse total shoulder replacement.

EXAMINER: What would you want to know from your examination before proceeding to surgery?

CANDIDATE: I would check that he has a good passive range of movement and if not I would refer to physiotherapy to see if this could be optimised. I would also check that deltoid is functioning well.

EXAMINER: How does a reverse shoulder prosthesis work biomechanically?

CANDIDATE: The centre of rotation of the shoulder is moved medially which increases the lever arm for the deltoid. This enables the deltoid to function more effectively through a greater range of movement and to compensate for a deficient rotator cuff.

Acromioclavicular joint arthritis

Primary arthritis of the ACJ is much more common than the glenohumeral joint. Asymptomatic AC joint degeneration is frequent and, therefore, symptoms do not correlate well with x-ray findings. Post-traumatic arthritis is more common than primary arthritis.

Clinical features

- Discomfort over the anterior and superior aspects of the shoulder
- Radiating pain aggravated with physical activity, especially above the head activities
- Feeling of popping, catching or grinding
- Tenderness over AC joint
- Provocative test: Cross-body adduction (Scarf) test causes pain in AC joint region

Investigations

- Radiograph: Zanca view to visualize the AC joint (10° cephalic tilt)
- Symptom relief from injection of local anaesthetic is diagnostic

Treatment

Non-operative

Activity modification, moist heat, NSAIDs, corticosteroid injections and physiotherapy. Ultrasound guidance can help to confirm intra-articular injection.

Surgical treatment

Excision of AC joint by an open or arthroscopic technique (do not excise >0.5–1.0 cm of the lateral end of clavicle to protect the coracoclavicular ligaments and prevent superior migration of the clavicle). Most surgeons prefer arthroscopic to open excision.

AC joint injury

Traumatic injuries to the AC joint are classified by Rockwood[7] as shown in Table 23.5.

Examination corner

Trauma viva

EXAMINER: This 45-year-old lady fell off a horse. You see her in the A&E and ATLS® principles have been followed. She has an isolated injury to the left shoulder. What do you seen on this photograph?

Table 23.5 AC joint injury

Type	Ligaments	Radiographs	Management
I	AC ligaments sprained	Normal x-ray	Non-operative
II	AC ligaments torn, CC ligaments sprained	Superior displacement of clavicle <25%	Non-operative
III	AC and CC ligaments torn	Superior displacement of clavicle 25–100%	Depends on symptoms/demand
IV	AC and CC ligaments torn	Clavicle displaced posteriorly through trapezius	Surgical repair/stabilisation
V	AC and CC ligaments torn	Clavicle displaced superiorly by >100%	Surgical repair/stabilisation
VI	AC and CC ligaments torn	Clavicle displaced inferiorly (rare)	Surgical repair/stabilisation

CANDIDATE: This is a clinical photograph of this lady's left shoulder from the anterior aspect. There is bruising and swelling of the shoulder with prominence of the distal clavicle.

EXAMINER: What is your diagnosis?

CANDIDATE: This could be a clavicle fracture or AC joint dislocation.

EXAMINER: How will you make the diagnosis?

CANDIDATE: I would like to see plain x-rays: An AP and a Zanca view with 10–15° of cephalic tilt.

EXAMINER: Here they are.

CANDIDATE: There is dislocation of the AC joint with gross superior displacement of the distal clavicle. This implies complete tear of the AC and CC ligaments. This is classified according to Rockwood as type V.

EXAMINER: What treatment would you recommend?

CANDIDATE: I would recommend surgical stabilisation to restore and maintain the AC joint anatomy.

EXAMINER: What would be the sequel of non-operative management?

CANDIDATE: The AC joint would remain dislocated and would likely remain unstable. This would result in pain, clicking and dysfunction of the shoulder.

Osteolysis of the distal clavicle

This is seen predominantly in weight lifters and is thought to represent repetitive microfracture of the distal clavicle. Patients present with pain in the AC joint region and x-rays show a lytic area in the distal clavicle. Treatment is non-operative with NSAIDs, activity modification and sometimes steroid injection. If symptoms persist, excision of the distal clavicle may be considered.

Shoulder instability

Joint instability is an abnormal symptomatic motion in the joint resulting in pain, subluxation or dislocation. Joint laxity is a clinically detectable degree of translation in the joint, which falls within a physiological range and is asymptomatic.

Factors affecting the stability of the shoulder are described at the beginning of this chapter.

Classifications for instability

- Degree – Subluxation or dislocation
- Chronicity – Acute or chronic
- Direction – Unidirectional or multidirectional
- Volition – Voluntary or involuntary

Historically, Thomas and Matsen's 'TUBS and AMBRI' classification[8] has been used, but this has been largely replaced by the Bayley triangle from Stanmore[9]. This recognises three 'polar groups' but also that patients can lie on a spectrum between groups.

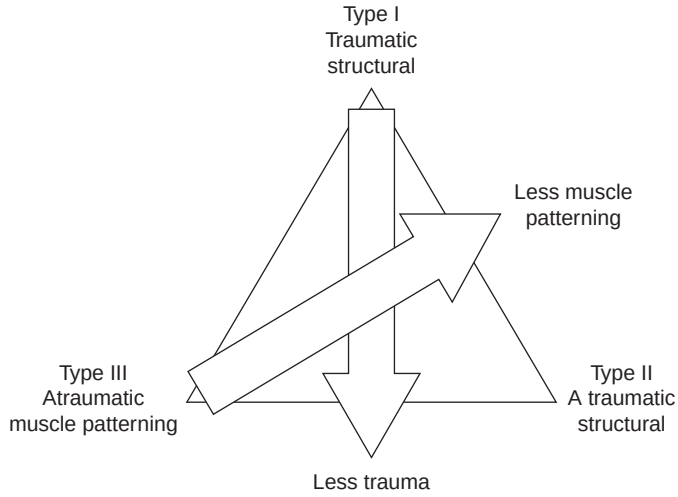

Figure 23.2 Stanmore classification(Bayley triangle)

Thomas and Matsen

- TUBS: **T**raumatic **U**nidirectional with a **B**ankart lesion. Treat with **S**urgery
- AMBRI: **A**traumatic **M**ultidirectional **B**ilateral. Treated with **R**ehabilitation and, if surgery is required, an **I**nferior capsular shift and closure of the rotator interval

Bayley (Stanmore) (Figure 23.2)

History

There is a definite history of trauma elicited in acute dislocations. Electrical shocks and seizures are usually associated with posterior dislocations because the combined strength of the internal rotators overwhelms the external rotators. Atraumatic instability causes discomfort in activities of daily living with discomfort even at rest.

Assessment

An acutely dislocated joint is very painful with muscles in spasm. The humeral head may be palpable depending on the direction of dislocation along with asymmetry of shoulder contour. Neurovascular status of the extremity should be assessed before and after any intervention. Stability tests useful for demonstration of instability include the fulcrum test, apprehension test, Jobe's relocation test and the jerk test. The laxity tests include the drawer test and sulcus test, and are used to compare with the laxity of the normal contralateral shoulder. Testing of the strength of muscles around the shoulder should complement the examination.

Investigations

- Radiographs: In the acute setting, an AP view, scapular lateral view and/or axillary view are taken to attain information with regard to direction of dislocation, associated fractures and possible blocks for relocation. The Stryker notch view demonstrates humeral head defects. The West Point axillary view demonstrates glenoid defects

- CT scan is useful for greater detail of anatomy
- MR arthrogram can reveal associated rotator cuff and labral tears
- Arthroscopy is invasive but is useful to assess structural damage in the shoulder accurately and is a dynamic investigation
- Electromyography (EMG) is useful in muscle patterning instability

Treatment

Acute dislocations

Acute dislocations should be reduced as gently and expeditiously as possible after a complete set of radiographs. Various techniques of reduction used are

- Hippocratic method – Foot in the axilla (historical)
- Traction–countertraction method
- Stimson method: Prone with application of downward traction
- Kocher method: Redislocation rates are slightly higher with this technique. Considered dangerous by some surgeons due to risk of iatrogenic injury, particularly humeral fracture
- Spaso technique: Supine with longitudinal traction and external rotation

Posterior dislocation is reduced with longitudinal lateral traction followed by external rotation. Unreduced and chronic dislocations need open reduction. Chronic dislocation with minimal discomfort and good functional range can be managed with supervised neglect. Dislocation associated with greater tuberosity fracture has a lower rate of recurrence. The age of the patient at the time of initial dislocation is the major determinant of recurrent instability, with rates as high as 90% in patients <20 years. The dislocation rate decreases as the age of the patient increases. Dislocation in those of advanced age is commonly associated with rotator cuff tear. The important elements in post-reduction management are protection and rehabilitation to prevent recurrent instability. Immobilization in external rotation rather than the standard internal rotation position has been shown to have good results in a study by Itoi et al.[10]

When there is a dislocation – Reduce it, check if it is congruent (incongruent = bony or soft-tissue interposition), immobilize for 3–4 weeks to allow soft-tissue healing to commence, then assess clinically for stability. If stable, rehab with physiotherapy. If unstable, assess with MR arthroscopy for lesions that may require surgical intervention such as HAGL (humeral avulsion of inferior glenohumeral ligament, Hill–Sachs or Bankart). If the dislocation caused an axillary nerve palsy, check this at 3–4 weeks and, if no signs of recovery, request nerve conduction studies and refer to a nerve surgeon.

Recurrent instability

Principles of treatment

- Structural instability – Traumatic or atraumatic: Surgical stabilisation
- Non-structural instability – Muscle patterning: Non-operative management
- Mixed structural and muscle patterning: Non-operative management is the first line treatment. Surgery is indicated if the muscle patterning component can be corrected and the underlying structural instability remains a problem

Surgical interventions for structural anterior instability

Anatomical repairs

- Bankart repair: Reattachment of the Bankart lesion to the margin of the glenoid either by open or arthroscopic technique
- Large Hill–Sachs lesion: Transfer of infraspinatus to fill the defect to help prevent redislocation

Non-anatomical repairs

- Latarjet procedure: Transfer of the coracoid and conjoint tendon to the anterior glenoid rim
- Magnuson–Stack procedure: Advancement of the subscapularis
- Putti–Platt procedure: Imbrication and shortening of the subscapularis
- Glenoid or humeral osteotomies: Particularly if there is excessive anteversion of the glenohumeral joint

Surgical interventions for structural posterior instability

- Reverse Bankart procedure and capsular shift
- Reverse Putti–Platt procedure
- Boyd–Sisk procedure: Transfer of long head of biceps to posterior glenoid
- A reverse Hill–Sachs defect is managed by transfer of subscapularis or the lesser tuberosity

Treatment for non-structural instability and muscle patterning

Needs a multidisciplinary approach. The initial step is to assess the muscle patterning/function and the direction of instability. EMG studies are useful for evaluation. Patients with a muscle patterning disorder have a muscle coordination problem that forms the basis of the biofeedback exercises concentrating on improving joint position sense and relearning correct muscle movement patterns. The aim is to strengthen the shoulder musculature to achieve concavity compression.

After correction of muscle patterning, if the patient fails to respond to vigorous strengthening exercises, endurance and coordination, the surgical option is an inferior capsular shift procedure either by open or arthroscopic method. The principle is to tighten the anterior, inferior and posterior aspects of the capsule symmetrically by advancing its humeral attachment.

Neurological problems around the shoulder

Suprascapular nerve (C5, C6)

This comes off the upper trunk (C5, C6) of the brachial plexus and runs through the suprascapular notch to supply supraspinatus and then via the spinoglenoid notch to supply

infraspinatus. Specifically at the spinoglenoid notch the suprascapular nerve may be compressed by a ganglion associated with a labral tear[11].

- Compression at suprascapular notch: Affects both suprascapular and infrascapular muscles
- Compression at spinoglenoid notch: Affects infraspinatus

Clinical features

- Posterior and lateral shoulder pain
- Wasting of supraspinatus and weakness of abduction
- Wasting of infraspinatus and weakness of external rotation

Management

EMG and NCS are useful for diagnosis. A direct superior trapezius muscle splitting approach is used to decompress the suprascapular notch. Arthroscopic debridement is used for ganglia at the spinoglenoid notch.

Long thoracic nerve (C5, C6, C7)

This runs in close relation with the first rib and supplies the serratus anterior muscle. The serratus anterior pulls the scapula inferolaterally and rotates the inferior angle laterally.

Clinical features

Serratus anterior weakness results in **medial** winging of the scapula where there is superomedial elevation and the inferior angle is rotated medially. This is different from spinal accessory nerve involvement resulting in trapezius weakness and **lateral** winging where there is scapular translation inferolaterally and the inferior angle is rotated laterally.

Management

Observation is the standard treatment for idiopathic and non-penetrating trauma.

Surgical options include neurolysis in the early stages, and neurotisation and reconstructive procedures, including tendon transfers (pectoralis major).

Spinal accessory nerve

This passes through the sternocleidomastoid muscle and travels through the posterior triangle onto the medial border of the scapula. It supplies the sternocleidomastoid and trapezius muscles. Common causes of injury are penetrating or blunt trauma and injury during surgical dissection in the posterior triangle of neck.

Clinical features

- Wasting of sternocleidomastoid and trapezius
- Lateral scapular winging
- Secondary impingement

Management

- Non-operative treatment if injury is >12 months old with good compensation
- Surgical exploration within the first 6 months of injury
- Surgical options include neurolysis, direct repair or nerve grafting, dynamic muscle transfer techniques and scapulothoracic fusion when reconstruction is not possible

Thoracic outlet syndrome

This is a diagnosis of exclusion and is based on history and symptoms. Common causes are cervical rib and anomalies of scalenus muscle.

- Neurogenic type

  Upper plexus type
  Lower plexus type
- Vascular type

Sites of compression

- As the plexus passes over the first rib
- Under the clavicle by the subclavius tendon
- Underneath the conjoint tendon inserting into the coracoid process

Clinical features

- Pain in the shoulder and neck region radiating to the forearm and hand
- Paraesthesia radiating along the arm
- Loss of sensation of little and ring fingers
- Change in colour of hands or chronically reduced pulse
- Provocative tests include Adson's test, Wright's test, Roo's test
  - Adson's test: head extended and rotated towards affected side. Ipsilateral arm extended and abducted 30° and palpate the radial pulse. The test is positive if the pulse disappears when the patient takes a deep breath and holds it
  - Wright's test: Shoulder abducted and externally rotated, head rotated away from the affected side and palpate the radial pulse. The test is positive if this position diminishes the radial pulse
  - Roo's test: shoulders braced back, elbows flexed 90°, fingers rapidly flexed and extended overhead for 1 minute. The test is positive if this reproduces the symptoms

Investigations

- Plain radiographs of chest and cervical spine
- CT for suspected bony anomaly not visible on x-ray
- MRI for suspected cervical pathology, tumour or radiation plexitis
- Doppler, arteriography/venography if vascular symptoms predominate
- EMG/NCS if neurological symptoms predominate

Treatment

- Non-operative treatment is with physiotherapy to correct postural imbalances

- Surgical treatment involves resection of the first rib (cervical rib if present) with release or excision of the anterior and middle scalene muscles and excision of any anomalous structures. This can be done through transaxillary or supraclavicular approaches
- Venous thrombolysis or arterial thrombectomy and vascular reconstruction are urgently indicated in acute presentations

Quadrilateral space syndrome

This is caused by compression of the axillary nerve and posterior humeral circumflex artery in the quadrilateral space, which is bounded by the teres major and minor muscles, the humeral shaft and the long head of the triceps[12].

Clinical features

- Affects the dominant arm in young adults
- Poorly localized anterior and lateral shoulder pain
- Tenderness in the quadrilateral space near teres minor insertion

Investigations

- MRI scan: Selective atrophy of teres minor muscle/posterior paralabral cyst
- Arteriogram: Occlusion of posterior circumflex humeral artery with the arm in abduction and external rotation caused by oblique fibrous bands

Treatment

- Decompression of posterior paralabral cyst
- Release of oblique fibrous bands through posterior approach

Brachial neuritis (Parsonage–Turner syndrome)

This is a non-traumatic condition which is immune-mediated or inflammatory in nature.

Clinical features

- Severe, acute onset of periscapular pain
- Motor weakness in shoulder and arm with sensory loss

Investigations

- EMG/NCS: Patchy, multifocal neurological process
- MRI: High signal intensity in affected muscles

Treatment is supportive and is directed towards reducing pain. It has a favourable prognosis, though some residual defects may remain.

Examination corner

Adult pathology viva

EXAMINER: (Showing photographs of right shoulder.) This is a 45-year-old patient. Describe the findings.

CANDIDATE: There is wasting of deltoid and pectoralis major and medial winging of the scapula.

EXAMINER: What do you mean by medial winging?

CANDIDATE: It is caused by serratus anterior weakness. The functioning trapezius pulls the scapula superomedially and rotates the inferior angle medially.

EXAMINER: This patient has pain around the shoulder and this problem started spontaneously 7 years ago and motor symptoms remain non-progressive. What do you think is going on?

CANDIDATE: It could be brachial plexus neuritis.

EXAMINER: How could you help this patient?

CANDIDATE: I would examine the patient's shoulder to assess the functional restrictions and would like to know the patient's expectations.

EXAMINER: He has full range of movements and full cuff function. He wants to improve the appearance of the shoulder.

CANDIDATE: As he has no functional restriction, I would not offer any surgical treatment. I would advise against any surgery to improve the appearance. If he needs it I would refer him to a pain management team to control his chronic neuropathic pain.

EXAMINER: Do you think this muscle wasting will improve?

CANDIDATE: Unlikely, as it has remained the same for the last 7 years.

EXAMINER: (Showing clinical photograph of the left shoulder of a 20-year-old male.) What do you see?

CANDIDATE: I can see a scar measuring 4 cm in the posterior triangle of the neck with marked wasting of the trapezius.

EXAMINER: Would you expect to see winging of this patient's scapula?

CANDIDATE: Yes, but this time I would expect lateral winging secondary to weakness of trapezius and unopposed action serratus anterior.

EXAMINER: Is it a brachial plexus injury?

CANDIDATE: No. I suspect an injury to the spinal accessory nerve. The scar in his neck suggests either penetrating trauma or previous neck surgery.

EXAMINER: How do you manage this patient?

CANDIDATE: That depends on the onset of symptoms. How and when did the problem start?

EXAMINER: It started after he had a lymph node biopsy last month.

CANDIDATE: In that case I would recommend urgent exploration and direct repair or nerve grafting as soon as possible.

Miscellaneous conditions

Long head of biceps (LHB)

The LHB originates from the supraglenoid tubercle and the glenoid labrum. It helps in glenohumeral compression in abduction–external rotation. It also helps to prevent proximal humeral migration, especially in cuff dysfunction. LHB tendinosis is mostly associated with other shoulder pathology. Primary tendinosis is rare and is associated with trauma in young adults.

Clinical features

- Pain in the anterior aspect of the shoulder aggravated by overhead activity
- Palpable snap or click with internal to external rotation in overhead position is associated with biceps instability
- Tenderness in bicipital groove, moving laterally with external rotation and medially with internal rotation
- Rupture causes acute pain and audible pop, accompanied by balling up of the biceps known as 'Popeye sign'
- Provocative tests include Speed's test, Yergason's test and biceps instability test

Investigations

- Plain radiography
- Ultrasound scan
- MR arthrogram to identify associated SLAP lesions
- Arthroscopy can diagnose and treat most of the lesions

Treatment

- Non-operative treatment for tendinosis includes use of NSAIDs, steroids and physiotherapy for strengthening rotator cuff muscles
- Intra-articular tendinosis

  Debridement: <50% tendon involved
  Tenotomy or tenodesis: >50% involved
- Subluxation/dislocation: Tenotomy or tenodesis

SLAP lesions

Superior labrum from anterior to posterior (SLAP) tears are usually associated with glenohumeral instability (inferior to the equator of glenoid) and rotator cuff tears (superior to the equator of glenoid). MR arthrogram is useful for diagnosis.

Snyder classification[13]

- Type I: Fraying of superior labrum but not completely detached
- Type II: Detachment of labrum and biceps anchor
- Type III: Torn labrum turned down into the joint (bucket-handle tear)
- Type IV: Bucket-handle type tears that extend up into the biceps tendon
- Complex types involve combination of two or more types

Clinical features

- History of trauma
- Transient episodes of weakness and numbness ('dead arm')
- Biceps tenderness
- Provocative tests include O'Brien test, anterior slide test and crank test
  - O'Brien test: Shoulder elevated to 90°, adducted 10–15° across the body, internally rotated. Patient resists downward pressure on the forearm by examiner. The test is repeated in external rotation, which should relieve any pain felt on testing. Pain in the AC joint region is AC joint related. Deeper shoulder pain implies labral pathology

Treatment

- Type I: Simple debridement
- Type II: Arthroscopic repair
- Type III: Excised with biceps anchor securely fixed
- Type IV: If more than one-third of biceps tendon is involved, suture and repair; debridement if less
- Complex tears: Repair of type II lesion and resection of other lesions

Frozen shoulder

Frozen shoulder, also known as adhesive capsulitis, is a glenohumeral joint contracture that occurs after minimal or no trauma and arises as a fibrotic process intrinsic to the capsule. The histological is similar to that of Dupuytren's disease, with proliferation of fibroblasts, transformation to myofibroblasts and increased collagen deposition. However, the clinical picture in both these conditions is different, with Dupuytren's being a progressive and pain-free deformity of the fingers and frozen shoulder being a painful and self-limiting condition. It is much more common in diabetic patients and is also associated with thyroid dysfunction.

Criteria for diagnosis of frozen shoulder

- History of restricted shoulder range of motion without an inciting factor
- Global stiffness – Markedly affecting external rotation
- Plain radiographs with normal joint space and no focal periarticular abnormality. Osteoarthritis is an important differential diagnosis to exclude

Natural history

- Phase 1 – Painful 'freezing' phase: Aching pain begins at night and persists during the day. Ache is unrelated to activity, it is worse at rest and disturbs sleep. The shoulder is held in adduction and internal rotation for comfort
- Phase 2 – Progressive stiffness: 'frozen' phase: Lasts 3–12 months. There is global restriction with restricted activities of daily living. With time pain diminishes with a narrow comfort zone
- Phase 3 – Resolution: 'thawing' phase: Slow gain in motion and comfort over 12–42 months. Complete recovery may not occur

Shoulder examination will reveal diffuse tenderness. Active and passive range of motion is tested in six standard motion arcs and compared with the opposite normal shoulder. Post-traumatic stiffness has asymmetrical restriction of shoulder range of movements depending on the site of injury.

Investigations

- Radiographs: To differentiate primary and secondary frozen shoulder
- MRI: If required to differentiate from cuff/labral/bony pathology

- Arthroscopy: Demonstrates a tight anterior capsule which can be released to improve range of movement

Management

- Supportive treatment includes use of analgesics, topical treatment with ultrasound, TENS and intra-articular steroid injections
- Gentle stretching exercises with or without strengthening
- Manipulative therapy to release adhesions. A safe sequence for shoulder manipulation – Flexion, extension, abduction and adduction, external and internal rotation (FEAR) under GA or regional block
- Surgical release of adhesions arthroscopically, releasing the rotator interval, middle glenohumeral ligament and coracohumeral ligament to improve the range of movement
- Rehabilitation after intervention is crucial in preventing recurrence of stiffness

Calcific tendonitis

Calcific tendonitis is a condition of unknown aetiology characterized by the build up of calcium hydroxyapatite crystals within the tendons, which undergoes spontaneous resorption with subsequent healing of the tendon. The most common site of occurrence is within the supraspinatus tendon and at a location 1.5–2.0 cm away from the tendon insertion on the greater tuberosity.

Stages[14]

- Pre-calcific stage: Fibrocartilaginous transformation occurs at the site of predilection triggered by hypoxia
- Calcific stage: Subdivided into formative and resorptive phases
- Formative phase: Calcium crystals are deposited in matrix vesicles
- Resorptive phase: Characterized by appearance of thin-walled vascular channels at the deposit
- Post-calcific stage: Granulation tissue with young fibroblasts remodels the space

Clinical features include pain which is most pronounced during the resorptive phase, a reduced range of motion and in long-standing cases, atrophy of the supraspinatus muscle. It is not associated with any abnormalities in calcium and phosphorus metabolism.

Investigations

- Radiographs for diagnosis, initial assessment and follow-up
- Ultrasound is more sensitive and is used for diagnosis and treatment

Treatment

- The first line of treatment is conservative, with emphasis on pain control followed by range of motion leading to strengthening exercises
- Needling and lavage under ultrasound or arthroscopic guidance
- Extracorporeal shock wave lithotripsy (ESWL)

Examination corner

Adult pathology viva

EXAMINER: A 70-year-old gentleman is seen in fracture clinic with severe pain in the right arm. What do you seen on his x-rays?

CANDIDATE: These are AP and lateral plain radiographs of this gentleman's right humerus including the shoulder and elbow. There is a lytic lesion in the humeral diaphysis with a wide zone of transition and extensive cortical involvement.

EXAMINER: What do you think it could be?

CANDIDATE: This could be a bone tumour, either primary or secondary, infection or myeloma.

EXAMINER: How will you make the diagnosis?

CANDIDATE: After a full history and examination I would request a bone scan to determine whether this is an isolated lesion. I would request a myeloma screen, an MRI scan of the lesion and a CT scan of the chest, abdomen and pelvis to look for a primary.

EXAMINER: This is an isolated, painful secondary bone tumour. There are multiple metastases elsewhere from what looks like a renal primary. How are you going to manage this lesion?

CANDIDATE: I would discuss with the multidisciplinary team including the oncologists to see whether radiotherapy is an option.

EXAMINER: Would you consider prophylactically nailing this humerus?

CANDIDATE: That would depend upon the Mirel's score which is based on four factors: Pain, location, involvement of cortex and whether the lesion is lytic or blastic.

EXAMINER: What is his Mirel's score?

CANDIDATE: His pain is severe so that's 3. It is the upper limb so that's 1. It is lytic so that's 3 and based on these plain x-rays it involves more than two thirds of the cortex so that would be 3. Overall his score is 10. The threshold for intervention would be a score of 8 or more.

EXAMINER: How would you technically go about nailing this humerus?

CANDIDATE: I would position the patient in the beach chair position and use a deltoid splitting approach onto the rotator cuff. I would carefully split the cuff muscle longitudinally and protect it during the procedure. I would use a long nail to stabilise the whole bone and I would repair the cuff split at the end.

EXAMINER: OK, let's say this is a different patient now and they have a similar lesion but you have confimed that this is an isolated metastasis from a renal primary. How does this change things?

CANDIDATE: With an isolated metastasis it may be possible to treat curatively. I would liase with the renal and oncology teams to determine whether the renal tumour was curatively resectable and I would liaise with the local bone tumour centre for management of the metastasis.

EXAMINER: Are there any particular concerns when operating on renal mets?

CANDIDATE: They can be highly vascular and may benefit from embolisation preoperatively.

Glenohumeral arthrodesis

Shoulder arthrodesis is an end-stage salvage option for the failing, painful joint that cannot undergo or has failed reconstruction.

Indications

- Paralysis of deltoid and rotator cuff
- Infection with loss of glenohumeral cartilage
- Refractory instability
- Failed reconstructive procedures

Contraindications

- Patients who lack functional scapulothoracic motion
- Paralysis of periscapular muscles
- High risk of pseudarthrosis – Charcot arthropathy
- Advanced bilateral shoulder disease

Patients undergoing shoulder arthrodesis require preoperative counselling for a full understanding of their postoperative limitations and functional capacities. The optimal position for arthrodesis is '30–30–30': 30° of forward flexion, 30° of abduction and 30° of internal rotation, with modifications based on patient body size or other patient-specific factors. The aim is for the patient to be able to get their hand to their mouth.\

Techniques

Techniques of shoulder arthrodesis are broadly divided into:

- Extra-articular
- Intra-articular offers the simplest and most direct method
- Combination of both

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Chapter 24

Brachial plexus core topics

David R. Dickson and Chye Yew Ng

Introduction

- World-wide, motorcycle accidents are the leading cause of adult traumatic brachial plexus injury (BPI), which typically occurs in young males
- They are relatively uncommon injuries and have been estimated to occur in only 1.2% of polytrauma patients[2]
- There are two basic clinical presentations: Partial palsy involving the upper roots (C5, 6 or C5, 6, 7) and total palsy (C5–T1)
- Infraclavicular BPI to the terminal branches may occur secondary to shoulder trauma[1]
- The incidence of obstetric BPI (OBPI) in the Western World is thankfully low *(while not impossible, you are unlikely to come across children with OBPI in the examination)*
- Narakas' Rules of Seven 70s[3]
  - 70% of brachial plexus injuries are due to road traffic accidents (RTAs)
    - 70% of these involved motorbikes
  - 70% have multiple injuries
  - 70% are supraclavicular injuries
    - 70% of these have at least 1 root avulsion
  - 70% of root avulsion involve the lower plexus
  - 70% of root avulsions will leave the patient with chronic pain

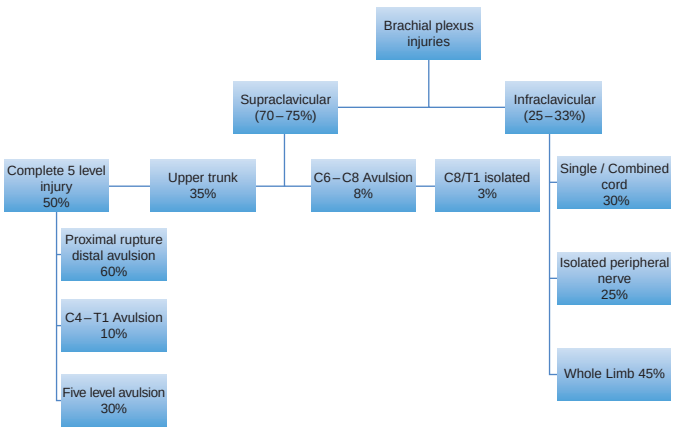


Figure 24.1 Distribution and location of BPI

Aetiology

- Arm in upward traction (hanging onto a branch) – Lower root avulsion/traction
- Arm in downward traction (weight falling onto shoulder) – Upper root avulsion/traction
- Mechanisms
  - RTA (particularly motor cyclists)
  - Birth trauma (shoulder dystonia, large infants, maternal obesity, diabetes, cephalopelvic disproportion and forceps delivery)
  - Shoulder girdle trauma (dislocation, proximal humeral fractures, hyperextension injury)
  - Gunshots
  - Iatrogenous (e.g. clavicle plating)

Candidates should be aware of the classification but it is not necessary to memorise the specific numbers.

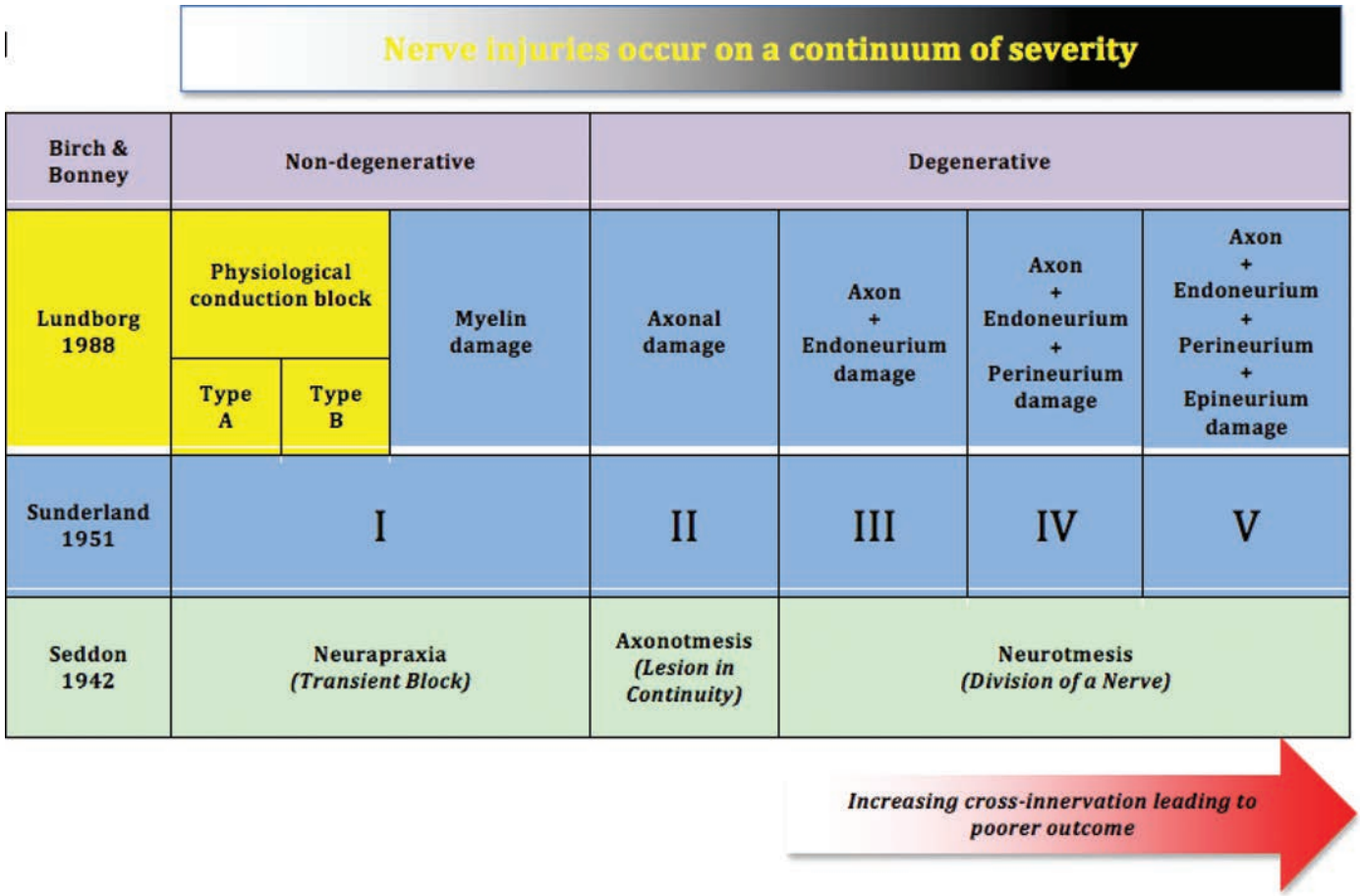
Leffert classification

I Open
II Closed
  IIA Supraclavicular
    Pre-ganglionic
    Post-ganglionic
  IIB Infraclavicular
III Radiotherapy
IV Obstetric
  IVA Erb's (upper root)
  IVB Klumpke's (lower root)
  IVC Mixed

Pathoanatomy

- Traction can result in three main injury patterns
  - Root avulsion (pre-ganglionic injury)
  - Stretch
  - Rupture (post-ganglionic injury)
- Any combination of these injuries can occur within the same patient

Table 24.1 Classifications of Nerve Injuries



- Myelinated peripheral nerve fibres are surrounded by Schwann cells and a loose vascular tissue called the **endoneurium**
- Individual nerve fibres are collected into bundles called fascicles which are covered by the **perineurium**
- Each nerve has a number of fascicles which are surrounded by the **epineurium**
- Lundborg introduced the concept of physiological conduction block*:
- Type A
  - Intraneural circulatory arrest
  - Metabolic block with no nerve fibre pathology
  - Immediately reversible
- Type B
  - Intraneural oedema
  - Increased endoneurial fluid pressure
  - Reversible within days or weeks
- Important to appreciate that any nerve injury is often mixed and there could be a spectrum of severity in any lesion (Sunderland 'VI')
- A pragmatic distinction is between a non-degenerative (Sunderland I) or a degenerative (Sunderland II–V) lesion (Table 24.1)

Anatomy of the brachial plexus

The brachial plexus is formed from the **ventral** primary rami of C5–T1 spinal nerves. A small number of patients can have variable contributions from C4 ('pre-fixed' plexus) and T2 ('post-fixed' plexus).

- It is organised into five components: Roots, trunks, divisions, cords and branches. (Remember the mnemonic **R**ob **T**aylor **D**rinks **C**old **B**eer)
  - Five roots
  - Three trunks (upper, middle, lower)
  - Six divisions (two from each trunk)
  - Three cords (posterior, lateral, medial)
  - Multiple branches
- The cervical roots are composed of ventral (motor) and dorsal (sensory) roots from the spinal cord
- The motor nerve cell bodies lie within the spinal cord

- The dorsal root ganglion holds the cell bodies of the sensory neurons, an injury proximal to this is described as pre-ganglionic
- The two roots combine to form the spinal nerve proper which then exits through the spinal foramen
- The rootlets have no protective layer as they originate from the spinal cord which contributes to their vulnerability to avulsion injury
- The roots, however, have a protective layer formed by the dura and are able to move freely within the foramen
- The upper roots of C5, C6 and C7 are tethered to their respective transverse processes, whilst C8 and T1 are not. It is thought that this may explain the greater degree of avulsion injuries seen at C8 and T1
- The roots (ventral rami) then pass between the scalenus anterior and medius muscles
- The roots then merge to form the trunks in the posterior triangle of the neck
  - C5 and C6 form the upper trunk. Erb's point where C5 and C6 become confluent
  - C7 continues as the middle trunk
  - C8 and T1 form the lower trunk
- The trunks then divide into anterior and posterior divisions (behind the clavicle), which in turn form three cords (named in relation to the axillary artery) –
  - Anterior divisions of the upper and middle trunks form the lateral cord
  - Posterior divisions of all the trunks form the posterior cord
  - Anterior division of the lower trunk forms the medial cord
  - The cords give rise to the terminal branches

Branches

The three branches from the roots are:

- Long thoracic nerve
- Dorsal scapular nerve
- Nerve to subclavius

There is one branch from the trunks:

- Suprascapular nerve

None from divisions

('3–5–5 rule' – Describes branches from cords).

- There are three branches from the lateral cord:
  - Lateral pectoral nerve
  - Lateral head of median nerve
  - Musculocutaneous nerve
- There are five branches from the medial cord:
  - Medial pectoral nerve
  - Medial cutaneous nerve of the arm
  - Medial cutaneous nerve of the forearm
  - Medial head of median nerve
  - Ulnar nerve

(The ulnar nerve is the most important branch and the rest of the branches begin with the word medial.)

- Five branches of the posterior cord are:
  - Subscapular nerve – Upper
  - Subscapular nerve – Lower
  - Thoracodorsal nerve
  - Axillary nerve
  - Radial nerve

(Acronym: 2 STAR – Two **S**ubscapular nerves, **T**horacodorsal nerve, **A**xillary nerve and **R**adial nerve)

Tips on approaching clinical examination for FRCS

- The challenge lies in localisation of the lesion. This can only be achieved by having a thorough understanding of the anatomy of the brachial plexus. The extent, level, severity and chronicity of a brachial plexus injury will determine the physical signs that are manifested
- While there are over 50 named muscles to be tested, it is not practical (and there is not enough time in FRCS exam) to allow you to examine every single muscle in the upper limb. You then have to rely on pattern recognition
- Useful to have a glance at the hand which would give you clues as to whether this is partial palsy (good hand) or total palsy (poor hand)
- A totally flail arm and hand represent total palsy
- Patients who have had reconstructive surgery (i.e. have scars) may present greater challenges during examination as they may have variable degree of recovery

Pre-ganglionic vs post-ganglionic lesion

- A potential pitfall in candidates is the desire to arrive at a narrow and specific diagnosis right at the start. However, it is more important to demonstrate your logical thinking and deduction during the examination
- While it is tempting to establish whether this is a pre-ganglionic or post-ganglionic lesion, remember that no clinical, radiological or neurophysiological evidence is absolutely accurate. Often this can only be established at surgery
- The following are clues that you may identify and mention to the examiners in order to demonstrate your *higher order thinking*
  - Horner's syndrome
    - *Partial ptosis* of the upper eyelid
    - *Miosis* (constricted pupil)
    - *Anhidrosis* (loss of sweating on one half of the face)
    - *Enophthalmos* (eye appears sunken)

- The T1 root lies close to the T1 sympathetic ganglion. Evidence of injury to the T1 sympathetic chain as evidenced by a Horner's syndrome would infer that the T1 root has probably been injured
- If rhomboids or serratus anterior are weak then a pre-ganglionic injury should be suspected
- If chest x-ray is shown, look for elevated (paralysed) hemi-diaphragm (phrenic nerve palsy C3, C4, C5)
- Fractures of the transverse processes of the cervical vertebrae or a fractured first rib indicate a high-energy injury with likely intradural injury of the lower two roots
- Scapulothoracic dissociation is often associated with root avulsion and major vascular injury

Clinical examination

- Inspection
  - Best to start with the patient stood with both arms and torso exposed
  - Look at the face for Horner's syndrome
  - Look for surgical scars
  - Muscle wasting – Shoulder girdle, arm, forearm and hand
  - Posture of the limb
  - Scapula winging
- Exclude fixed contractures by gentle passive movements
- Motor testing
  - Requires knowledge of the Medical Research Council (MRC) grading
    - 0 – No contraction
    - 1 – Flicker
    - 2 – Active motion (gravity eliminated)
    - 3 – Active motion (against gravity only)
    - 4 – Active motion (against resistance)
    - 5 – Normal power
  - If a muscle is weak, repeat testing in the horizontal plane in order to eliminate gravity, e.g. abducting the shoulder to test elbow flexion/extension power
  - Muscle testing is an active process involving
    - Look (for contraction and movement of the limb)
    - Feel (for contracted muscle/tendon)
    - Move (to test resistance)
  - Be systematic. Start proximally and work distally

Standing from the back

- Trapezius (spinal accessory – XI, C3, C4)
  - *Can you shrug your shoulders?*
- Rhomboids (dorsal scapular nerve – C4, C5)
  - *Push your shoulder blades together?*
- Serratus anterior (long thoracic nerve – C5, C6, C7)
  - The classic test is wall-press test
  - In BPI, the patient may be unable to lift the arm
  - The arm should be supported by the examiner with one hand and the patient asked to push forward as if trying to open a door. At the same time the examiner should hold the lower pole of the scapula with another hand
- Latissimus dorsi (thoracodorsal nerve – C6, C7, C8)
  - While the arm is supported in a flexed position, ask the patient to push down (while the examiner palpates for musle contraction)
- Deltoids (axillary nerve – C5, C6)
  - Extend, abduct and flex the shoulder to test the posterior, middle and anterior parts respectively (unless the muscle is clearly wasted)
  - Demonstrate specific signs (if isolated nerve palsy suspected)
    - Swallow-tail sign
      - The patient is asked to extend the shoulder while bending the trunk forward. A result of 20° or greater of extension lag relative to the normal side indicates a positive sign
    - Abduction internal rotation
      - Actively and maximally abduct the shoulder in internal rotation with the elbow flexed. Abduction lag relative to the normal side indicates a positive sign

Standing from the front

- Pectoralis major (lateral and medial pectoral nerves)
  - Clavicular head (C5, C6)
    - Atrophy would imply lateral cord injury
    - Ask the patient to touch their contralateral shoulder (and the examiner palpates for evidence of contraction)
  - Sternocostal head (C7, C8, T1)
    - Atrophy would imply medial cord injury
    - Ask the patient to push against the hip (and the examiner palpates the axillary fold)
- Rotator cuffs
  - Supraspinatus (suprascapular nerve – C5, C6)
    - Test shoulder abduction in the scapular plane with the thumb pointing downwards
  - Infraspinatus (suprascapular nerve – C5, C6)
    - Test external rotation with the shoulder in adduction and the elbow flexed
  - Teres minor (axillary nerve – C5, C6)

  - Test external rotation with the shoulder in abduction and the elbow flexed
- Subscapularis (upper and lower subscapular nerves – C5, C6, C7)
  - Belly-press sign. Ask the patient to bring the elbows forward while pressing the belly. A flexed wrist relative to the normal side indicates a positive sign

Next, proceed with the following composite testings to demonstrate the myotomes (levels) involved (accept some degree of variability in the books you have read – As not all humans are born the same!)

- Elbow flexion (C5, C6)
- Elbow extension (C7, C8)
- Forearm supination (C6)
- Forearm pronation (C7, C8)
- Wrist flexion/extension (C6, C7)
- MCP joint flexion/extension (C7, C8)
- Grip (C8)
- Fingers abduction (T1)

- Sensory testing
  - Establish normal sensation in an uninjured area (such as forehead or sternum)
  - First, assess the dermatomes and then if felt necessary, examine according to the terminal branch distribution
- Check radial pulse and offer to test the reflexes

Investigations

Radiology

- Key radiographs are cervical spine, chest and shoulder
  - Avulsion fractures of the tranverse processes are associated with root avulsions
  - Chest radiographs
    - Rib fractures (important if considering intercostal nerve transfers)
    - Look for apical pleural cap which may be associated with first rib fracture
    - Paralysis of the hemidiaphragm (phrenic nerve palsy)
- CT myelography - invasive
  - The gold standard in diagnosing root avulsions
- MRI – Non-invasive
  - Findings consistent with a severe injury include
    - pseudomeningocele (T_1-weighting)
    - empty root sleeves (T_2-weighting)
    - cord shift away from the midline
  - May show subtle denervation changes in muscles

Histamine test

- Of historical interest only
- Used as a diagnostic tool to differentiate between pre-ganglionic and post-ganglionic lesions
- Histamine is placed on the skin. The skin is then scratched. A normal reaction with an area of skin served with an intact nerve is the triple repsonse
- The triple response is vasodilatation, wheal formation and flare response
- When the nerve is damaged proximal to the dorsal root ganglion there is also a normal response but the skin is anaesthetic
- If the nerve damage is distal to the dorsal root ganglion then there is vasodilatation and wheal formation but no flare response. This is because a flare response requires an intact axon in continuity with its cell body (located within the dorsal root ganglion)

Neurophysiology

- Remember this is an aid to the overall diagnostic (and prognostic) process
- Perform at least 2–3 weeks after the injury (earlier studies may be falsely reassuring)
- Communication between the surgeon and the neurophysiologist is crucial to the correct interpretation of the results
- Nerve conduction studies (NCS)
  - Can evaluate both sensory and motor components
  - Diagnose root avulsions – SNAPs will be normal despite loss of sensation. In a post-ganglionic injury the SNAPs will also be absent
- Electromyography (EMG)
  - After a few days the denervated muscle shows decreased motor unit potentials (MUPs) which fire at a higher rate
  - After 4–6 weeks the muscles begin firing at their own inherent 'pacemaker' which appears as fibrillations on the EMG
  - Afer 8–12 weeks no MUPs will appear if the nerve injury is complete
  - Can show evidence of muscle recovery long before there is any clinical activity

Management

- Three key questions
  1. Who needs surgery
  2. When surgery should be done
  3. What surgery – Prioritizing the restoration of which motor functions
- Who requires surgery?
  - Those who have no hope of spontaneous recovery – Such as root avulsions
  - Those in whom there is no clinical and/or neurophysiological evidence of improvement after serial examinations
- When should you proceed to surgery?
  - Following nerve avulsion/rupture, apart from end-organ degeneration (motor end-plate), there is also central neuronal death by apoptosis *('A race against time')*
  - There is, thus, a trend towards early exploration for diagnostic and prognostic purposes
  - Some would still allow a period of time for spontaneous recovery to occur. Opinions vary but 3-months post injury is generally accepted as the key timepoint to decide on intervention

- Surgical priorities
  - Restore elbow flexion
  - Restore shoulder abduction (stability)
  - Restore hand function

Range of nerve surgery

- Neurolysis
  - Involves release of scar tissue around a nerve in continuity
  - Scar may form outwith or inside the epineurium, preventing recovery or causing pain
- Nerve repair
  - Primary end-to-end repair is rarely possible, except in those with an acute laceration and a narrow zone of injury
- Nerve graft
  - Prerequisites for successful nerve grafting include healthy proximal stump, tension-free neurorrhaphy, good tissue bed and reconstruction performed in a suitable time frame
  - Autograft (sural) remains the gold standard although decellularised allograft is also available
- Nerve transfer (or neurotisation)
  - Involves the transfer of a functioning fascicle or nerve branch (expendable donor) to a denervated muscle
  - Principles of motor nerve transfer
    - Donor nerve near target motor end plates
    - Expendable donor nerve
    - Pure motor donor nerve
    - Donor-recipient size match
    - Donor function synergy with recipient function
    - Motor re-education improves function
  - Some commonly performed nerve transfers
    - Restoration of elbow flexion
      - Ulnar nerve fascicle (FCU) to musculocutaneous nerve branch (biceps)
      - Median nerve fascicle (FCR) to musculocutaneous nerve branch (brachialis)
    - Restoration of shoulder function and stability
      - Spinal accessory nerve to suprascapular nerve
      - Radial nerve branch (triceps) to axillary nerve (deltoid)

Salvage/late reconstructions

- In delayed presentations (>1 year) or when the outcome of nerve surgery is likely to be poor, consider the following reconstructive options
  - Arthrodesis
    - Shoulder
    - Wrist
    - Thumb base
  - Tendon transfers
    - Shoulder
      - Trapezius to deltoid
    - Elbow
      - Steindler flexorplasty is designed to mobilize the flexor/pronator mass from the medial epicondyle to a more proximal point on the humerus (tenodesis effect)
  - Free functioning muscle transfer
    - Gracilis to restore elbow flexion

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Chapter 25

Paediatric oral core topics

Kathryn Price and Antoine de Gheldere

Introduction

This section is an overview of important paediatric topics that tend to appear regularly in the examination. It should point you towards the more important areas of the paediatric syllabus. Topics are included for two primary reasons:

1. They have been repeatedly asked in previous exams
2. They are often the subject of confusion, and textbooks are sometimes the source of it!

The paediatric oral can be awkward as the examiners expect you to have both a comprehensive range and depth of paediatric knowledge. Candidates may struggle unless they have had a reasonable working exposure to paediatrics, ideally 6 months as part of a higher surgical training rotation. Some topics are very predictable – In the oral candidates will invariably get one of the 'big three' (developmental dysplasia of the hip (DDH), Perthes' and slipped upper femoral epiphysis (SUFE)).

The paediatric oral for the most part consists of clinical photographs and radiographs acting as prompts to lead you into a particular topic. With the introduction of standardized questions for the oral examinations, it is unlikely that the dreaded video of gait analysis in a cerebral palsy (CP) patient will be shown.

The paediatric oral component of the exam now involves three questions, each lasting 5 minutes. A typical example might involve:

1 A paediatric trauma question – Perhaps involving growth plate injury
2 One of the 'big three' (Perthes, SUFE or DDH)
3 A further topic such as congenital talipes equinovarus (CTEV), Brodie's abscess, CP or in-toeing

The key elements for success in the oral are:

- To have worked for 6 months in a paediatric orthopaedics higher surgical training job. Knowledge is important but if you have not been near a paediatric orthopaedic clinic it will be obvious to your examiner
- Correctly gauging the depth of paediatrics knowledge required for the oral. Aiming too high can be a disaster and can swallow precious time needed for other areas; too low and you will certainly fail. Remember the exam is to test you at the level of a Day 1 Consultant in the *generality* of orthopaedic and trauma surgery. Knowing absolutely everything about CTEV is commendable but of no use if you know nothing about DDH
- Similar principles apply with your reading material. Some paediatric books are a little flimsy, while others are highly subspecialized textbooks that are difficult to use for revision
- Go on a well-recommended and established paediatric course. There has been some feedback suggesting that several courses may be a little too detailed for the exam but it is unlikely that you will emerge with too much knowledge from such a course! Towards the end of your preparation for the exam, ask your local paediatric FRCS (Tr & Orth) examiner to viva you at least once, but preferably on a couple of occasions
- Try to set up a study group of trainees about to sit the examination and regularly meet up to go through different areas/topics of revision

Principles of paediatric orthopaedic history and examination

Whilst each region of the body demands specific attention there are some important features of examination in the paediatric population that set it apart from adult practice:

- Many children are too young to volunteer an accurate history – There is an increased reliance on examination and investigation in this situation
- Children are growing and developing. There is a moving baseline which can make for diagnostic difficulty but it also makes for a forgiving skeleton when it comes to healing
- Injury must always be interpreted in the context of developmental stage – And be alert to non-accidental injury (3 month olds do not 'fall off the bed')
- Where knee pain *think* hip and where hip pain *think* knee
- Where night pain *think* neoplasm until proven otherwise
- History
  - Should include a note of family history which may raise the likelihood of an inherited condition
  - Must include attention to the pregnancy and birth history. Prematurity, birth trauma, etc. can have ongoing implications

- Should evaluate motor development and take note of milestones reached – Always ask age at which the child walked
- Should carefully consider related symptoms from other systems

Milestones: Mean age (95th centile)

Head control – 3 months (6 months)
Sitting – 6 months (9 months)
Stand – 8 months (12 months)
Walk – 12 months (18 months) – Remember 1 year is a mean 'normal' walking age so 50% of children will walk later, but 18 months to 2 years is generally taken as the upper limit of normal

- Examination (whilst tailored to a given problem) for lower limbs in general:
  - Should include an assessment of gait by observation
  - Should include inspection of the spine, hips, knees and feet
  - Should include a clinical check of lower limb torsional alignment and length
- Follow-up of children with significant musculoskeletal pathology is often carried through to skeletal maturity

Examination corner

Normal variants

The candidate is shown a picture of a 3-year-old girl standing. The feet and patellae are both in turned.

EXAMINER: This 3-year-old girl has been referred to you by her GP due to parental concerns regarding her in-toeing. How would you assess her?

CANDIDATE: I would take a history from the family to establish how long they have been concerned, if there was any history of problems such as CTEV. I would also like to establish a developmental history, and family history of limb problems and the trend as to whether things are improving, staying the same or getting worse. After this I would examine the child.

EXAMINER: So, the family state that she is normally fit and well, no other concerns from birth. She is reaching milestones normally. They have noticed her feet turning in since she started to walk and are unsure if it is still the same. They feel that she is very clumsy and trips over her feet on a regular basis. What examination would you like to do?

CANDIDATE: I would like to see her walk, run, walk on her heels and on tiptoes. Then I would examine her on the couch for any leg length discrepancy, range of movements of all the joints, particularly hips and check for any tight tendons. Most importantly I would turn her prone to do a rotational profile. Lastly, I would check the spine to ensure there was no pathology there.

EXAMINER: What are you looking for on a rotational profile and how would you do this?

CANDIDATE: With the child prone you can assess for femoral rotation, tibial torsion and foot deformities. The femoral rotation is assessed by allowing the legs to rotate outwards and then inwards. Movement away from the midline represents internal rotation of the hip, movement towards the midline represents external rotation. You can also perform Gage's test, feeling for the point where the greater trochanter is the most prominent during movements to assess the version of the hip.

After this, the thigh-foot angle gives you an idea of tibial torsion, be it internal or external. The foot itself can then be examined to see if there is any deformity there or if the border is straight.

This assessment lets you assess how many limb segments are involved in the rotational problem and to what extent.

EXAMINER: OK, when you do this assessment you find that there is 35° of femoral anteversion, with no other obvious problems identified?

CANDIDATE: Well, I would reassure the parents that this is part of the normal spectrum of what we see. Persistent femoral anteversion is extremely common and will remodel without intervention in >90% of cases by the age of 9 years. I would explain that the only way to correct this would be with bilateral derotational femoral osteotomies, which would be a potentially life-threatening procedure in a child this age. The vast majority never need any intervention, however, if it is still a clinical problem by the age of 9 then we will gladly review back in clinic to discuss intervention. I would reassure them that we will not have missed the opportunity to intervene, and that the surgery is possible even in to adult life if deemed necessary.

EXAMINER: They ask if there is anything else that they can do to try to avoid this?

CANDIDATE: I would explain that it is the normal muscle pull that causes the bone to remodel. I would not limit them in any way or do anything differently. The only thing I would advise would be to avoid them sitting with their legs in the W position, and advise crossed legs if possible.

EXAMINER: OK, they are happy to accept that.

The candidate is shown a clinical photograph of a 2-year-old boy with planovalgus feet.

EXAMINER: This young man has been referred to your clinic following parental concerns regarding his foot position. How would you assess him?

CANDIDATE: I would take a history first regarding pregnancy, birth, development and family history. I would enquire as to how long the feet have been noted to hold this position. I would also want to know if he complains of any discomfort.

EXAMINER: He is otherwise fit and well with no concerns. The parents say that his feet have always rolled over since he has been walking.

CANDIDATE: OK, I would examine the child. I would want to see him walk and run. I would try to get him to stand on tiptoes. If this is not possible then I would assess Jack's test and feel for hindfoot movement passively to establish if it flexible or fixed. After this I would assess him on the couch to check all of the lower limbs for joint movements, tendon contractures (particularly gastrocnemius) and assess rotational profile and the spine.

EXAMINER: From what you can see it seems that he has flexible planovalgus feet with no other concerns. What would you advise the family?

CANDIDATE: I would explain that flexible flat feet are extremely common in children this age. It is due to ligamentous laxity allowing the foot to sag down on weight-bearing. However, when the soft tissues are tensioned the arch restores normally. Unless this is symptomatic then it requires no treatment. The vast majority will have an arch reconstitute by the age of 7, for those that don't it is rarely a problem. It only becomes an issue if the gastrocnemius complex is tight.

EXAMINER: If he was a bit older and was complaining that his feet hurt when he walked what would you advise?

CANDIDATE: If there were any tightness of the gastrocnemius complex then I would send them for stretches. They can also try medial arch supports, however, I would warn them that some children find these helpful and some find them more painful. I would only see them back again if there were further concerns.

EXAMINER: If the planovalgus foot was fixed and not flexible, what would you be thinking?

CANDIDATE: The main cause for a rigid flat foot in a child would be tarsal coalition. In small children it can be extremely difficult to see on x-rays due to the high proportion of cartilage in the tarsus. In an older child I would try to see it with x-rays, CT or MRI depending on results and symptoms.

EXAMINER: OK, let's move on.

The candidate is shown a clinical photograph of an 18-month-old child with bilateral symmetrical genu varum.

EXAMINER: This child has been referred to your clinic due to parental concerns over bowlegs. They feel that things have got progressively worse since he started walking and want him assessed.

CANDIDATE: OK, I would start with taking a history relating to pregnancy, birth, development, and family history. After this I would find out what concerns the parents have got, when they first noticed the bowlegs and whether they think things are progressing, improving or staying the same. After this I would examine the child.

EXAMINER: He is a normal, fit and healthy little boy. They have always noticed him being bow legged but definitely think that things are getting worse at this stage. What examination would you do?

CANDIDATE: I would assess him standing for any leg length discrepancy, his posture and the leg position. I would watch him walk and then assess him on the couch to look for the range of movement of all joints; any increased tone, any contractures and then assess rotational profile and his spine.

EXAMINER: OK, you find that this is just an isolated genu varum with no concerns about anything else. What would you do from here?

CANDIDATE: I would explain to the parents that all children at this age have bowlegs. The Salenius curve showed that we would expect them to be varus until the age of 2, then become overly valgus by 3 and return to a normal adult alignment by the age of 7. Having said this, only time will tell if he will follow the normal pattern. At this point I would carefully document the degree of varus and do no further test unless there were concerns about vitamin D deficiency or trauma. I would reassure the parents that the likelihood is that the leg alignment would correct itself over the next few years without intervention. I would suggest that they take photographs of his legs today and then keep an eye on things over the next year. I would review back once more in a year's time to ensure that things are improving. If there are concerns at that stage then I would do blood tests and a mechanical axis x-ray.

EXAMINER: What would be in your differential diagnosis?

CANDIDATE: Physiological varus, trauma, infections, blounts disease, tumours, metabolic bone disease.

EXAMINER: OK, let's move on.

The candidate is shown a clinical photograph of a 3-year-old girl walking on tiptoes.

EXAMINER: This young lady has been referred to you with persistent tiptoe walking. How would you assess her?

CANDIDATE: I would take a history regarding pregnancy, birth, any perinatal concerns, developmental milestones and general health. I would then ask specifically about the tiptoe walking. When it began, if it all the time, if it is worse when she runs, etc, then I would examine her.

EXAMINER: Parents tell you that everything has been normal with no concerns. She has always seemed to walk on tiptoes and they don't really notice a difference between walking or running. What would you look for on examination?

CANDIDATE: I would want to see her walk and run. I would want to see her walk on her toes and try to walk on her heels. I would do an assessment of her tone on the couch looking for clonus and brisk reflexes. After this I would do all of the normal checks for joint movements. I would test for contractures of the adductors, hamstrings, rectus femoris and the gastrocnemius complex. I would also want to check her spine.

EXAMINER: What are you trying to exclude?

CANDIDATE: Any underlying problem really, but primarily cerebral palsy. Mild cerebral palsy can present in early childhood with persistent tiptoe walking due to increased tone. They would typically have rate-related symptoms and have signs of an upper motor neurone lesion on examination.

EXAMINER: Assuming all of those things are normal and this is just idiopathic toe walking what would you advise?

CANDIDATE: I would explain to the family that this is quite common in young children. In the first instance I would send them to physiotherapy to learn some stretching exercises and advise them to encourage her to walk flat footed as much as possible. The majority of cases will resolve by school age.

EXAMINER: What would you advise if she were 6?

CANDIDATE: For older children I would still suggest stretches in the first instance. If this is not successful then we can try serial casting to correct the problem with or without splintage afterwards. Tendon lengthening is the last resort after all else has failed and there is a clear functional problem.

EXAMINER: And what kind of tendon lengthening would you do?

CANDIDATE: Usually a HOKE percutaneous lengthening in these cases.

EXAMINER: OK, let's move on.

Paediatric trauma

It may be tempting to skip over this area and hope your adult trauma knowledge will be adequate, but there are important aspects to paediatric trauma that are not appropriately covered by adult trauma knowledge. A comprehensive treatment of this topic is not appropriate to this text; however, there are some important features of paediatric trauma that will be highlighted. Important principles underlie paediatric fracture treatment:

- Periosteum is thicker, more vascular and more active than in adults – Healing is fast and the periosteal hinge is often an effective aid to maintaining reduction (and can block reduction if not understood!)
- Children's bones remodel well in the plane of joint motion
- Fractures involving physes can result in progressive deformity – We must 'respect the physis'

Increased vascularity and porosity of paediatric bones (cf. adult) give rise to increased plasticity manifested as incomplete fractures:

- Failure in compression – Buckle/torus fracture
- Failure in tension – Greenstick fracture

Healing bones can overgrow (increased blood supply and stimulation to physes of injured limb), so overlapping of (femoral) fractures of 1.5 cm is often accepted.

You must understand and be able to describe the physeal injury classification of Salter and Harris[1] (Figure 25.1).

To Salter's original classification, two further classes have been added – Injury to the perichondrial ring of LaCroix and missing physis (where part of the physis has been lost, usually in a road-drag injury).

This has prognostic value, with progressively more chance of growth arrest as you move up through the classification.

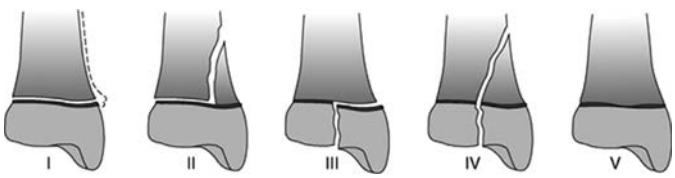

Figure 25.1 Salter–Harris fracture types

The elbow

This is a testing area clinically and requires your attention. Inability to discuss a supracondylar fracture and its management would certainly be a pass/fail issue in the eyes of an examiner.

In part the topic is complicated by the many ossification centres:

- Capitellum (6 months)
- Radial head and medial epicondyle (5 years)
- Trochlea (7 years)
- Lateral epicondyle (9 years)
- Olecranon (11 years)

This gives rise to confusion regarding the normal appearances – You should critically assess the following when viewing plain radiographs of a paediatric elbow:

- Carrying angle 7°
- Baumann angle. A line perpendicular to the axis of the humerus, and a line that goes through the physis of the capitellum. Normal value is 70–75°
- The teardrop or hour glass is formed by the anterior margin of the olecranon fossa and the posterior margin of the coranoid fossa with the capitellum forming the inferior portion. The capitellum and trochlea should be superimposed. These lines indicate that a correctly positioned lateral radiograph has been obtained
- Medial epicondylar epiphyseal line angle. This is formed by the intersection of the long axis of the humeral shaft and the line formed by the medial epicondylar growth plate. Normally 25–45°
- Humerotrochlear angle. This angle is formed by the shaft of the humerus and the axis of the condyles on the lateral x-ray 40°
- Lateral capitellar angle is measured by the intersection of a line parallel to the midpoint of the distal humeral shaft and one drawn through the midpoint of the capitellum. Normally 30°
- Anterior humeral line is drawn along the anterior margin of the humerus and passes through centre of capitellum
- Anterior coronoid line is drawn on a lateral radiograph along the coronoid and continued proximally. It should just touch the capitellum anteriorly in a normal elbow. If the capitellum is angled or displaced anteriorly, this line intersects or lies posterior to the capitellum

Supracondylar fracture

The supracondylar fracture is the most frequent injury to this area:

- 95% are hyperextension injury

- Olecranon in fossa as fulcrum
- Assessment – Clinical (deformity, distal neurovascular deficit), 15% neurological injury (usually neurapraxia of anterior interosseous nerve (AIN) – Look for IPJ index and thumb flexion), radiographs (orthogonal views, note posterior fat pad)
- Gartland classification
  - Type I: Minimally displaced = simple immobilization/splintage
  - Type II: Hinged = stabilised on posterior hinge at 120° flexion *or* fix as for type III
  - Type III: Off-ended = closed/open K-wiring[a]
- Formally opening the joint is reserved for cases that do not reduce closed. An anterior approach is considered when vascular compromise is suspected
- Vascular compromise should lead to swift surgical reduction. Thereafter:
  - Pink and pulse = splint
  - Pink, no pulse = splint and monitor (collateral circulation is usually sufficient)
  - White, cool, no pulse = explore
- Active motion should be commenced at 3 weeks (stiffness is the enemy thereafter)
- Cubitus varus is the commonest iatrogenic complication with a poorly reduced/stabilised distal fragment pulled into varus and rotated. This is best avoided but can be addressed by a late valgus osteotomy of the distal humerus
- Remember tardy ulnar nerve palsy with cubitus valgus

T-condylar fracture

The t-condylar fracture is less common

- Axial impaction results in trochlear–capitellar separation
- Aim for anatomical joint reduction
- Fix condylar fragments first (closed or via posterior approach)
- Fix condylar fragment to shaft with K-wires or plate

Physeal separation

Physeal separation can occur and poses diagnostic difficulties owing to the cartilaginous nature of the young elbow

- Rotatory force – NB. Consider non-accidental injury
- Salter–Harris I or II in infants and toddlers
- Confused with 'dislocation'
- Consider arthrogram
- MUA and K-wire (only attempt up to 1 week)

[a] The issue of how to K-wire a supracondylar fracture has generated disproportionate attention. What matters is achieving a stable hold. This can either be with divergent laterally based K-wires or crossed K-wires. There is nothing in the literature to favour one technique significantly over the other. For crossed wires many practitioners choose to visualize and thereby protect the ulnar nerve from the medial K-wire through a small incision.

Lateral condylar fracture

- Varus force on supinated forearm
- Salter–Harris type IV
- Milch classification – Poor reliability
- Jakob classification
  - Stage 1 – Intact cartilage hinge
  - Stage 2 – Complete fracture, minimal displacement
  - Stage 3 – Rotational displacement
- Pin stage 2 and 3 via lateral approach

Complications include

- Late presentation (>6/52)
  - Anatomical correction difficult due to remodelling
  - Consider subtotal coronoid osteotomy or intra-articular osteotomy
- Non-union
  - May present with ulnar neuritis, cubitus valgus
  - Fix in best functional position ± bone graft

Medial condylar fracture

- Rare
- Open reduction with internal fixation (ORIF) if >2 mm displacement

Epicondylar fractures

Lateral

- Immobilize then early active range of movement (ROM)
- Where displaced >5 mm, consider excision

Medial

- Valgus load to extended elbow
- 50% occur with posterolateral elbow dislocation
- Note that there is a risk of ulnar nerve entrapment with closed reduction
- Closed vs open reduction debatable – ~ORIF >5 mm displacement

Radial neck fractures

- 50% isolated, 50% with proximal ulnar fracture
- Usually Salter–Harris I or II
- Valgus stress injury
- Functional limitation greater in:
  - >10 years
  - >30°
  - >3 mm
- Closed reduction/flexible intramedullary nail/ORIF
- Removal of K-wire and ROM at 3/52

Olecranon fractures

- Uncommon
- Minimal displacement – plaster of Paris (POP) 3/52

- >5 mm displacement – Closed reduction . . . ORIF = tension-band wiring (TBW) or screw(s)

Pulled elbow

- A toddler's injury with classic presentation
- Axial traction by a pull on the hand or wrist. The forearm is usually held in incomplete extension, and partially pronated.
- X-ray is not necessary but if obtained exclude fractures (including plastic deformation of ulna).
- Treatment with closed reduction using either forearm hyper-pronation or supination-flexion methods

The ankle

- 5% of all children's fractures
- 17% of physeal injuries
- Note 6 mm growth per annum at distal tibia and fibula; hence, potential for angular and limb length growth disturbance

Poland (1898)[2] highlighted three characteristics of paediatric ankle fractures:

1. Physis as plane of fracture
2. Physis weaker than ligaments
3. Growth arrest risk

Classification of fractures can be:

- **Mechanistic**
  - Lauge–Hansen, Dias–Tachdjian[3]
    - Helpful with reduction
    - Poor interobserver reliability
- **Anatomical**
  - Salter–Harris
    - Good reproducibility
    - Prognostic value
  - Vahvanen and Aalto[4]

There are three pitfalls to be avoided:

1. False-negatives (missed fractures) – Consider supplementary mortise and oblique views
2. False-positives (normal variants) – There can be many secondary ossific nuclei at the foot and ankle, e.g. os subtibiale beneath the medial malleolus
3. Persistent displacement – Consider periosteal interposition in the fracture site

Transitional fractures at the ankle occur whilst the growth plate is closing, at the age of 13–14 years. In order, this progresses from centre medially and, finally, laterally. In these injuries the 'golden rule' of respecting the physis becomes secondary to the need to achieve articular congruity since 50% are said to go on to degenerative change if there is >2 mm articular step. Growth arrest is said to complicate these injuries in up to 40% of cases – Complete bilateral epiphyseodesis may be desirable to avoid angular or length deformity.

- Tillaux
  - External rotation mechanism
  - Anterior tibiofibular ligament avulsion
  - Salter–Harris III
  - Managed with closed or open reduction
- Triplane
  - External rotation mechanism
  - Salter–Harris III on AP + Salter–Harris II on lateral = Salter–Harris IV
  - 2–3– Or 4 part
  - CT is desirable to plan surgical approach
  - Require ORIF

Polytrauma

Paediatric major trauma requires attention to advanced paediatric life support principles. These are parallel to the more familiar territory of ATLS® but there are crucial differences. Not every orthopaedic trainee undertakes ATLS® training, but it would be a good idea to familiarize yourself with the course book (although you might wish to avoid spending time on the detailed analysis of arrhythmias!).

- Injury patterns are different in children. Remember the bumper of a car strikes the tibia of an adult but the head/torso of a toddler. Energy imparted per unit area will be greater the smaller the child
- Physiological reserve is greater in a child, which is good news, but the benefit is tempered by its capacity to obscure underlying impending cardiovascular collapse (a child can lose 30% of circulating volume and remain normotensive). Therefore, when a child arrests it is usually very bad news indeed (that is not to say it is great news in anyone)

It is important not to be phased by the fact that resuscitation involves a child – Airway (with cervical spine control), breathing and circulation remain the top priorities in paediatric resuscitation:

- The airway in a child is narrower and shorter and more prone to blockage
- Breathing should be maintained by jaw thrust – Chin lift is not appropriate in trauma and in infants (where it exacerbates obstruction)
- Circulation is primarily assessed by colour, capillary refill, skin temperature, pulse and blood pressure
- Disability assessment should actively look for the greater incidence of head injury in paediatric trauma cases

Cervical spine

In a compliant child the principles of assessment are similar to those in an adult – The NEXUS study showed 100% sensitivity,

20% specificity and 100% negative predictive value of 5 clinical features in determining the need for radiographs:

1 Midline tenderness
2 Altered alertness
3 Intoxication
4 Neurological compromise
5 Distracting injury and tenderness

Where indicated, assessment of radiographs needs to be systematic – Alignment, Bones, Cartilages, Soft tissues (ABCS). There are some specific issues to be aware of in children's C-spine radiographs:

- The upper C-spine is most vulnerable (owing to the relatively large cranial mass and, therefore, a high fulcrum)
- Pseudosubluxation – Of C2 on C3, and C3 on C4, up to 16 years of age is caused by relative ligament laxity and flat facet joints permitting AP movement; this is a normal variant
- The anterior atlanto-dens interval (AADI) up to 5 mm – AADI is greater than in adults (up to 3 mm)

Figure 25.2 Normally (and in pseudosubluxation) a line through the anterior aspect of the spinal process of C1 and C3 should also pass through the anterior aspect of the spinal process of C2

- Spinal cord injury without radiological abnormality (SCIWORA) is more common in those under 8 years
- Lordosis is less apparent in young children
- Growth plates can mimic fractures – Look for smooth edges and corners suggestive of the former
- C1 posterior arch ossifies at 3 years
- Dens ossifies at 3–8 years
- C1 anterior arch ossifies at 6–9 years
- Prevertebral (retropharyngeal) soft tissues at C3 should be <2/3 AP diameter of vertebral body. From C4 down (glottis and below) the prevertebral tissues are doubled
- Widened (distraction) or narrowed (extrusion) disc injuries should be sought

CT is reserved for inadequate plain imaging and where plain imaging raises concern (Figure 25.2).

Examination corner

Paediatric trauma

The candidate is shown an x-ray of an off-ended Gartland III supracondylar fracture of the elbow in a 4-year-old child.

EXAMINER: A 4-year-old child is taken to the ED by his mother following a fall from a climbing frame after school. This is the x-ray which was taken, have a look and tell me what you see.

CANDIDATE: This is an AP and a lateral x-ray of the right elbow of a 4-year-old boy. There is a widely displaced, off-ended supracondylar fracture which would be a Gartland type III. The proximal bone spike is extremely prominent and close to the skin suggesting it has button-holed through the soft tissues. I would be concerned about the possibility of an open injury and the viability of the skin overlying that spike. I would like to know if it is closed and neurovascularly in tact.

EXAMINER: There is no cut in the skin overlying the spike of bone but the skin is tethered and looks white over that point. With regards to the neurovascular status, there is no palpable radial pulse and the capillary refill is prolonged to 5 seconds. He refuses to voluntarily move his fingers and states that he has tingling in his whole hand.

CANDIDATE: OK. We know that there is very significant displacement and the vascularity and nerves are being compromised by the pressure. We also know that the skin is compromised by that spike of bone. This is a surgical emergency and so we will need to get him to theatre ASAP. I would inform my consultant, the theatre team and the anaesthetist about the child's condition and prep him for theatre.

EXAMINER: What would you do to prepare for theatre?

CANDIDATE: I would splint the arm for comfort. I would mark and consent for closed or open reduction with K-wire fixation of the fracture. I would also consent them for exploration ± vascular exploration and repair and fasciotomies to leave all options open. I would tell them to remain nil by mouth (NBM); however, under these circumstances we would not wait for adequate starvation

times anyway. I would inform the vascular/plastics team about the possibility of requiring vascular repair if it fails to re-perfuse when we straighten the arm and get them up to theatre.

EXAMINER: Assuming that you are the consultant and you now have the child asleep in theatre, what will you do?

CANDIDATE: I would attempt to reduce the fracture closed in the first instance and stabilise it with crossed K-wires. After this I would reassess the arm for perfusion.

EXAMINER: What would you do if you could not reduce it closed?

CANDIDATE: In this circumstance, I would perform an open reduction via an anterior approach to the elbow. This would allow me to visualize and reduce the fracture and allow free access to the vessels to inspect them and repair if necessary.

EXAMINER: OK, let's assume that you have managed to reduce it closed. After reduction the hand becomes pink and well-perfused and the pulse returns. What would you do?

CANDIDATE: I would place them in an above elbow backslab with the elbow slightly extended to reduce any pressure on the vessels and observe them extremely closely for 24–48 hours for any signs of compromise.

EXAMINER: OK, what about if the arm remains pale with poor capillary refill?

CANDIDATE: The first thing to do is assess the reduction. If the fracture is perfectly reduced then the vessels will not be trapped in the fracture and we should assume that the vessel has been injured. We would then call our colleagues in either vascular or plastic surgery depending on local protocol to explore and repair the vessel.

If the fracture is not properly reduced then I would attempt a further reduction and stabilisation. Sometimes it has not fully reduced because some soft tissue is stuck in the fracture kinking the vessel. If after this repeat manoeuvre the vascularity improves I would treat it like the first patient. If this made no difference then I would continue on with exploration of the vessel.

EXAMINER: What would you do if the perfusion improved but the pulse did not return?

CANDIDATE: Again this depends slightly on the reduction. If the fracture is fully reduced then it is extremely unlikely that the vessels are being kinked or trapped in the fracture. As long as perfusion is good in the hand then I would observe and wait in the same manner we did with the first child. It is well-established that the brachial artery goes in to spasm after these kinds of injuries.

CANDIDATE: If the fracture was not fully reduced then I would repeat the reduction again to ensure that this was not a problem. Again, if the pulse was definitely absent preoperatively and the perfusion was good with a warm hand at the end of the procedure I would observe. If there was any concern over the vascularity of the hand then I would explore the vessel.

EXAMINER: Is there anything else that you base your decision on regarding exploration of the vessel?

CANDIDATE: I know that it was published in JBJS that the presence of a preoperative anterior interosseous nerve (AIN) palsy made it more likely that the brachial artery was damaged. There has been plenty of literature stating that you do not need to explore the artery unless the perfusion is compromised and the editorial following that paper suggested that the reader should explore with caution! Certainly in out unit, the policy is that as long as the hand is well-perfused and the fracture is reduced that you do not need to explore. I think that it can be extremely difficult to get small children to show good AIN function without an injury, let alone under these circumstances! Also, the majority of brachial artery injuries are intimal tears which will tend to clot off even after repair. So, my only indication for exploration would be inadequate perfusion after the surgery.

EXAMINER: OK, let's move on.

The candidate is shown an x-ray of a supracondylar fracture which is off-ended (Gartland III)

EXAMINER: This is the x-ray of a 3-year-old girl brought to the ED by her father after falling off a friend's trampoline. Tell me what you see.

CANDIDATE: This is an AP and a lateral x-ray of the left elbow of a small child showing a completely displaced supracondylar fracture (Gartland III).

EXAMINER: Just run through the Gartland classification for me.

CANDIDATE: The Gartland classification for supracondylar fractures of the elbow has types I–III. Type I is completely undisplaced, type II has pure extension with the posterior hinge remaining in tact, and a type III is where the fracture is completely displaced or has rotation/varus/valgus.

EXAMINER: OK, so how would you manage this young girl?

CANDIDATE: I would check that the injury was closed and neurovascularly intact.

EXAMINER: It is.

CANDIDATE: Good, then I would splint it in an above elbow back slab and mark and consent her for theatre for an manipulation under anaesthetic (MUA) and K-wiring. I would speak to my consultant and the anaesthetist/theatre team to try to get the surgery done as soon as possible.

EXAMINER: Would you take her to theatre in the middle of the night?

CANDIDATE: Assuming it was closed and neurovascularly intact, then I would not take her at 3 in the morning! I would push to get the case done late in the evening to reduce the risk of her developing neurovascular issues and reducing the chances of needing an open reduction.

EXAMINER: If you leave these injuries overnight does it increase the chances of needing to perform an open reduction?

CANDIDATE: There has been work from the USA stating that it does not increase the risk of needing an open reduction. What you need to bear in mind when you are quoting that paper is that they manipulated all of the fractures under ketamine in the ED before admission! That means that there is not an

increased risk of open reduction if you reduce the fracture and then leave them over night. Not relevant if you are leaving them off-ended, which would be the standard practice in the UK.

EXAMINER: OK, so now you are the consultant in theatre and the child is asleep and ready to go. You manipulate it back in place without too much difficulty. How are you going to fix it?

CANDIDATE: Well, the two options are to do two lateral wires or crossed K-wires. The benefit of doing two lateral K-wires is that you avoid placing a wire next to the ulnar nerve as it rounds the medial epicondyle. If you use 2 mm K-wires it has been shown to be as stable as crossed 1.6 mm wires. The problem is that it is much more technically difficult to do. In a little elbow it can be tricky to get the wires across, you need to get good spread of the wires at the fracture site and good bicortical hold. Also, some fracture types are not suitable for this technique. It should also be noted that you can still damage the ulnar nerve as the K-wire protrudes through the opposite cortex.

The other option is to use 1.6 mm crossed K-wires. This is technically less demanding but gives a greater risk of damaging the ulnar nerve. I would use this technique as it is the one that I am most familiar with. To minimize the risk of damage to the ulnar nerve I would fix the lateral side first, and then gently extend the elbow to reduce the tension on the nerve. I would perform a mini-open approach on the medial side so that I could be clear that the ulnar nerve was free and then fix the medial side.

EXAMINER: OK, let's move on.

The candidate is shown an x-ray of a displaced hip fracture in a 9-year-old child which has fractured through the physis leaving the epiphysis in the acetabulum.

EXAMINER: This is the x-ray of a 9-year-old boy brought in by ambulance having been hit by a car as he walked across the road. It was estimated by witnesses that the car was travelling approximately 25 MPH. What can you see on the x-ray?

CANDIDATE: This is an AP pelvic x-ray of a 9-year-old boy. The most obvious abnormality on it is a fracture to the right hip. There appears to be a fracture running through the physis with the head in place in the acetabulum and the metaphysis displaced posteriorly. I cannot identify any additional fractures in the pelvis but considering the mechanism I would be concerned regarding any missed fractures.

EXAMINER: What would your management be for this child?

CANDIDATE: Well, in the first instance he would need to have a trauma call and be dealt with as per ATLS® protocols. This was a high-energy mechanism and so we would have to exclude additional injuries. As much as he has a hip fracture, the leading cause of death in children is head injury followed by occult visceral injury.

EXAMINER: OK, let's assume that there has been a full ATLS® assessment and this is an isolated injury. Do you know any classification systems for this injury?

CANDIDATE: I know of the Delbet classification for hip fractures in children, types I–IV. Type I is through the physis, type II is transcervical, type III is basicervical and type IV is intertrochanteric. Type I is further subclassified as to whether the epiphysis stays with the metaphysis or whether there is significant displacement between the two.

EXAMINER: That's right, so what would you tell the family regarding the risk of avascular necrosis (AVN) based on that classification system?

CANDIDATE: I would inform them that with this injury pattern the risk of AVN is between 90% and 100%.

EXAMINER: OK, so how would you manage this child?

CANDIDATE: I would inform my consultant and theatres/anaesthetist about the child. I would make sure that he had a group and save and any other relevant investigations based on his past medical history. I would mark and consent him for theatre for ORIF of the right hip fracture and get him to theatre ASAP.

EXAMINER: What would you do surgically assuming you were adequately trained?

CANDIDATE: I would attempt a closed reduction of the fracture in the first instance to see if I could reduce it. If I was lucky enough to reduce it anatomically closed then I would fix it in situ using cannulated screws. If I could not reduce it closed then I would perform an open reduction through an anterolateral approach. I would visually reduce the fracture and fix it with cannulated screws. In both instances I would evacuate the haematoma, by aspiration if I managed a closed reduction.

EXAMINER: What would you say regarding risks when consenting them for the surgery?

CANDIDATE: Bleeding, infection, neurovascular damage, avascular necrosis, mal-union, non-union, leg length discrepancy, progression to osteoarthritis, need for future procedures.

EXAMINER: OK, let's move on.

The candidate is shown an ankle x-ray of a 14-year-old boy with a minimally displaced Salter–Harris II fracture seen on the lateral projection.

EXAMINER: This young man is 14 years old and attended ED following a footballing injury to this ankle. What do you see on the x-ray?

CANDIDATE: This is an AP and lateral x-ray of the right ankle. The most obvious abnormality is a minimally displaced Salter–Harris type II fracture of the distal tibia seen on the lateral projection. I cannot see any other fracture lines on this x-ray but I would be concerned about the possibility of a triplane injury in this age group.

EXAMINER: If you wanted to determine if this was a triplane injury then what further tests would you do?

CANDIDATE: I would do a CT scan of the ankle to fully define the fracture pattern.

EXAMINER: OK, so you get a CT scan and it confirms your suspicions that this is a triplane injury with an intra-articular extension with a 2 mm gap. What would you do now?

CANDIDATE: Well, this is a displaced intra-articular fracture which needs reduction and fixation. I would plan to take him to theatre

to fix this – Probably with two screws, one in the epiphysis and one in the metaphysis. Commonly you can reduce the joint surface by just placing a large reduction clamp across it, but if there was any concern then I would open it to ensure reduction.

EXAMINER: Would you be concerned about damage to the physis during the surgery?

CANDIDATE: No. Commonly you can fix this with screws running parallel to the physis without damaging it. If there was some reason that this would not be possible then accurate reduction and stable fixation of the fracture is definitely the priority. By definition of the injury being a triplane, the physis is closing anyway and is not a priority.

EXAMINER: OK, let's move on.

Examination corner

Non-accidental injury

The candidate is shown an x-ray of a 9-month-old boy with a spiral femoral shaft fracture.

EXAMINER: This x-ray was taken after a 9-month-old boy was brought to the ED by his mother following an unwitnessed fall. Tell me what you think it shows.

CANDIDATE: Well this is an AP and lateral x-ray of the right femur in a 9-month-old boy. The most obvious abnormality is a displaced spiral fracture of the diaphysis. I would be very interested to know the ambulatory status of this child and the history so that I could assess the risk of non-accidental injury (NAI).

EXAMINER: Well, the child is not walking yet, he is freely crawling and starting to reach for furniture but not coasting. Mum says that she was out of the room when she heard a crash, then she found him crying on the floor unable to weight bear.

CANDIDATE: Did she say that his leg was trapped in anything?

EXAMINER: No, she just found him in the middle of the floor.

CANDIDATE: OK, well on that history I would be extremely concerned for this child's safety due to the risk of NAI. I would admit him for gallow's traction to treat his femoral fracture and inform the consultant on-call, my paediatric colleagues and the safeguarding team.

EXAMINER: What would you say to the mother?

CANDIDATE: I would tell Mum that this is a very unusual injury to have occurred in a child of this age who is not ambulatory. If it did occur from him just stumbling in the middle of the floor, then we would be concerned about his bone quality as this would normally not result in a femoral fracture. We would need to admit him to do some further investigations to see if we can find the cause of this injury.

EXAMINER: What further tests would you arrange?

CANDIDATE: Well in the first instance we would need to do a formal assessment of the chance of this being NAI. In this age group a femoral shaft fracture is extremely unusual but can occur by chance. We would need to check whether or not there have been any previous concerns regarding the child or any siblings.

The child will need to be examined by a consultant to look for any further evidence of harm. The most common feature seen is skin lesions. We would also need to feel all of the limbs to look for signs of healing fractures and look in the eyes for any retinal haemorrhages.

If after this assessment, the team feels that NAI cannot be excluded then we would have to perform a skeletal survey to look for associated injuries ± CT head depending on hospital protocol. We would also need to perform blood tests looking for any underlying cause for the injuries such as osteogenesis imperfecta or vitamin D deficiency.

EXAMINER: What would you be looking for on a skeletal survey?

CANDIDATE: Essentially anything abnormal! The skeletal survey can give you some indication of bone quality and any deformity associated with conditions like rickets suggesting an underlying metabolic bone problem as opposed to NAI. If the child is being abused then we may see multiple injuries, healing fractures of different ages. Rib fractures, humeral fractures and metaphyseal corner fractures are very specific to NAI, but they are only seen in the minority of cases of abuse. Unfortunately, the vast majority of NAI-related injuries are exactly the same as accidental injuries sustained by children every day.

EXAMINER: What factors would make you think an injury was not accidental in these cases?

CANDIDATE: You will always be more suspicious in cases where abuse has been questioned before. With regards to the history:

- Delayed presentation after an obvious injury
- History of injury does not match the injury pattern
- Changing story from the parent, or differing accounts from interested parties
- Recurrent unexplained injuries
- Ambulatory status – Those who are not walking rarely sustain diaphyseal spiral fractures whereas it is extremely common in toddlers
- Unusual family dynamics – Sometimes you can see very odd dynamics between parents and children suggesting there is more going on than meets the eye

EXAMINER: Okay, so let's assume that it was NAI and that all of the appropriate action has been taken. How would you treat this child with regards to the femoral fracture?

CANDIDATE: Personally I would treat them on Gallow's traction until there was good callus formation and then let them go home non-weight-bearing, if that is the designated discharge plan.

EXAMINER: OK.

The candidate is shown an x-ray of a midshaft off-ended radius and ulna fracture with 40° of angulation and early callus formation in a 9-year-old girl.

EXAMINER: This child was brought to the ED after the child was sent home from school. It was the first day back after the summer

holiday; teachers were concerned about the shape of her arm. Mum is unaware of any injury.

CANDIDATE: Well this is an AP and lateral x-ray of the right forearm. It shows an off-ended midshaft fracture of both the radius and ulna. There is translation of the radius and there is approximately 40° of dorsal angulation associated with it. There is a periosteal reaction and what would appear to be early callus formation.

EXAMINER: What are your thoughts?

CANDIDATE: I would be extremely concerned about NAI in this situation. The fact that there are signs of healing on the x-ray indicates that this injury is at least a week to 10 days old. It was an unstable fracture and so must have been extremely painful and unlikely to be missed by the child or the parents. The angulation on the x-ray is significant and it was sufficiently deformed that the school picked it up on observation on the first day.

The fact that there has been a significant delay to presentation, the fact that Mum claims there is no injury history, and the fact that no-one has noticed this girl having an extremely angulated and unstable injury is extremely concerning.

EXAMINER: Is this the kind of injury that you would expect to see in NAI?

CANDIDATE: Yes and no! We know that there are injuries which are very specific for NAI, such as rib fractures, humeral shaft fractures in <18/12 and metaphyseal corner fractures. Although these injuries are unlikely to have occurred without NAI, they form the significant minority. The bulk of NAI related injuries are exactly the same as occur through accidents.

Although this injury is by no means classical of NAI, I have a very high index of suspicion due to the delayed presentation, the neglect of its treatment for the first 10 days, and the claims from Mum that she was completely unaware of a problem until she was sent home from school.

EXAMINER: What would your management be if you saw this child in ED?

CANDIDATE: I would check and document the neurovascular status before and after splinting the arm with a plaster. I would examine the child from head to toe to look for any other injuries, including bruises and bite marks. I would admit the child from ED so that they were in a place of safety and make enquiries regarding any other children that may be in the home. I would inform the consultant on call, the paediatric team and the safeguarding team of my concerns. Then most likely she would require a skeletal survey.

EXAMINER: Okay, so that was done and a diagnosis of NAI was made following multiple fractures being seen on the skeletal survey. If we assume that the proper authorities are managing the social side of things, how would you manage the fracture?

CANDIDATE: Well, I don't think that it is acceptable to leave in its current position! At the very least it would require a trip to theatre for an MUA to correct the angulation and attempt to reduce it. I think that it is unlikely that you would be able to fully reduce the fracture at this stage leaving you with the decision about leaving it straight but off-ended and hoping that it remodels or accepting that you will have to do an open reduction and internal fixation.

EXAMINER: What would you do if it remained off-ended?

CANDIDATE: I think that in a 9 year old I would open it and plate it.

EXAMINER: Why not elastic nails?

CANDIDATE: I think that you will have to do a proper open reduction with a decent sized wound due to the callus formation. At this stage, your wound would be big enough to plate it. I appreciate that nails would be less invasive to remove down the line than a plate, but this could be an extremely challenging nailing due to callus and remodelling in the canal. All in all I think that I would plate it and if needed remove the plate.

EXAMINER: OK, let's move on.

Developmental dysplasia of the hip (DDH)

There is a very good chance that you will be asked about DDH in your children's orthopaedics oral. It is an 'A listed' topic. This in itself is not particularly helpful as it is a big, complex and controversial subject to learn. For starters it is very important to be able to recognise it on a clinical photograph or radiograph. Quite where the discussion will then go is anybody's guess.

Background

DDH encompasses a large spectrum of conditions from mild dysplasia through to frank irreducible dislocation of the hip. It is the most common paediatric hip condition presenting to orthopaedic services in the UK.

The incidence depends on the definition used. Early instability is extremely common, but >90% of cases will resolve by the age of 6– 8 weeks without intervention. Those using universal ultrasound screening quote the incidence as being 1/400 live births. The actual treatment rates are more in the region of 4–6/1000 live births. The left hip is affected 3 times more commonly than the right, and the process is bilateral in 20% of children.[b]

Aetiology

There are multiple theories regarding the aetiology of DDH. Its development is likely to be multifactorial rather than being attributable to any one cause. Common theories include:

- Mechanical factors – This theory states that intrauterine crowding leads to DDH through forcible adduction of the legs. This is supported by the predominance of DDH in first pregnancies (due to increased abdominal and uterine

[b] The examiners may ask you why the left hip is significantly more affected than the right. The left hip is adducted against the mother's lumbosacral spine in the most common intrauterine position (left occiput anterior). In this position less capital cartilage is covered by the bony acetabulum and dysplasia is favoured.

Table 25.1 Advantages and disadvantages of universal and selective ultrasound screening programmes for DDH.

Type of programme	Advantages	Disadvantages
Universal ultrasound screening	Minimizes late presentation Allows early treatment for the majority of cases Does not rely on clinical examination by junior doctors	Costly May lead to overtreatment through early assessment
Selective ultrasound screening	More cost effective Prevents overtreatment	More likely to lead to late presentation Most cases of DDH do not have risk factors

tone), multiple pregnancies and oligohydramnios. It similarly supports the association with packaging disorders such as torticollis, plagiocephaly and foot deformities[c]. The left hip is more frequently affected, and this is believed to be because the most common intrauterine position leaves the left leg adducted against the lumbar spine. Similarly, breech positioning in utero is associated with an increased incidence of DDH

- Genetic factors – There is an association with family history for DDH although there is no clear pattern of inheritance. Having a first degree relative with DDH increases the child's risk to 12%. If the relative is an identical twin then this risk increases to 36%. Rates also vary considerably between populations, with there being very high rates of DDH in Japan, but virtually no incidence in Black Africans
- Hormonal factors – Hormonal imbalances in the mother can be a risk factor for DDH. It is thought that the high circulating levels of progesterone and relaxin leading up to birth result in excessive laxity of the hip joint capsule allowing instability. This theory explains the very high rates of instability during the first 6 weeks of life that resolves spontaneously. It also explains the increased incidence in females who have their own hormonal production
- External factors – Communities that advise swaddling of babies (wrapping them tightly in a blanket), have high levels of DDH due to the forcible adduction of the hips. Communities that carry children on their hip with the legs abducted have negligible rates. This may partially explain the difference in incidence between races

Pathoanatomy

Early treatment in DDH is important as there is a clear progression of bony remodelling and contractures, which prevent reduction without major surgery. This process is as follows:

1. Laxity of the hip joint capsule allows reducible subluxation of the femoral head
2. As the hip subluxes the head everts the acetabular labrum with a small rim of acetabular cartilage. Pressure from the femoral head on the superior lip of the acetabulum causes segmental depression of the femoral head and anteverts the neck
3. As the hip dislocates it inverts the limbus (labrum, capsule and rim of acetabular cartilage) and allows interposition of the psoas tendon between the head and the cup. The psoas tendon pressing on the capsule causes the classic hourglass constriction. Pressure from the femoral head on the ilium leads to formation of a false acetabulum superior to the true socket
4. The presence of the femoral head within the acetabulum is required for its normal development. After dislocation the socket becomes very shallow with overgrowth of cartilage in the floor. The anterosuperior aspect of the acetabulum fails to develop leaving the hip uncovered anteriorly and laterally. There is hypertrophy of the ligamentum teres and the transverse acetabular ligament further blocking reduction
5. After dislocation has been prolonged, the musculature around the hip becomes contracted preventing reduction

This is why abduction bracing is commonly successful in early infancy, but usually fails as the child becomes older.

Oral questions

What is developmental dysplasia of the hip?
What are the risk factors for DDH?
What other conditions are associated with DDH?
What is Ortolani's test?*
What is Barlow's test?*

*An ability to describe these tests clearly will demonstrate that your theoretical knowledge extends into the clinic – Make certain you can do this. Their sensitivity decreases as the child gets bigger.

Screening programmes for DDH

Screening for DDH has been commonplace for decades. It is well-established that earlier identification and treatment of DDH can reduce the magnitude and duration of treatment. A recent study has shown that the risk of open reduction surgery increases progressively with late presentation, with 86% of cases requiring major surgery when presentation is later than 10 months of age[5].

[c] In the exam, you may be asked about associated hip abnormalities in a child with one of these abnormalities. Uncertainty has now been cast on the association with CTEV (clubfoot).

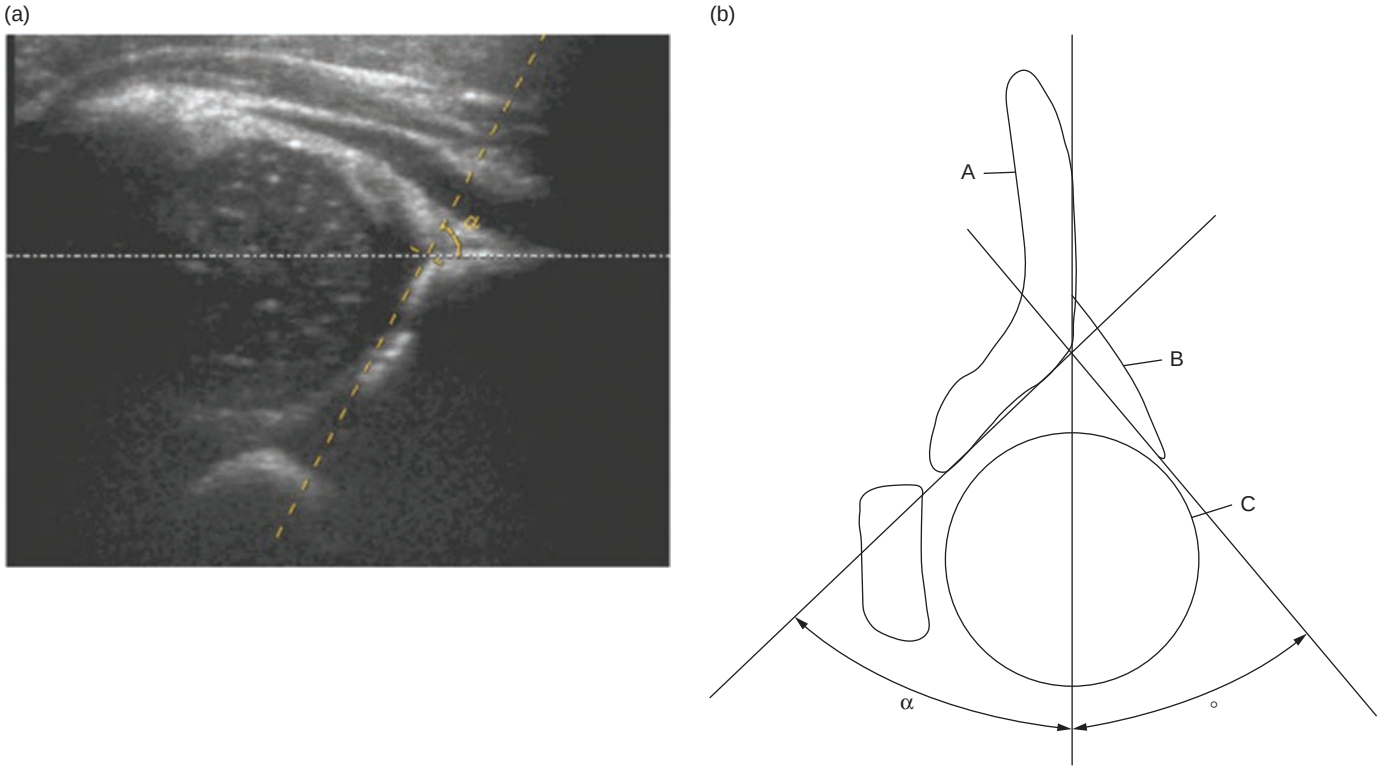

Figure 25.3 (a) Ultrasound image of the infant hip showing the Graf α angle calculation. The femoral head is >50% covered by the bony acetabulum. (b) Picture showing the features of an infant hip ultrasound. A – Bony ilium, B – Acetabular labrum, C – Femoral head, α – Graf α angle, β – Graf β angle

Clinical examination of all newborns for DDH is routine, assessing leg length, range of movement and the Ortolani and Barlow tests. The effectiveness of clinical examination is questionable, with the sensitivity being as low as 36% in inexperienced hands. Therefore, ultrasound screening for DDH has been established as the gold standard to assess hip morphology and stability.

Two types of screening programmes exist: Universal and selective ultrasound screening. In universal programmes all newborns are assessed by ultrasound, whereas in selective programmes only those with clinical concerns on examination or those with risk factors for DDH will be scanned[6]. The pros and cons of both systems are shown in Table 25.1

In the UK, most centres operate a selective ultrasound-screening programme based on the Newborn Infant Physical Examination (NIPE) programme[d]. This advocates clinical examination of the hips at birth and then again at 6–10 weeks of age. It has removed the 6–9 month, and 1-year hip check that was previously advised by the Standing Medical Advisory Committee (SMAC). The NIPE recommends ultrasound assessment for:

- Any child with clinical concerns regarding DDH
- Any child with a risk factor
  - Family history
  - Breech presentation
  - If resources allow – congenital talipes equinovarus (CTEV), metatarsus adductus, torticollis, oligohydramnios, high female birthweight

[d] Newborn Infant Physical Examination website: www.newbornphysical.screening.nhs.uk/

Radiological assessment

Ultrasound

Ultrasound is used in children younger than 4 months as the proximal femur is largely cartilaginous at that age. The consensus method for screening assesses the Graf angles, the femoral head coverage and tests dynamic stability. The Graf method uses the α angle and the β angle. The α angle lies between the ilium and the bony acetabulum, and the β angle is formed between the ilium and the acetabular labrum. An example ultrasound is shown in Figure 25.3a and 25.3b.

The Graf α angle should be 60° or more. If the value is less than this then the acetabulum is shallow. The Graf β angle should be <55°. If the hip is well located in the socket it should be easily covered by the labrum. As the hip starts to dislocate, the labrum is pushed out increasing this angle.

X-ray

The AP pelvis x-ray is used for the assessment of children over the age of 4 months. An example x-ray is shown in Figure 25.4a with an example of the lines used for assessment in Figure 25.4b.

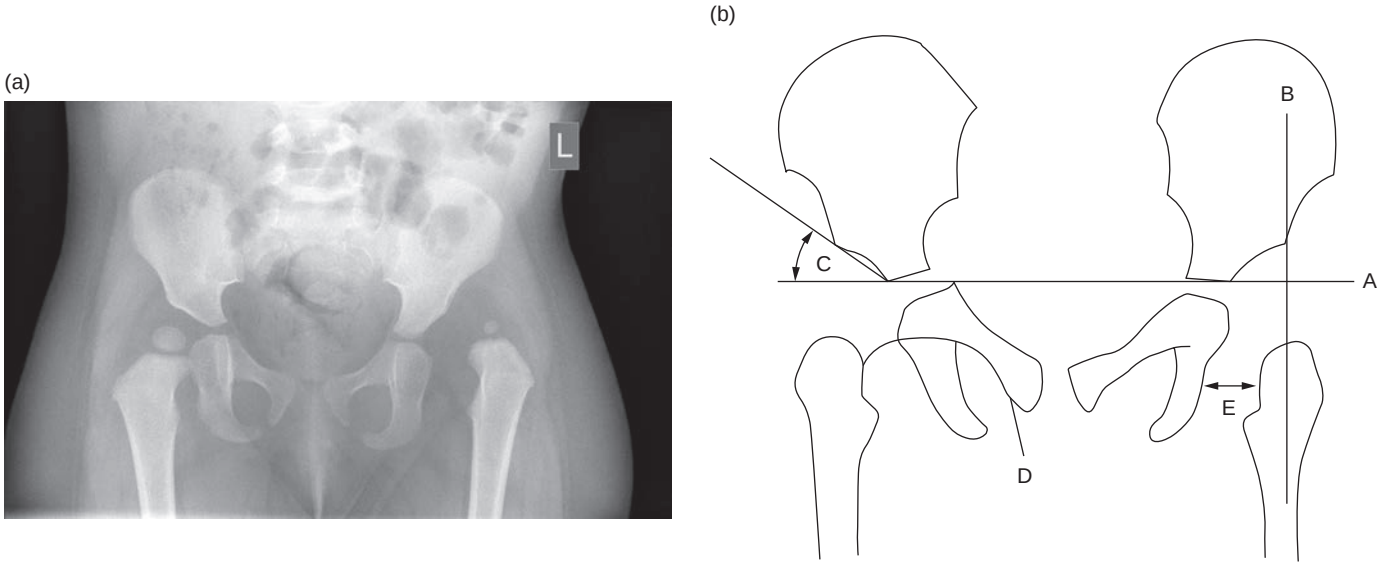

Figure 25.4 (a) AP pelvic radiograph of an 18-month-old girl presenting with left sided DDH. (b) Picture showing the lines used for assessment of DDH. A – Hilgenreiner's line, B – Perkins line, C – Acetabular index, D – Shenton's line, E – Medial clear space between ischium and medial femur

The x-ray shown in Figure 25.4a shows the classic features of DDH. The metaphysis of the left hip lies in the upper outer quadrant of a grid formed by Hilgenreiner's and Perkins lines (should normally be in the inferior medial quadrant). Shenton's line is disrupted, there is increased medial clear space (should be no >5 mm) and the acetabular index is increased. The proximal femoral ossific nucleus is smaller in the left hip than in the right.

Treatment

The principles of treatment for DDH are to obtain concentric stable reduction of the hip as early as possible whilst limiting the risk of complications. The development of the hip is dependent on the femoral head being stable within the acetabulum, and remodelling potential decreases with age. The vast majority of remodelling for the acetabulum occurs before the age of 4 years, but will continue to a degree until 6 years of age. Early reduction of the hip reduces the need for pelvic osteotomy by maximising remodeling potential.

Blood supply to the femoral head

The major risk of DDH treatment is avascular necrosis. Although the proximal femur has a plentiful blood supply, the physis acts as a complete barrier to flow. This means that any damage to the supply for the epiphysis will result in an avascular insult.

The profunda femoris artery supplies the medial and lateral circumflex femoral arteries. The lateral circumflex femoral artery supplies the majority of the anterolateral portion of the chondroepiphysis and the greater trochanter. The medial circumflex femoral artery gives off the posterosuperior ascending branch which pierces the posterior aspect of the hip capsule and ascends with the synovial reflection, tightly tethered to the back of the femoral neck. This artery gives off metaphyseal and epiphyseal branches to supply the bulk of the proximal femur. The physis acts as a complete barrier to diffusion making these vessels end-arterial.

The main blood supply for the femoral epiphysis in a child comes from the posterosuperior ascending branch of the medial circumflex femoral artery. Because it is tethered tightly to the posterior aspect of the femoral neck, if the leg is placed in extreme flexion and abduction it can be compressed on the rim of the acetabulum. Similarly, extreme adduction and internal rotation stretch the vessel and compromise the blood supply. This is why extreme positioning in a hip spica is contraindicated.

Treatment methods

Observation

Many hips assessed in a child during the first few weeks of life are seen to be physiologically immature or display instability. Ninety per cent of these children will stabilise without any intervention by the age of 6 weeks[7]. These children should be treated with double nappies or just observation, with a repeat ultrasound assessment after 6 weeks of age. The vast majority will not require any further intervention.

Abduction bracing

In the first 6 months of life, abduction bracing is the first line treatment. In the UK this is most commonly in the form of a Pavlik harness. The harness holds the hip in mild flexion and abduction to provide the optimum conditions for hip reduction. This allows the child to kick their legs and actively push the femoral head in to the acetabulum encouraging development.

Table 25.2 Features of the anterolateral and medial approach for open reduction of the hip.

	Medial approach	Anterolateral approach
Age of child	<1 year of age	Any age
Advantages	– Mechanical blocks to reduction are easily seen and accessible – Avoid splitting the apophysis	– Can address all blocks to reduction and perform capsulorrhaphy – Can address pelvis and false acetabulum through same incision – Away from the MCFA
Disadvantages	– Cannot address the false acetabulum, inverted limbus or perform capsulorrhaphy – Risk damage to the medial circumflex femoral artery (MCFA) causing AVN	– Split the apophysis – The acetabulum is very deep making it difficult – Risk damage to the lateral femoral cutaneous nerve of the thigh

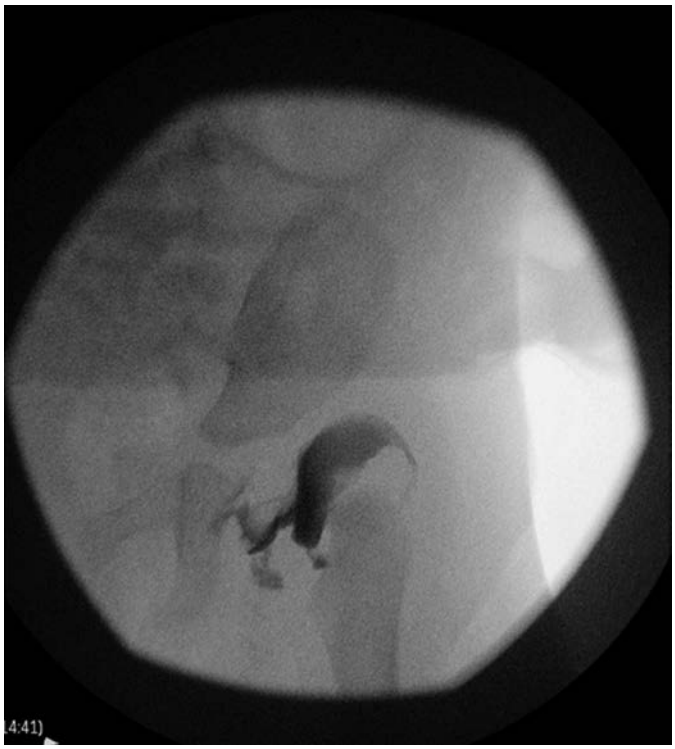

Figure 25.5 Picture of a hip arthrogram in DDH showing the left hip to be dislocated. The acetabulum is shallow with pulvinar in the floor. The psoas tendon is interposed between the head and the socket with an hourglass constriction of the capsule

In order for a Pavlik harness to be used the hip must be reducible on abduction and flexion. For irreducible hips, a maximum of 1–2 weeks can be allowed in a harness to see if it will reduce. If the hip remains dislocated then the harness must be removed or it can cause avascular necrosis, deformation of the femoral head and development of a false acetabulum.

Closed reduction of the hip

For those children who have failed Pavlik harness treatment, or those presenting too late to implement it, an attempt at closed reduction is the next logical step. This involves examination under anaesthesia (EUA), arthrogram and, possibly, tenotomies. An example of an arthrogram is shown in Figure 25.5.

There are three possible outcomes following an attempted closed reduction:

1. The hip reduces well and is stable throughout a good range of movement
2. The hip will reduce but needs to be held in an extreme position to keep the head reduced
3. The hip is irreducible due to obstructions

The child in scenario 1 has had a successful closed reduction. They should be treated in a hip spica for 6 weeks, then changed to another spica for a further 6 weeks.

The child in scenario 2 needs careful assessment. The 'safe zone of Ramsey' is the range of abduction through which the hip remains reduced in the acetabulum. If the hip requires wide abduction to keep the hip reduced, adductor tenotomies can sometimes improve the stability and increase the safe zone. If the hip can be held reduced in >45° of abduction, then this will typically be accepted. If extreme positioning is required to hold the reduction then the procedure should be abandoned to prevent development of AVN and plans made for an open reduction.

In scenario 3 the child clearly requires an open reduction. The arthrogram can be helpful to plan the need for any associated procedures that may be required. If the acetabulum is extremely shallow, then a pelvic osteotomy may be required. If the head is very high and cannot be easily brought down to the level of the acetabulum, then it is likely that a femoral shortening osteotomy will be needed.

Structures which may prevent reduction of the femoral head into the acetabulum are:

- Bulky ligamentum teres
- Hypertrophied transverse acetabular ligament
- Inverted limbus (acetabular labrum, capsule and rim of acetabular cartilage)
- Psoas tendon
- Hour glass constriction to capsule
- Pulvinar (fibrofatty material in the acetabulum)

Open reduction surgery

For those children where a closed reduction has failed, an open reduction is required. This can be performed through an anterolateral or medial approach, the former being more commonly used. Table 25.2 lists the features of these two techniques.

The goals of open reduction surgery are to remove all blocks to reduction, take down the false acetabulum and perform a capsulorraphy to stabilise the hip. At the end of the procedure the child is placed in a hip spica, typically in the human position to reduce the risk of AVN for 6–10 weeks.

Associated procedures

At the time of performing an open reduction it may become necessary to address the femur or acetabulum in order to attain and sustain reduction. These are as follows.

Proximal femoral osteotomy can be required for either shortening or derotation purposes. For older children with high-riding dislocations, there has commonly been contraction of the musculature preventing reduction. In these circumstances a pure shortening osteotomy is required to allow reduction of the hip without excessive pressure on the head causing AVN.

In those children with extreme femoral anteversion, whereby extreme internal rotation of the leg is required in order to keep the hip reduced, derotation is advised. This should be back to the normal level of anteversion for age, not 15°.

Pelvic osteotomy may be required in order to provide a congruent hip joint. If the acetabulum is so shallow or deficient anteriorly that the hip cannot be maintained in the socket, then this procedure may be required at the time of open reduction. Many surgeons defer this procedure to allow time for remodeling. If the acetabulum is still shallow by the age of 4, then consideration should be given to performing a redirectional osteotomy such as a Salter.

Figure 25.6 Treatment algorithm for children with DDH

Timing of surgery

There is significant controversy regarding the timing of surgery for DDH with regards to the risk of avascular necrosis. There are three schools of thought regarding management:

- The first group believe that the hip should be reduced at the earliest possible time to allow the maximal remodelling potential. These surgeons will progress through Pavlik harness, closed reduction and then straight on to open reduction as needed without delay, feeling that with modern techniques the risk of AVN is acceptable
- The second group will treat babies with a Pavlik harness and then attempt a closed reduction after this fails. However, they will postpone open reduction surgery until the age of 13 months or until the proximal femoral ossific nucleus appears as this is felt to be protective against AVN
- The third group feel that the risk of AVN is too great for any treatment other than a Pavlik harness. If this fails, they will wait until 13 months before attempting a closed or open reduction

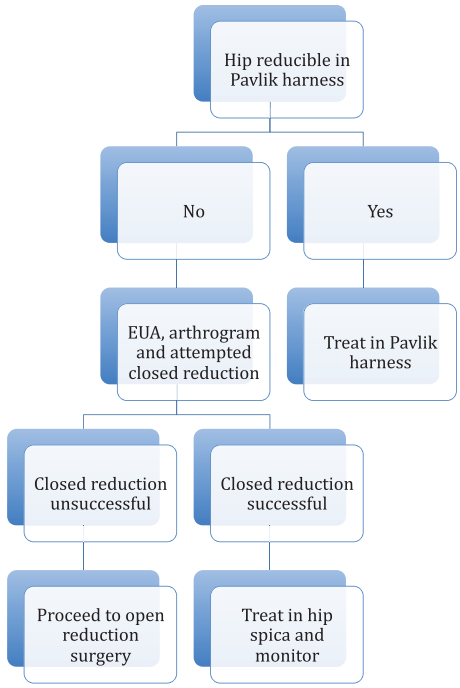

Figure 25.6 Treatment algorithm for children with DDH

There is a clear body of support in the paediatric orthopaedic community for each of these treatment plans. Earlier reduction gives the best chance of remodelling and reduces the need for further procedures such as pelvic osteotomies. We know that delaying surgery past 10 months of age significantly increases the need for open reduction surgery[8]. On the other hand, avascular necrosis is the worst of complications and the only problem we really don't have a good answer for. As long as you understand the reasoning behind your decision, you can safely give any of these answers in the exam.

Outcome

For those treated early with a Pavlik harness, the risk of requiring hip arthoplasty is not significantly greater than that of the normal population. It has been stated that 50% of children requiring open reduction surgery will go on to require hip arthroplasty in their 50s. Certainly outcome is related to the morphology of the hip at skeletal maturity, making femoral and acetabular osteotomy to correct residual dysplasia desirable[8].

Examination corner

Paeds oral 1

- DDH – Late presentation and role of arthrogram
- Economics of preoperative traction before open reduction

Paeds oral 2: DDH in an 18-month-old child

- History and examination
- Arthrogram findings
- Management options

Paeds oral 3
- Clinical finding in a neonate with suspected DDH

Paeds oral 4
- DDH management
- Open reduction indications and approaches
- Which soft tissues should be released or excised to ensure reduction
- Complications

Paeds oral 5
- An ultrasound image of DDH
- Anatomical structures and features on the ultrasound scan

Paeds oral 6

EXAMINER: What radiographic features are present in DDH?

CANDIDATE: The characteristic features include: Break in Shenton's line, increased acetabular index, delayed ossific nucleus, development of a false acetabulum, proximal and lateral migration of femoral neck and centre edge angle $<20°$.

EXAMINER: What would you find by examining the child?

CANDIDATE: On inspection, the child would have asymmetrical groin folds. They would have limited abduction and a leg length discrepancy. Galeazzi's sign demonstrates this, where the affected side appears shorter when the knees and hips are flexed. If the hip is unstable but congruent, Barlow's test would be positive. Ortolani's test would be positive if the head is dislocated.

EXAMINER: The child is 8 months old. What would your management be?

CANDIDATE: I would investigate this child's hip by ultrasound screening and an x-ray. To confirm the diagnosis I would perform an arthrogram under general anaesthesia. On confirming the diagnosis I would manage this with close reduction and spica cast.

EXAMINER:
- How do you do an arthrogram?
- What can you see on the arthrogram?
- How do you check the adequacy of reduction?
- What are the complications of treatment?
- How do you reduce the incidence of AVN?

Paeds oral 7
- Management of DDH presenting at 2 years
- Smith–Peterson approach to the hip

Paeds oral 8: Clinical photograph of Ortolani's test
- How is it performed?
- Sensitivity
- Management at 8 months
- General discussion about screening

Paeds oral 7

The candidate is presented with an AP x-ray of the pelvis for an 18-month old child with an established unilateral high-riding dislocation of the left hip (Figure 25.7)

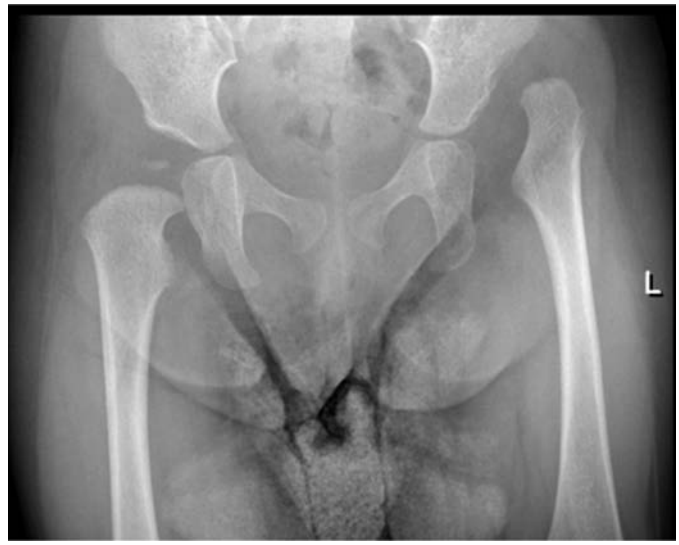

Figure 25.7 AP radiograph pelvis

EXAMINER: Tell me what you can see in this x-ray?

CANDIDATE: This is an AP pelvic x-ray of a 18-month-old child. The most obvious abnormality is a high dislocation of the left hip. There is disruption of Shenton's line, the metaphysis is lying in the superolateral quadrant of the squares made by Hilgenreiner's and Perkin's lines. The acetabulum is shallow with an acetabular index of approximately 35° and the proximal femoral epiphysis of the left femur is much smaller than the one on the right. The most likely diagnosis would be a late presenting DDH, but I would need to examine the patient to exclude other causes such as neuromuscular conditions.

EXAMINER: Yes, this was a late presentation for DDH. What would your management plan be for this child?

CANDIDATE: The principles of treatment are to get a stable concentric reduction as soon as possible whilst minimizing the risk of complications – In particular avascular necrosis. My plan would be to take this child to theatre to perform and EUA and arthrogram and attempt a closed reduction.

EXAMINER: Do you think that is likely to be successful at this stage?

CANDIDATE: No, I would tell the parents that I thought it would be unlikely to work but every now and then we are pleasantly surprised! I would tell them that if the hip went in closed then we would treat them in a hip spica. If the hip would not reduce we can get the information needed to plan which procedures may be required in association with an open reduction.

EXAMINER: Which procedures do you think may be necessary?

CANDIDATE: With a high-riding dislocation it is common to need to perform a shortening proximal femoral osteotomy to reduce the pressure on the femoral head and reduce the risk of AVN and re-dislocation. At the age of 18 months it is possible to perform a Salter pelvic osteotomy. If the acetabulum is extremely shallow and the hip is unstable after reduction of the hip, I would do this at the same sitting. If the hip was in joint and stable I would probably allow the child time to remodel, but warn the parents that it may be necessary to perform the procedure at a later date.

EXAMINER: What sort of age would you plan to do a Salter pelvic osteotomy if required?

CANDIDATE: Most acetabular remodelling occurs by the age of 4, so I would not do it before this stage. If the cup was still very shallow by 4 and not progressing well then I would list them at that stage. If it

was not too far off and still improving, then I would give them up until 6 years.

EXAMINER: What is the latest that you would do a Salter osteotomy?

CANDIDATE: The Salter relies on the elasticity of the pubic symphysis. It is recommended to perform the procedure between 18 months and 6 years. People have done this procedure later than 6 but personally I would not like to try it!

The candidate is shown an ultrasound picture of a dislocated hip.

EXAMINER: This is an ultrasound picture of a hip in a child with DDH. They are currently 3 months of age and the hip has failed to reduce in the Pavlik harness. It is now dislocated and irreducible. What would be your management plan from this point?

This question relates to the timing of interventions in DDH with respect to the risk of avascular necrosis. For children presenting in the first few months of life the established treatment sequence is to try abduction bracing, then a closed reduction if this fails, proceeding to open reduction as a last resort. There are three schools of thought regarding management of these children where an open reduction is required:

- The hip should be reduced as soon as possible and, therefore, you should offer a closed reduction, and then proceed immediately to open reduction if that fails. Although there is a risk of avascular necrosis, they do not believe that this is sufficient to warrant delaying hip reduction and losing the best remodelling potential for the acetabulum
- The second group will proceed to attempt a closed reduction as the risk of AVN is low. However, if this fails they will abandon treatment until the child is 13 months or the ossific nucleus develops in the hip. This is felt to be protective for the blood supply reducing the risk of AVN during open reduction surgery
- The last group will only try a Pavlik harness. If this fails, they believe that the risk of AVN with any other intervention is unacceptably high until the ossific nucleus develops. They would wait for this or until the age of 13 months before trying a closed reduction, proceeding to open reduction as needed

At this time, there is insufficient evidence to make a clear judgment as to which theory is correct. There is currently a UK wide multicentre trial running which will hopefully answer this question in years to come. At this point I believe that I would offer a closed reduction, as we know that delaying the time to surgery makes it significantly more likely that open reduction surgery will be required. The risk of avascular necrosis is low at this stage, and early reduction reduces the need for pelvic osteotomy in the future. Also, the risk of AVN following open reduction surgery is higher at 13 months than closed reduction at 4 months. I would not however, proceed to an open reduction until the age of 13 months or the development of the ossific nucleus to protect the blood supply.

EXAMINER: How would do the approach for an open reduction?

CANDIDATE: With a fully consented patient under general anaesthesia, I would position them supine on a radiolucent table with a sandbag under the pelvis. Routine prep and drape with chlorhexidine. I would perform an open adductor tenotomy primarily, dividing the tendons close to the bone to reduce bleeding. Then I would perform a bikini line incision over the anterior superior iliac spine. I would dissect down to fascia and carefully identify the interval between Sartorius and tensor fascia lata protecting the lateral femoral cutaneous nerve. I would split the apophysis of the crest stripping the outer table subperiosteally. I would then divide the straight head of rectus femoris after placing a stay suture in the tendon. Clearing the tissue from the hip capsule over the acetabular brim medially, the psoas tendon is divided at this level. I would then perform a t-shaped capsulotomy to open the hip.

EXAMINER: If you needed to perform any additional procedures, which approach would you use?

CANDIDATE: The pelvic osteotomy can be performed through the same incision by stripping the inner table subperiosteally following the apophysial split. If you needed to perform a femoral osteotomy then I would do a separate incision for a direct lateral approach.

Pelvic osteotomies

Principles

There are three categories of pelvic osteotomies:

- Re-directional – Where the acetabulum is deficient in one area, e.g. anterior deficiency in DDH
- Volume-reducing – Where the acetabulum is shallow, being deficient superiorly, e.g. in neuromuscular conditions such as cerebral palsy
- Salvage – Where the femoral head is not contained or the joint in incongruent, e.g. in avascular necrosis or Perthes' disease

Redirectional osteotomies

Redirectional osteotomies address a deficiency in one region of the acetabulum. In order to provide cover to this area, the acetabulum needs to be reoriented. This requires both columns to be cut proximally and the acetabulum to be rotated around a distal point. The procedure performed depends upon the age of the child.

- 18 months to 6 years of age – Salter osteotomy. This osteotomy hinges on the pubic symphysis, which is elastic in young children. The inferior fragment is then brought anteriorly and laterally to improve coverage of the hip before a bone block is placed to hold position. The bone graft is typically secured using two wires. (Figure 25.8a and b)
- 6 years to teenage – Triple pelvic osteotomy (Tonnis). After the age of 6 the pubic symphysis loses its elasticity. The ischium and pubis must be cut to allow the rotation to occur. Once position has been achieved, the fragment is secured using screws. (Figure 25.8c)
- Teenage to adulthood – Periacetabular osteotomy (PAO). Once the triradiate cartilage has fused, it is possible to perform a PAO. This keeps the posterior wall intact and keeps the point of rotation close to the acetabulum allowing greater correction. Once position has been achieved, the fragment is secured using screws. (Figure 25.8d)

The prerequisites for performing a redirectional osteotomy are that there is good movement of the joint, the deficiency can be addressed with the osteotomy and that the joint is congruent. Caution is advised to the surgeon performing femoral osteotomy at the same time as a re-directional osteotomy. By covering an area of deficiency, the surgeon uncovers another aspect of the hip. For example, during open reduction surgery for DDH, excessive derotation of the femur with a Salter osteotomy may result in posterior dislocation of the hip.

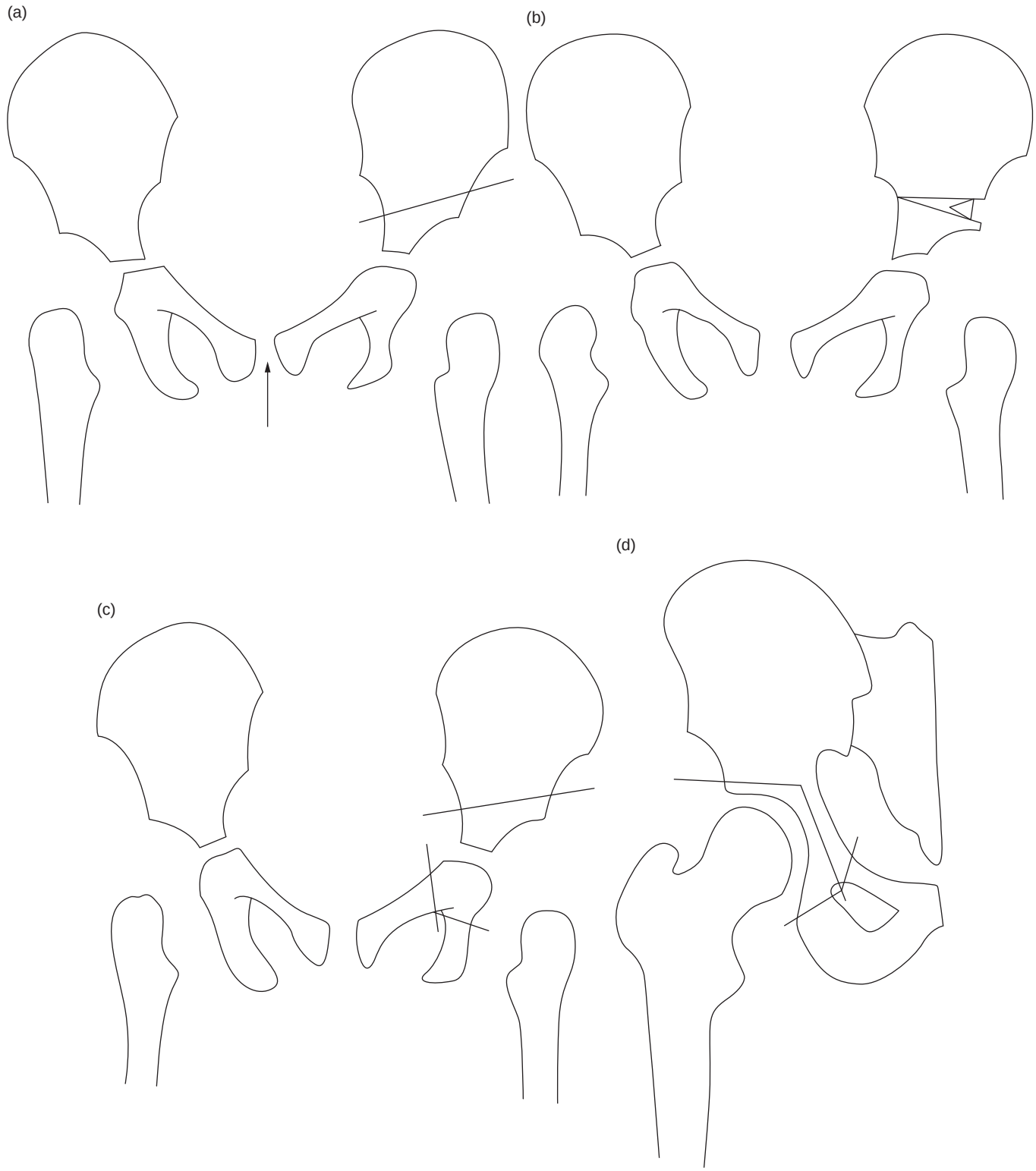

Figure 25.8 (a) Picture showing the cut required for a Salter pelvic osteotomy. (b) Picture showing the end position following a Salter osteotomy with bone graft interposed. (c) Picture showing the location of the cuts for a triple pelvic osteotomy. (d) Picture showing the location of the cuts for a periacetabular osteotomy

Volume-reducing osteotomies

The volume-reducing osteotomies are also referred to as posterior-hinging osteotomies. They are typically used in neuromuscular conditions such as cerebral palsy, where muscle imbalance causes the femoral head to gradually erode the superior acetabulum as the hip migrates. In order to

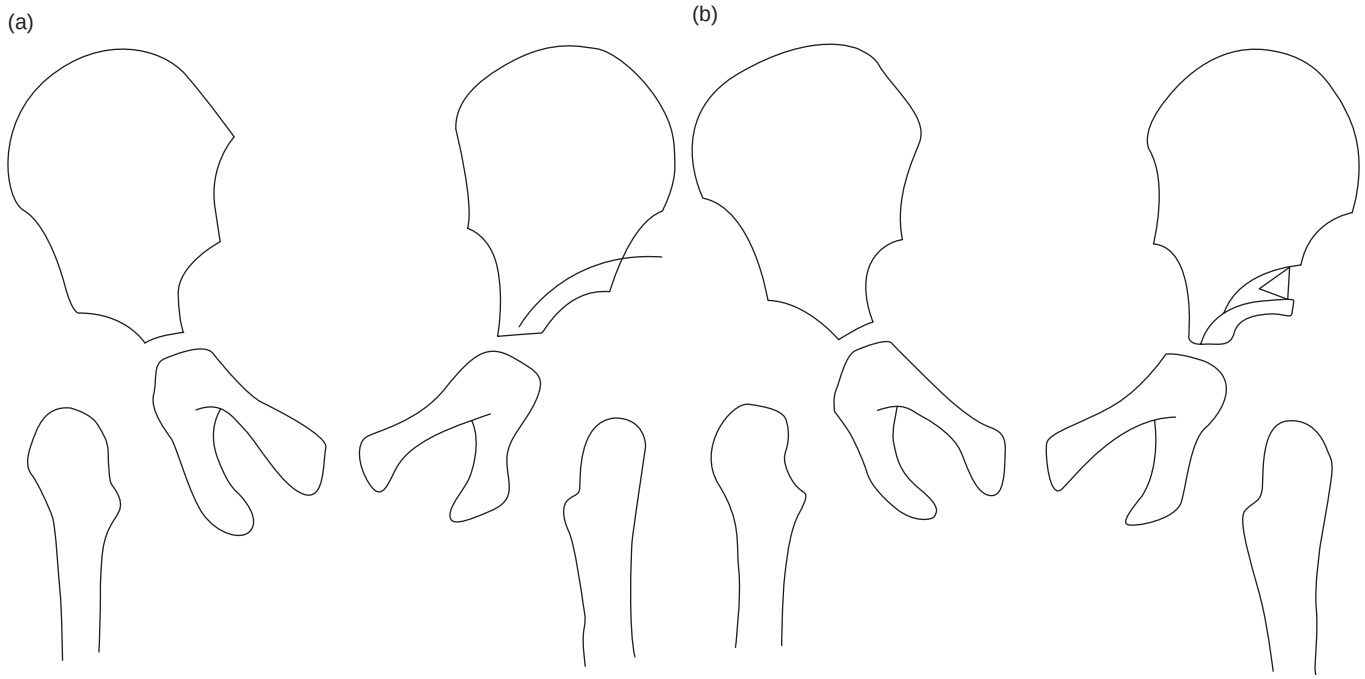

Figure 25.9 (a) Picture showing the location of the cut for a volume-reducing osteotomy. (b) Picture showing the position of the osteotomy following correction with bone block in situ

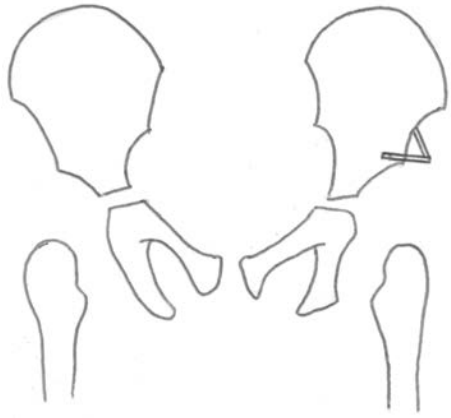

Figure 25.10 Picture of a Shelf pelvic osteotomy. Creates a bony shelf to cover the extruded part of the epiphysis

restore hip congruency, osteotomes are used to lever down the superior lip of the acetabulum and bone graft is used to hold the position. Dega osteotomy changes volume by cutting the outer cortex of the pelvis above the acetabulum down to the triradiate cartilage and deflected downwards. Pemberton osteotomy is similar to Salter's osteotomy but a small arc of the posterior column at the triradiate cartilage is left uncut and the acetabular roof is hinged on this arc to allow anterior or anterolateral coverage. The obvious advantages include that it is more stable than a Salter osteotomy, does not require metal fixation, and rotation rather than translation is more likely. Pemberton changes direction more than volume.

Prerequisites for this type of osteotomy are that there is good hip movement and the joint will be congruent. It must be noted that the acetabulum is commonly still deficient laterally due to erosion. These procedures commonly need to be associated with a proximal femoral osteotomy to seat the hip deeply in the acetabulum.

Salvage osteotomies

The salvage pelvic osteotomies are used when all else has failed! These can be used in any condition at any age. These osteotomies can be used when the femoral head is no longer contained fully in the acetabulum, or where the head is misshapen leading to incongruency. The most common procedure is the Shelf osteotomy, where corticocancellous bone graft forms a shelf over the lateral extent of the acetabulum to augment its surface. This allows a greater contact area and so decreases contact pressures for the hip, and stabilises the hip by preventing any further subluxation or hinge abduction.

The graft must be placed as close to the joint surface as possible. The hip capsule lies on the undersurface of the graft and undergoes metaplasia to form fibrocartilage in relation to the forces put through it on weight-bearing. Unless the shelf is placed just above the hip capsule then it will not allow the weight-bearing surface to be extended.

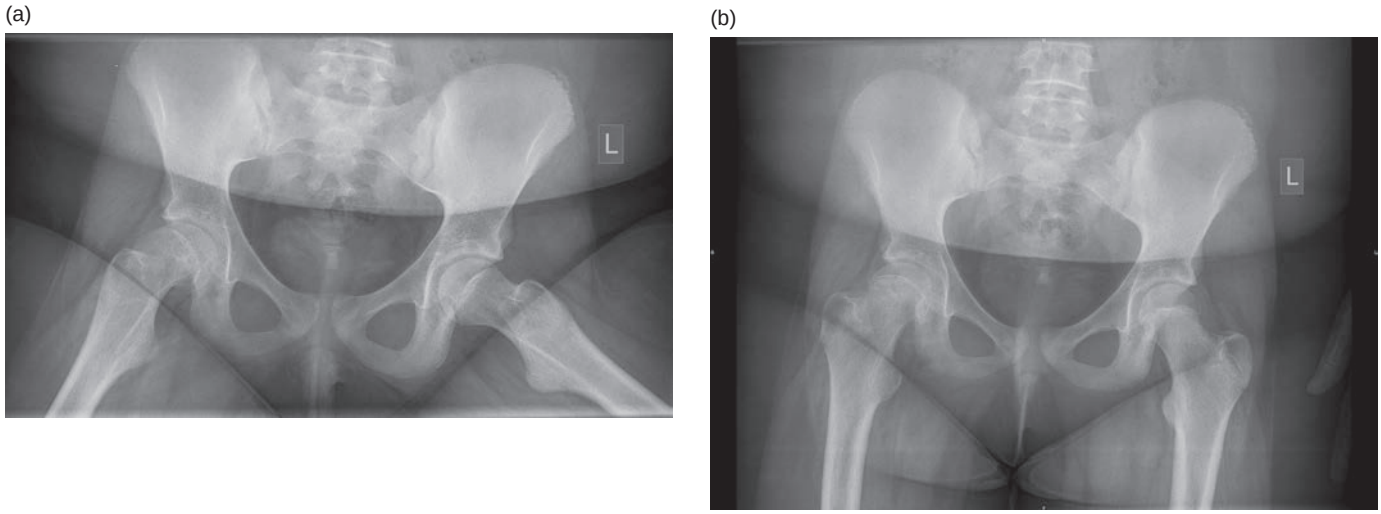

Figure 25.11 (a,b) Example AP and frog-leg lateral x-rays of a child with a right chronic mild SUFE demonstrating the features described above

Slipped upper femoral epiphysis (SUFE)

Background

SUFE is one of the most significant pathologies affecting the developing hip. There is a relative weakening of the perichondral ring of the physis making it more susceptible to sheer stress. This allows the metaphysis to slip superiorly, anteriorly and laterally. This deformity of the proximal femur can lead to decreased range of motion, pain and is likely to be one of the leading causes of cam-type impingement and development of early osteoarthritis[1].

The incidence is quoted as being 2–3/100 000 children; however, this appears to be on the increase. This may be due to our increased awareness of the condition or the increase in adolescent obesity. Boys are more commonly affected than girls (2 : 1), and the condition is thought to be bilateral in 25% to 60% of cases if you include pre-slips.

Aetiology

There are two main groups of children that develop SUFE: Teenagers and younger children with underlying endocrine abnormalities. For those presenting in teenage years, the underlying cause appears to be mechanical. For the younger children, the endocrine imbalance leads to weakening of the physis allowing the slip. Some of the main theories are discussed below:

- Mechanical factors
  - The perichondral ring of the proximal femoral physis provides resistance to shearing stress. During the adolescent growth spurt it becomes stretched and loses its integrity
  - Retroversion of the femoral neck is extremely common in SUFE. This positioning of the neck increases sheer forces across the physis
  - The physis is more vertically aligned in children sustaining SUFE, again increasing the sheer force
  - Obesity is a common risk factor as 5–6 times body weight affects the femoral head and neck during jumping and running, causing repeated trauma to the already weakened physis
- Endocrine anomalies
  - Children presenting with SUFE as a result of endocrine anomalies typically present much earlier and have a greater incidence of bilateral slips
  - Extremes of stature and hypogonadism may predispose to SUFE
  - Increased incidence with growth hormone deficiency, GH therapy, hypothyroidism and hyperparathyroidism
  - Chronic renal failure and Turner's syndrome have an association with SUFE
  - Deficiencies in sex hormones lead to an increased incidence of SUFE as they improve the integrity of the physis. After menarche girls are protected by their circulating oestrogen levels, making slips very rare

Diagnosis

SUFE classically affects children in and around adolescence (girls: 11–13, boys: 14–16). The child commonly presents with groin or knee pain, or even with a painless limp on an externally rotated leg.

Table 25.3 Management plan for acute treatment of SUFE

	Acute	Acute on chronic	Chronic
Mild slip	PIS	Gentle reduction of acute portion, then PIS	PIS
Moderate slip	PIS	Gentle reduction of acute portion, then PIS	PIS
Severe slip	ORIF – Timing controversial	Gentle reduction of acute portion, if moderate or mild PIS, is still severe then ORIF	PIS + corrective osteotomy

There is a wide range of clinical findings depending on the severity of the slip. For those with a mild slip, there will probably be a mild limitation of internal rotation in flexion in comparison to the other side. For severe slips, there will be a leg length discrepancy with the affected leg lying adducted and externally rotated. Movements will be restricted with obligate external rotation with flexion of the hip.

AP and frog-leg lateral x-rays should be obtained to identify and classify a SUFE. It should be noted that the positioning for the lateral view should be extremely careful to prevent any further displacement of the slip. In severe slips it will not be possible to get a lateral view; however, the diagnosis is clear on the AP x-ray in this circumstance.

Features seen on the AP pelvic x-ray in SUFE[e] (Figure 25.11a):

- Klein's line (the line extending along the superior aspect of the femoral neck) should intersect the lateral portion of the epiphysis, but this intersection is reduced or absent in SUFE
- Shenton's line is disrupted as the neck migrates superiorly
- Loss of the normal overlap of the femoral neck metaphysis and the ischium (Scham's sign)
- Steel's metaphyseal blanch sign is the density in the femoral neck caused by the overlap with the slipped epiphysis
- Reduced epiphyseal height

The frog-leg lateral film is extremely helpful for diagnosis in minor slips and for grading the severity of the slip. An example is shown in Figure 25.11b

Classification

SUFE can be classified according to stability (ability to weight-bear), duration of symptoms and the severity of the slip.

Loder classification

The Loder classification divided slips into stable or unstable. A child with an unstable slip is said to be in so much pain that they are unable to mobilize even with crutches.

[e] Classic oral question: 'What are the six radiographic features of a SUFE?'

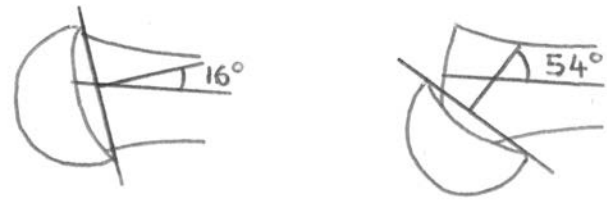

Figure 25.12 Picture showing the calculation of Southwick's angle in a mild SUFE (left) and a moderate SUFE (right)

Children with stable slips are mobile and weight-bearing to some degree.

The relevance of the stability of the slip is with regards to AVN. Loder described 96% good results in those with stable slips, but reported 47% AVN occurring in those with unstable slips. More recent studies have confirmed that AVN is more common in unstable cases[9].

Duration of symptoms

SUFE may also be classified according to the duration of symptoms:

- Acute slips – <3 weeks duration of symptoms
- Acute on chronic – Acute exacerbation of symptoms following chronic slip
- Chronic slips – >3 weeks duration

The relevance of the timeframe of the slip is with regards to surgical management. Those with chronic slips will have remodelling changes on the posterior and inferior border of the femoral neck in an attempt to stabilise the head. As the blood supply to the epiphysis travels along the posterior aspect of the neck, if the surgeon attempts to reduce a chronic slip they can stretch the vessel over the callus leading to AVN.

Severity of slip

SUFE is classified according to the severity of the slip using Southwick's angle. This is the angle between a line perpendicular to the epiphysis and the femoral neck line on a frog-leg lateral view (Figure 25.12). Slips are defined as:

- Mild – $<30^0$ difference from the other side
- Moderate – 30^0 to 60^0 difference from the other side
- Severe – $>60^0$ difference from the other side

It is important to realize that the angle stated is in comparison to the contralateral side. In a normally aligned hip Southwick's angle should be 0°. However, it has been recognised in recent years that the majority of children sustaining a SUFE have got retroversion of the hips affecting this value. If both hips have sustained a SUFE, then it should be assumed that the normal hip angle would be 0°.

Management

The goals of operative management in SUFE are to prevent any further progression of the slip, avoid avascular necrosis and provide the best long-term outcome possible for the child.

Pinning-in-situ (PIS)

For all minor or moderate slips, the general consensus is PIS is the best treatment. We know from past experience that manipulation of the slip to provide an anatomical reduction commonly resulted in AVN, particularly true in chronic slips. This is because the posterior blood supply for the epiphysis becomes tented over the remodeling changes from the SUFE and the vessels contract to accommodate their new position. By forcing the epiphysis back to an anatomical position the vessel is overstretched compromising the blood supply. The exception to this rule is the acute on chronic slip. It is acceptable in this circumstance, with very gentle positioning, to reduce the head to the chronic slip position prior to PIS.

The modern technique for PIS is to use a single cannulated screw placed as centrally as possible within the head on both AP and lateral views. The surgeon must be extremely careful to avoid penetration of the joint with the screw as this will lead to chondrolysis. This complication has been substantially reduced since the technique of single screw fixation has been adopted as opposed to multiple wires.

The screw typically requires an anterior entry point on the femoral neck to allow a clear shot at the centre of the epiphysis. This also provides protection to the vessels located at the inferoposterior aspect of the neck. Once adequate position of the guide wire has been achieved, a screw should be passed. The goal is to place five threads into the epiphysis and maintain good hold on the metaphyseal fragment with multiple threads as well. This gives the lowest risk of implant failure.

Hips treated with PIS are protected against further slippage with the minimum risk of developing AVN. They may still have problems long-term with femoroacetabular impingement, but this may be dealt with later as described below.

Reduction of severe slips

The severe slip presents a management dilemma. With this level of displacement the patient will definitely go on to develop problems with impingement without intervention. Unfortunately, the risk of AVN is also at its highest within this patient group.

Table 25.4 Reasons for and against prophylactic pinning of the normal hip in unilateral SUFE

Reasons for prophylactic pinning	Reasons against prophylactic pinning
Slips are bilateral in 25–60% of cases	Slips are unilateral in 40–75% of cases
A contralateral slip may be an unstable severe slip leading to AVN	Risks of surgery include infection, fracture, and chondrolysis
The child is definitely pre-disposed to SUFE	
If hip function is to be affected by a unilateral slip, preserving function on the other side is even more important	
Current complication rates following percutaneous screw fixation are very low	
Helps to minimize leg length discrepancy	

Chronic severe slips

For the chronic severe slip the two options for fixation are PIS in association with a corrective osteotomy to improve alignment, or open reduction and fixation. The majority of surgeons prefer to perform PIS and then correct the alignment through an osteotomy as this is felt to be the best compromise in terms of minimizing future complications without causing AVN. There are, however, surgeons who feel that open reduction is indicated in this group as they will definitely progress to osteoarthritis without correction. This open reduction may either be performed through an anterolateral approach with a trochanteric flip or by fully dislocating the hip depending on the training and preference of the surgeon. If the surgeon chooses to perform an open reduction then it is imperative to shorten the neck and remove any remodelling changes from the posterior surface of the neck. If this step is neglected then AVN is almost certain to develop.

Acute severe slips

The acute severe slip is a different scenario entirely. In this circumstance there is no remodelling posteriorly to endanger the vasculature. There is general consensus that open reduction of these slips is advisable. The timing of surgery is controversial. Work from Southampton, amongst others, has suggested that the risk of AVN in acute severe slips is related to the timing of surgery[10]. They found that the risk of AVN increased substantially past 24 hours of presentation. After this stage they advised placing the child on traction for a few weeks to convert the situation to a chronic severe slip and then manage appropriately. This has obvious implications for the

timing of tertiary referrals. The technique for open reduction is very similar to that for the chronic slip.

Acute treatment of SUFE summary

For all children with SUFE, the acute management is aimed at reducing the risk of AVN and preventing further slippage. Assessment must subsequently be made as to whether late procedures such as corrective osteotomies or osteoplasty will be required.

Management of the contralateral hip

There is controversy regarding the management of the contralateral hip in a unilateral SUFE. In young children or those with endocrine disorders the answer is simple. The rate of bilaterality in these cases is very high and so the other hip should be prophylactically stabilised.

For older children without an underlying condition the decision is harder. The reasons for and against prophylactic pinning are shown in Table 25.4.

There is increasing evidence that prophylactic fixation is worthwhile. Maclean and Reddy published their results of unilateral SUFE management in JBJS in 2006[11]. For those undergoing prophylactic pinning of the contralateral hip there were no complications. For those left unstabilised, 25% went on to develop another slip despite careful monitoring in clinic. Some slips were unstable and one child went on to develop AVN.

The author's viewpoint is that you have already identified a child who is predisposed to SUFE. Prophylactic single screw fixation at the time of surgery for the other hip is quick, safe and effective with very few complications. If that child progressed to develop an acute severe slip of their other hip with subsequent AVN then that situation was avoidable.

Late procedures

The two main late treatment plans to consider for SUFE are osteoplasty and corrective proximal femoral osteotomy.

Osteoplasty can be performed either open or arthroscopically. Shaving the extra bone of the metaphyseal hump prevents cam-type impingement as the hip is flexed and abducted. This procedure can improve the range of motion, reduce pain and potentially slow the progression to osteoarthritis. This is particularly helpful for mild and moderate slips.

Corrective proximal femoral osteotomy can be used to minimize residual deformity following SUFE. The aim is to realign the proximal femur to improve the range of movement and prevent impingement and progression to osteoarthritis. Multiple osteotomies have been described for correction of the deformity. The location of the osteotomy ranges from the intertrochanteric region to the physis itself. The closer the osteotomy comes to the physis the greater the possible correction that can be achieved. Unfortunately, the closer that the surgery is performed to the physis the greater is the risk of AVN. Again the surgeon is forced to balance the power of correction vs the risk of AVN. With the advent of arthroscopic osteoplasty, it seems to be more reasonable to perform an intertrochanteric osteotomy to reduce the risk of AVN. This can reduce the deformity to the equivalent of a mild slip that can be easily dealt with by osteoplasty.

Prognosis

The risk of development of osteoarthritis of the hip is dependent on the shape and congruency of the joint at skeletal maturity. Those developing AVN have got the worst prognosis for obvious reasons. Recent publications have suggested the residual changes from SUFE are one of the most common causes of cam-type impingement leading to early osteoarthritis[12]. Therefore, management of SUFE should first and foremost attempt to minimize deformity and prevent AVN. Long-term management should attempt to prevent femoroacetabular impingement to improve the longevity of the hip.

Examination corner

Paeds oral 1: Severe unstable SUFE

- Outline your management, including AVN rates and types of corrective osteotomy

Paeds oral 2: SUFE: Radiographic spot diagnosis

- Predisposition
- Management of severe grade III slip
- Fish and Dunn osteotomies: Examiner wanted to hear the word 'shortening'. Subcapital osteotomy without shortening carries an unacceptably high risk of AVN owing to stretching of the contracted posterior vessels as the head is reduced on the femoral neck

Paeds oral 3: Radiograph of severe SUFE

- Classification of slips, particularly the Loder classification system
- Incidence
- Management of severe slips: Pin in situ vs osteotomy
- Discussion about various osteotomies and complications of each (higher incidence of AVN in more proximal osteotomies such as Dunn compared to the Southwick biplanar osteotomy)

EXAMINER: Do you know any papers in the last year about management of severe SUFE?

CANDIDATE: I mentioned a review paper about management of SUFE[13]. This led on to discussion of another paper from Southampton concerning the timing of reduction and stabilisation of an acute, unstable SUFE[14]. The examiner knew both papers very well and we discussed the second paper in a fair amount of detail.

Paeds oral 4: Lateral radiograph of SUFE with history of sudden onset of knee pain

EXAMINER:

- What is the diagnosis?
- What will you find on clinical examination?

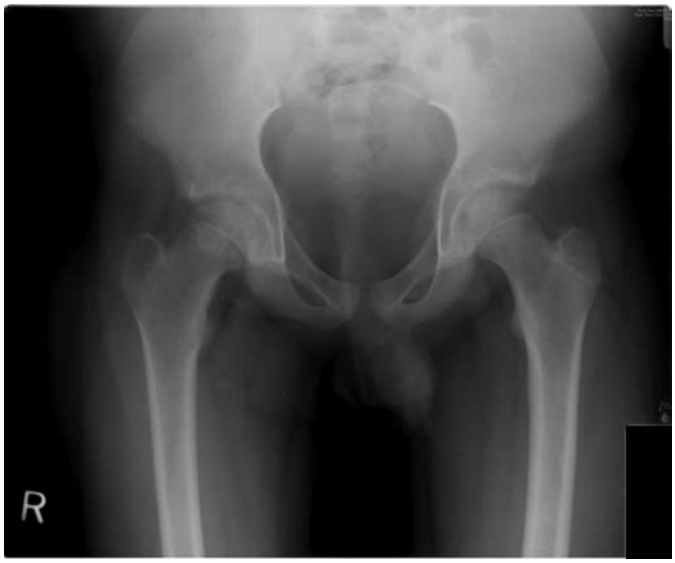

Figure 25.13 Radiograph mild SUFE

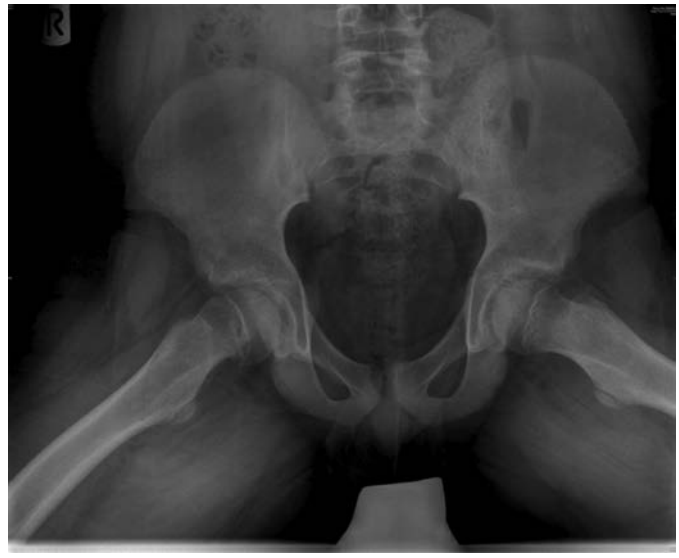

Figure 25.14 Frog-leg lateral x-ray of both hips

- What radiographic changes are present to indicate this as being an acute on chronic slip?
- What will you do?
- What complications can occur?
- What is the incidence of chondrolysis?

EXAMINER: Would you pin this boy's other hip? (It looked normal on the radiograph.)

CANDIDATE: No. But I will follow up with serial radiographs into maturity with advice to come back if there is knee or hip pain.

EXAMINER: What does the recent literature say on this?

CANDIDATE: There are papers that advocate fixing the normal side prophylactically. I would refrain except in situations of clinical need and (as he seemed to like the idea of prophylactic pinning, I added) in predisposed individuals.

EXAMINER: What are the predisposing conditions?

Paeds oral 5: SUFE: Aetiology, clinical presentation and management

Complications

- Outline the principles of single AO screw fixation and how you would do it
- Would you reduce the slip or fix in situ?
- Would you take out the screw – What does the literature say?

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The candidate is shown an x-ray of a very mild SUFE of the right hip (Figure 25.13).

EXAMINER: This is the x-ray of an 11-year-old girl who presented to your colleagues in the sports medicine clinic with an 8-month history of groin pain. They were concerned that there may be something more going on and so took this pelvic x-ray. What do you see?

CANDIDATE: Well, this is an AP pelvic x-ray of an 11-year-old girl. There is slight asymmetry of the hips, the right side is not completely aligned and appears to have lost some height in comparison to the other side. If we draw Klein's line along the superior border of the neck we can see that it runs through the epiphysis on the left hip, but just misses it on the right side. This is highly suggestive of a SUFE, I would like to see a frog-leg lateral to be sure.

The candidate is shown a frog leg lateral x-ray of both hips from the same child showing displacement of the right epiphysis (Figure 25.14).

EXAMINER: OK, here is the frog-leg lateral view.

CANDIDATE: Well it appears that there is displacement of the epiphysis on the right in comparison to the left. It is somewhat difficult to appreciate clearly because there has already been remodelling changes. I would say that this is a chronic mild SUFE.

EXAMINER: How would you classify a SUFE?

CANDIDATE: There are a number of classification systems available. The classification based on timing divides slips in to acute, acute on chronic and chronic. Acute injuries having occurred within a few weeks without any remodelling having occurred.

Another classification is based on the severity of the slip. This is based on the Southwick angle. That is the line perpendicular to the physis in relation to the line of the neck on the frog-leg lateral. The classification is based on the difference in comparison to the other side assuming that hip is unaffected. If the difference is $<30°$ then the slip is mild, 30–50° is moderate and $>50°$ is severe.

The Loder classification has also been devised referring to the stability of the slip. If they are able to weight bear then the slip is deemed stable and has a much better prognosis. If they are in so much pain that they are unable to mobilize even with crutches, then it is unstable carrying a significant risk of AVN.

The overall classification of SUFE is based on a combination of these systems. This would make this slip a chronic, mild, stable SUFE.

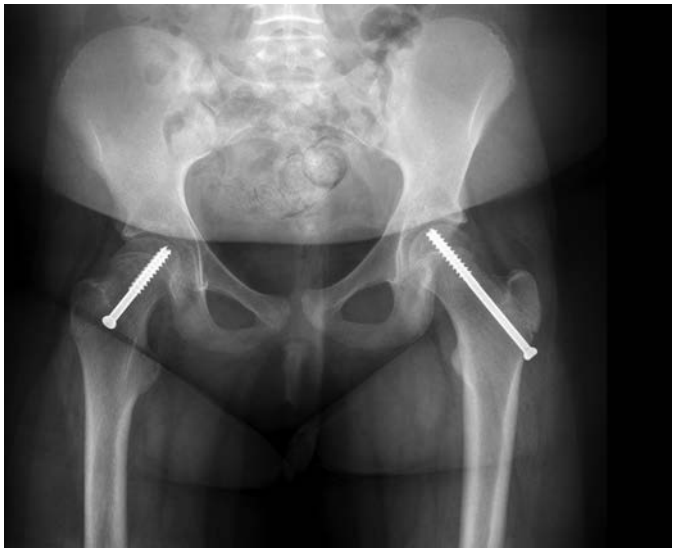

Figure 25.15 AP radiograph pelvis

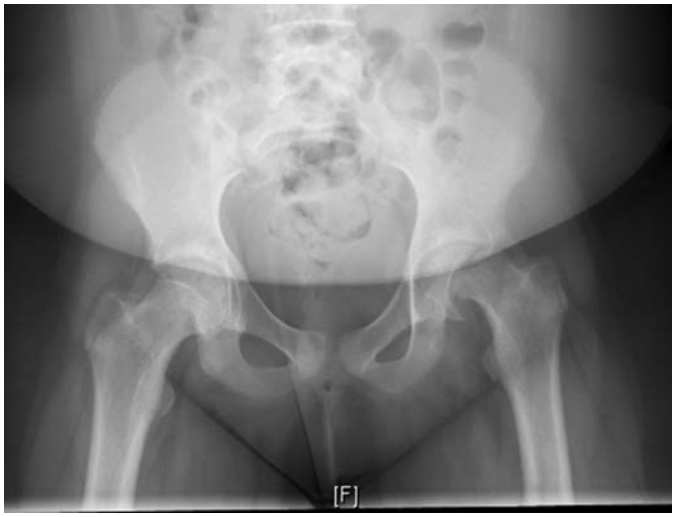

Figure 25.17 Radiograph severe acute SUFE

EXAMINER: What would be your management of this child?

CANDIDATE: I would take a full history and examine the patient. I would want to exclude underlying causes such as hormonal imbalances; however, at this age it would be relatively unlikely. I would explain the diagnosis to the family and the child and advise on pinning-in-situ for the right hip and I would advise pinning the left hip prophylactically.

EXAMINER: What would be your reasoning for pinning the other hip?

CANDIDATE: We don't know exactly why children get SUFE, but we do know that retroversion of the hip is a risk factor. It is thought to be bilateral in up to 40% of cases. By the fact that this child has presented with a right sided SUFE then you know she is predisposed. If she develops another mild, chronic SUFE then she will likely do very well with a PIS as she had on this side. If, however, she gets an acute severe SUFE then we know that her risk of AVN is going to be in the region of 50% regardless of what we do. With modern techniques of percutaneous single screw placement complication rates are extremely low. They published results of prophylactic pinning for SUFE from Nottingham and showed no serious complications.

As such, I would discuss this with the family and advise on prophylactic pinning. If they had very strong feelings against it, then I would just pin the right hip, otherwise I would pin both.

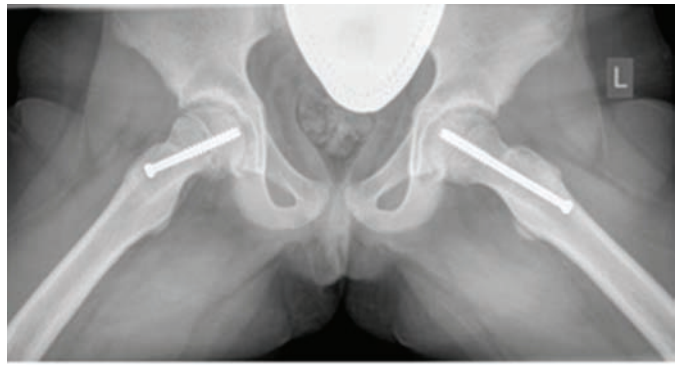

Figure 25.16 Frog-leg lateral radiograph pelvis bilateral screws in situ

The candidate is shown an AP and frog-leg lateral of the same child with screws in both hips (Figures 25.15 and 25.16).

EXAMINER: Well, it seems that the surgeon treating this child agreed with you. Here are the postoperative x-rays. What do you think about the quality of fixation?

CANDIDATE: Both screws are located centrally on both the AP and lateral and are contained within the head. The right-sided screw is closer to the joint than the left, but is clearly within the head.

EXAMINER: Why do you think that is?

CANDIDATE: In terms of fixation, you ideally want to have five threads across the physis to get a good fix. This can sometimes be more challenging on the slipped side.

EXAMINER: So, what would you do postoperatively?

CANDIDATE: Well the x-rays are very good. I would allow them to mobilize full weight-bearing on the left and partial weight-bearing on the right for 6 weeks to allow the physis to fuse and then place no further restrictions on her.

EXAMINER: OK, let's move on.

The candidate is shown an x-ray of a severe acute SUFE in a 12 year old (Figure 25.17).

EXAMINER: This young man is 12 years old. He was playing rugby when he felt something snap in his leg. He was brought immediately to ED and this x-ray was taken. Tell me what you think it shows (Figure 25.17).

CANDIDATE: This is an AP pelvic x-ray of a 12-year-old boy. There is a severe SUFE on the left hip with the epiphysis lying posterior and inferior to the metaphysis. The right hip looks to be OK on this film. Is there any further imaging?

EXAMINER: No, he was in too much pain to allow repositioning of the limb.

CANDIDATE: Were there any preceding symptoms from the hip?

EXAMINER: Not that he has reported. What would you like to do with this young man?

CANDIDATE: Well, this is an extremely rare and serious injury. As I am not a paediatric orthopaedic surgeon I would refer him on to one of my colleagues with more experience. If there was no-one appropriate in house then I would refer to the regional paediatric centre.

EXAMINER: OK, let's assume that you are now the paeds consultant in the regional centre – What are you going to do?

CANDIDATE: I would want him transferring in a blue light ambulance as a matter of urgency. Once he has arrived I would assess him clinically and get an urgent CT of the hip to clarify if it is definitely acute, or whether there is a chronic component. Then I would operate as soon as possible.

EXAMINER: What procedure would you do?

CANDIDATE: I think that I would advise an open reduction, shortening through the physis and screw fixation. I would not dislocate the hip as I have no experience in doing this and know that it can cause significant problems unless you have been trained to do so. If there were any signs of chronic change then I would carefully remove the posterior bone to prevent kinking of the vessels during reduction. I would also advise on prophylactic pinning of the other hip.

EXAMINER: What would you tell the family regarding prognosis from this injury?

CANDIDATE: I would say that it is a very significant injury. From the injury itself, the risk of avascular necrosis is anything up to 50% regardless of treatment. Reducing it quickly and effectively is the best chance of avoiding this. In terms of AVN, we will have to watch closely as it can take anything up to 18 months to present and we will only know the outcome after it has healed.

EXAMINER: Would your management be altered in any way if the injury was 3 days old when it was referred to you?

CANDIDATE: I know that there has been a paper published from Southampton suggesting that we should delay surgery if presentation is after 24 hours. They suggest that surgery should be performed within the first 24 hours or the child should be placed on traction for a few weeks to convert the injury to a chronic severe SUFE. They suggest that this reduces your incidence of AVN.

EXAMINER: Is that something that you believe?

CANDIDATE: I think that the numbers of these children are so small that it is difficult to really know. My inclination is that if they presented to me within the first 2–3 days then I would operate. This is on the basis that the blood supply may be just hanging on but stretched, and reducing it at this stage may avoid AVN. If it is longer than this, then the healing reaction has started and you will be causing a second hit which could kill the blood supply. Those ones I would put on traction and operate when it was chronic.

EXAMINER: And what would your management of a chronic severe SUFE be?

CANDIDATE: I believe that I would pin-in-situ and do a corrective osteotomy in the intertrochanteric region. If there were any further issues with impingement then we could arrange arthroscopic cheilectomy in the future.

EXAMINER: OK.

Legg-Calvé-Perthes' disease

Background

Legg–Calvé–Perthes' disease was first recognised in 1909 by Waldenstrom, who wrongly ascribed its cause to tuberculosis of the hip. In 1910 it was independently recognised as being caused by avascular necrosis by Legg (USA), Calvé (France) and Perthes (Germany). It is commonly referred to as Perthes' disease for brevity and we will use this term throughout this chapter.

The incidence of Perthes' disease has been falling over recent years both within the UK and globally. Currently we believe the incidence to be 1.5–4.0/100 000 live births, with a prevalence of 6–12/100 000 children. This level has decreased in the UK by 50% from 1990 to 2008[15].

The male : female ratio is 6 : 1, and the most common age of presentation is 3–7 years. The disease is bilateral in 15% of cases; however, it is never completely synchronous or symmetrical. If both hips are identically involved then the surgeon should seek an alternative diagnosis such as multiple epiphyseal dysplasia.

There is wide variation in incidence geographically, higher levels being associated with social deprivation. There is also a racial component with white children being most commonly affected[16]. The occurrence of Perthes' is affected by latitude, with an increase in incidence of 1.44/100 000 births for each 10° separation from the equator[16]. The lowest levels are reported in equatorial regions with the highest incidence being seen in Northern Europe[15].

There is a significantly increased incidence of genitourinary and inguinal anomalies in children with Perthes' disease. In particular hypospadias, undescended testes and inguinal hernia were frequently seen. Asthma is an independent risk factor for Perthes' disease even when steroid use has been accounted for. Generalized behavioural disorders have an association, although attention deficit disorder has not been shown to be linked[17].

Aetiology

Perthes' is an idiopathic avascular necrosis of the femoral head in childhood. Many risk factors have been identified over the

years; however, none of these account for the occurrence of disease in its entirety. The main theories are highlighted below:

- Genetic inheritance – It has been suggested that there is a genetic inheritance for Perthes' disease. This is difficult to establish due to confounding factors, such as social deprivation, smoking and diet. Also, there are a number of conditions that mimic Perthes' disease such as multiple epiphyseal dysplasia (MED). All of these conditions have a clear genetic inheritance potentially biasing results
- Thrombophilia – Thrombophilia has been suggested as a contributory factor. Multiple studies have shown an association, whereas many other papers have not demonstrated any link. This area remains controversial[18]
- Vascular deficiency – Recurrent infarction as opposed to one thrombotic event appears to be the underlying cause of Perthes' as seen in animal studies. Abnormalities of the hip vasculature both in vessel calibre and function have been identified in recent work adding further evidence to this theory[19]
- Environmental factors – There is a clear link to low social class and passive smoking[15]. This can partially account for the urban clustering seen in epidemiological studies. There are also links to dietary deficiencies, particularly vitamin D deficiency
- Endocrine anomalies – Children with Perthes' have a delayed bone age, and studies have shown reduced levels of somatomedins and abnormalities of the insulin-like growth factor-1 pathway[18]
- Other factors – There have been suggestions that Perthes' is caused by repeated microtrauma, inflammatory processes and subtle type II collagen deficiencies[18]

The prevailing opinion is that Perthes' is a multifactorial disease with genetic and environmental factors playing a role[18]. The model is that of the susceptible child undergoing a particular insult at a key stage of development.

Clinical presentation

The child may present with ongoing or recurrent pain in the groin or the knee. Frequently they are brought with a persistent painless limp.

On examination, the child is usually short for their age and very active. In early stages there may be nothing to see clinically, or there may be a slight limitation of internal rotation in flexion. As the disease progresses, there may be a fixed flexion deformity, restricted abduction and internal rotation. You may see obligate abduction and external rotation as the hip is flexed. Although 15% of cases are bilateral, it is rare to present with both hips being acutely affected at the same time.

Pathology

Several pathological findings have been shown in the hip in Perthes' disease related to the avascular insult and disordered growth:

- Synovial inflammation – Leading to widened joint space on x-ray
- Articular cartilage overgrowth – As cartilage continues to get nutrition from the synovial fluid even though the bone has lost its blood supply
- Physeal disorganisation – Columns of cells become disorganized and do not undergo normal calcification
- Epiphysis – There is initial necrosis and subchondral fracture formation. Sclerosis is followed by fragmentation as the head revascularizes and bone is resorbed. There is delayed ossification with bone healing occurring months after fragmentation
- Metaphyseal cysts – These are actually disorganized groups of cells extending from the physis into the metaphysis

Staging

Perthes' disease runs its course over a 2–3-year period. Waldenstrom described the stages of disease as follows:

- Evolutionary period
  - A – Initial stage – Dense epiphysis, irregular margin
  - B – Fragmentation stage – Epiphysis is flattened and divided
- Healing period – Epiphysis becomes homogenous, evidence of recalcification
- Growing period – Normal growth and ossification of deformed head
- Definitive period – Permanent residual features at skeletal maturity

This was modified and simplified by the Elizabethtown classification in to four stages:

- Stage I – Initial stage
- Stage II – Fragmentation stage
- Stage III – Healing phase
- Stage IV – Definitive stage

Classification

Salter and Thompson Classification[20]

The Salter and Thompson classification is used in the initial phase of Perthes' disease. This system is based on the extent of the subchondral fracture line on the frog-leg lateral film:

- Group A – <50% head involvement
- Group B – >50% head involvement

This classification gives the clinician an indication as to the severity of the disease. Unfortunately it only applies to the initial stage, and the subchondral fracture line is seen in fewer than 50% of cases (Figure 25.18).

Catterall classification[21]

The Catterall classification is more complicated and divides patients in to four groups based on severity of head involvement as seen on the AP and lateral x-ray of the hip:

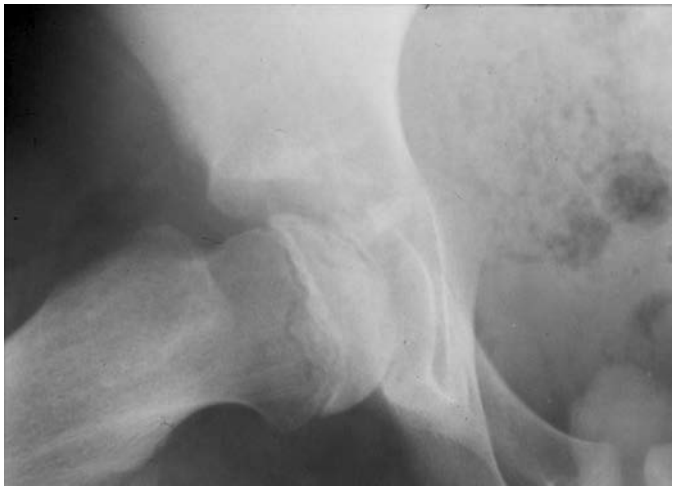

Figure 25.18 Frog-leg lateral x-ray showing a subchondral fracture line in early Perthes' disease

- Group 1 – Anterior epiphyseal involvement, no collapse, no physeal or metaphyseal involvement
- Group 2 – More extensive anterior involvement with some collapse centrally, pillars intact medially and laterally
- Group 3 – Near whole head involvement, 'head within a head' appearance. Central collapse with very small pillars medially and laterally intact. Metaphyseal involvement extensive with broad neck
- Group 4 – Whole head involvement and collapse. Physeal and metaphyseal involvement

With this classification system, the greater the proportion of head involved the worse the prognosis. It also makes reference to the fact that metaphyseal and physeal involvement indicate a more severe disease process and impact on the outcome.

Herring Lateral Pillar Classification[22]

The Herring Lateral Pillar classification is used during the late fragmentation phase of the disease. It is based on the level of collapse of the lateral pillar of the femoral epiphysis. This is defined as the lateral 15–30% of the head as seen on a true AP x-ray of the hip. They described three groups:

- Group A – No involvement
- Group B – >50% height maintained in comparison to the other side
- Group C – <50% height maintained in comparison to the other side

The relevance of the lateral pillar is in relation to the likelihood of containment of the hip. If it remains intact the lateral pillar acts as a buttress preventing subluxation of the head. Therefore, extensive lateral pillar involvement makes it much more likely that containment procedures will be required.

The classification was modified to include a B/C border group comprising a very narrow, poorly ossified lateral pillar or those with exactly 50% loss of height. This modification has not been shown to have a good inter-rater reliability[23] and adds little to the prognostic benefit of the system.

Determining prognosis in childhood

'Head at risk' signs

Catterall described both clinical and radiological 'head at risk' signs to warn the clinician of children likely to have a poor outcome.

- Clinical signs
  - Obesity
  - Decreased range of motion or recurrent admissions for pain
  - Adduction contracture in extension
  - Flexion with abduction
- Radiological signs
  - Calcification lateral to the epiphysis
  - Metaphyseal cysts
  - Increased medial clear space (signalling lateral subluxation)
  - Horizontal physis
  - Gage sign (lucent v-shaped defect in the lateral aspect of the physis)

Age and severity

The age of the child is extremely important in Perthes' disease, possibly due to the remodelling potential of the hip. Catterall identified three age groups:

- <6 years – Will typically do well unless there is severe involvement of the head
- 6 – 8 years – Will typically benefit from containment surgery unless very mild head involvement
- >8 years – Will typically do badly without surgery unless very minimal head involvement. Those with Herring C hips are thought to do so badly that it is questioned whether surgery is of benefit at all in this age group

Which children benefit from early surgery?

There are three main areas to consider when deciding whether or not a child would benefit from surgical containment: The clinical picture, the age of the child and the degree of head involvement.

The clinical picture is extremely important. In a child with multiple head at risk signs, recurrent pain and restricted range of motion, the surgeon should be concerned even if the child is young. This is the first factor to be considered.

After the clinical severity, the next important factor will be the age of the child. Those under the age of 6 will only do badly if the clinical course is very severe or if there is extensive head involvement. Those over the age of 8 will do poorly with much milder disease. Therefore, with increasing age the threshold for intervention should drop.

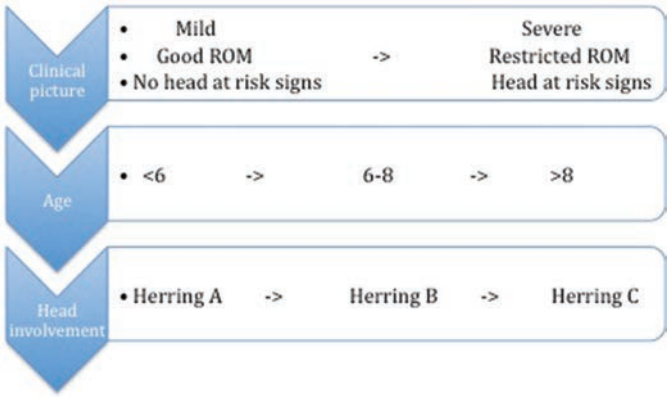

Figure 25.19 Determining prognosis in Perthes' disease

The last factor to consider is the level of head involvement. Even if the child is older, if there is only minor involvement of the head then they should do well. For those with total head involvement, particularly with greater involvement of the lateral pillar, the risk of future arthritis is significant.

It is extremely difficult in Perthes' disease to decide on the value of surgical intervention. For containment surgery, this should be performed before the head collapses and so the surgeon is really making an educated guess as to the likely outcome. Each patient is unique and so the decision for surgery must be considered individually for each case. The author uses the system below as a guide. Each of the three factors would be rated from good on the left to bad on the right. Greatest importance is given to the clinical presentation, however, if any factor is ranked on the right hand side of the diagram then surgery should be considered.

Management

The goal of treatment in Perthes' disease is to obtain and maintain a good range of movement of the hip, and to maintain containment of the femoral head in the acetabulum[18]. Due to the healing capacity and the remodelling potential of the juvenile hip, even if the head undergoes collapse it can still retain congruency if the head is contained and mobile. There are non-operative, medical and surgical means to try to achieve these goals.

Non-operative management

Non-operative measures include activity modification and physiotherapy to maintain range of motion. Surgeons differ in their opinion regarding the effect of physical activity in the outcome for Perthes' disease. Some feel that all impact activities should be restricted to prevent any unnecessary collapse of the head, whereas others feel that this is unlikely to make a difference. Bearing in mind that this process spans years in highly active small children, even if you advise no impact it is extremely unlikely that this will be achieved! There is no evidence that activity restriction prevents femoral head collapse[15,18], and it is worth noting that head collapse has been seen in children undergoing arthrodiastasis without any weight-bearing.

It is vital to maintain the range of motion to keep the sphericity and congruency of the joint. Physiotherapy can be helpful where there is some limitation of movement. In extreme cases, children have been admitted for periods of abduction traction to try to reduce contractures. If a child presents with decreased range of motion and needs repeated physiotherapy then that is a warning that the disease course is severe and consideration should be given to surgical intervention.

In previous years abduction bracing has been used to try to provide external containment of the hip. These braces have been shown to be ineffective, not actually preventing adduction of the hip. Studies comparing the outcome of bracing vs surgical containment have shown that in those children with a Herring Grade C hip, surgery achieved better results.

Medical intervention

In Perthes' disease there is a significant mismatch between bone resorption and formation. When revascularization occurs there is florid osteoclastic activity leading to weakening and fragmentation of the head. There is a notable delay before the osteoblastic activity occurs to heal and recalcify the head. This has led to significant interest in medical interventions which can decrease osteoclastic activity and increase osteoblastic function[15,18].

In recent years, the use of bisphosphonates has increased for many orthopaedic conditions including Osteogenesis Imperfecta. These drugs reduce osteoclastic resorption of bone and theoretically could prevent collapse of the femoral head. Early animal studies show very promising results in preventing femoral head collapse. There are concerns relating to the systemic use of bisphosphonates on the growing skeleton, intra-articular injection provides good results whilst limiting the systemic effect. Further work is needed before clinical recommendations can be made[18].

Other areas of promise are with the use of osteoprotegrin, BMP-2 and BMP-7. Osteoprotegrin works in a similar manner to bisphosphonates except that it reduces osteoclast formation as well as function. This appears to make it an even more effective drug than bisphosphonates in the early testing stages[18].

BMP-2 and BMP-7 are bone growth factors which have been commonly employed in the adult situation in order to try to stimulate bone healing. This strategy is aimed at increasing the osteoblastic function to get earlier healing and reduce the risk of femoral head collapse. Early results suggests that this may be effective, but there is a definite risk of heterotopic ossification with use around the hip[18].

Surgical intervention

Surgical intervention in Perthes' disease can be divided into preventive, remedial and salvage procedures[15]. These procedures are discussed below.

Preventive (containment) procedures

Preventive, or containment surgery, must be performed early in the course of the disease if it is to be effective[15]. These procedures are aimed at preventing subluxation of the femoral head to maintain a congruent joint. The most commonly used

Table 25.5 Advantages and disadvantages of commonly used containment techniques

Procedure	Advantages	Disadvantages
Proximal femoral varus osteotomy	• Internal fixation • Avoids any pelvic surgery • Effective procedure for containment of the head	• Limits abduction reducing range of movement • Shortens the leg • Alters proximal femoral anatomy for future arthroplasty
Acetabular augmentation procedures	• Internal fixation • Avoids alteration of femoral anatomy • Maintains leg length • Does not limit abduction	• Pelvic surgery where acetabulum is largely unaffected • Does not attempt to maintain femoral head sphericity
Arthrodiastasis	• Minimally invasive • Does not affect leg length • Does not affect anatomy of hip • Potentially avoid collapse completely	• External fixation • Pin site infections • Stiffness • Psychological issues

procedures are proximal femoral osteotomy, shelf osteotomy and arthrodiastasis. The advantages and disadvantages of these techniques are shown in Table 25.5.

Proximal femoral varus osteotomy is the most commonly performed containment procedure for Perthes' disease. Approximately 10–15° of varus is required to seat the femoral head more deeply in the socket and contain the lateral pillar[18]. In these circumstances, even if the head partially collapses it is likely that the joint will remain largely congruent improving outcome.

Some surgeons prefer to perform a Shelf procedure to augment the acetabulum. This achieves surgical containment by preventing subluxation and hinge abduction. The increased surface area reduces contact pressures by distributing load.

More recently arthrodiastasis has gained popularity. This is the placement of a hinged external fixator across the hip joint providing a small amount of distraction. This system will allow the hip to flex and the child to weight bear, but it prevents abduction of the hip. The concept of this system is to completely off-load the hip to prevent collapse of the femoral head. Early results have shown this to be effective; however, in extreme cases the head can collapse regardless of weight-bearing due to structural instability.

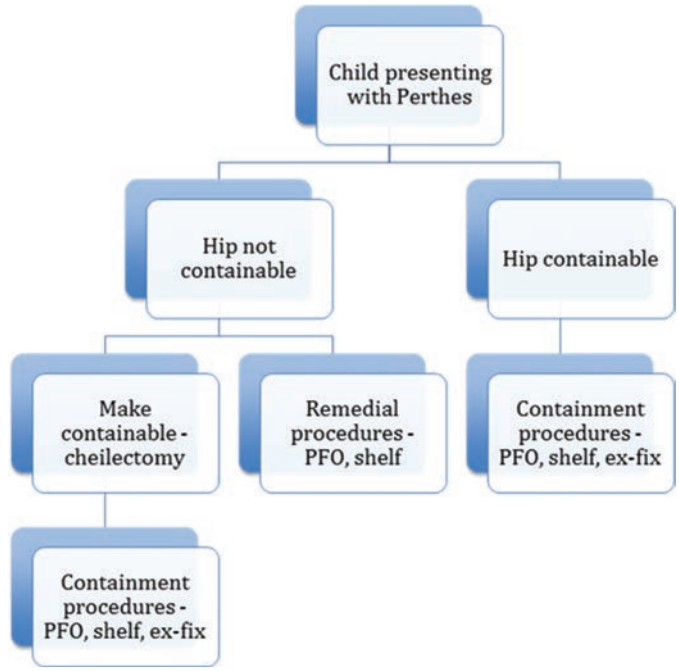

Figure 25.20 Treatment algorithm for surgical intervention in children with Perthes' disease

Remedial procedures

Remedial procedures are those attempting to limit the impact of the disease after the opportunity for containment has passed[15]. The goal of these procedures is to increase range of motion, limit pain, restore congruency as much as possible and slow the progression to osteoarthritis.

For those children with femoral head collapse, lateral subluxation and hinge abduction, cheilectomy can be very helpful. The subluxed fragment of head prevents full hip movement and makes containment surgery impossible. By removing this fragment (open or arthroscopically) the sphericity of the head can be restored allowing improved movement. This can be followed by containment procedures if desired.

An alternative solution in these cases is to perform an abduction extension proximal femoral osteotomy. The inferoposterior aspect of the femoral head is commonly spared in Perthes' and usually maintains its sphericity. By performing an abduction and extension osteotomy the surgeon brings this portion of the head in to the weight-bearing region and clears the extruded fragment. This improves range of motion and prevents hinge abduction, and may be augmented with cheilectomy if desired.

For children who develop a mushroom-shaped femoral head, acetabular augmentation is a good option. This allows the hip to be contained, prevents hinge abduction and increases contact area reducing contact pressures.

Salvage procedures

For those children who have had Perthes' disease and the head has healed in a non-spherical shape, salvage procedures may be considered. These are hip preservation techniques aimed at improving range of motion, preventing femoroacetabular impingement and subsequent progression to osteoarthritis. These procedures include osteoplasty, femoral neck reconstruction and redirectional pelvic osteotomies.

Recent papers suggest that arthroscopic osteoplasty for Perthes' disease is a useful procedure with good results[24]. It is worth noting that 39% of patients needed total hip arthroplasty (THA) 8 years following hip preservation procedures, and one third of patients requiring THA for Perthes' have had one of these techniques in the past[15]. Modern techniques may improve these statistics and we will have to await long-term outcome studies to confirm this.

Prognosis

The long-term prognosis for these children is based on the morphology of the hip at skeletal maturity. The Stulberg classification looked at the shape of the hip at maturity and described five groups[25]:

- Group I and II – Spherical congruent hips (normal or near normal)
- Group III – Aspherical congruent hips (flattened head but still round enough to allow good movement)
- Group IV and V – Aspherical non-congruent hips (flat head with or without abnormalities of the acetabulum)

For those children where the hip remained spherical and congruent (groups I and II), they did not develop early arthritis. For those with aspherical congruent joints (group III), they developed mild to moderate arthritis in late adulthood. For patients with aspherical non-congruent hips (groups IV and V), they typically developed severe arthritis before the age of 50.

This is why the management of Perthes' disease is aimed at maintaining a congruent joint. By containing the hip it has the best chance of remodelling to become spherical. Even if this is not achieved, we know that as long as the hip remains congruent then the survival of the hip is not severely impaired. It is only in those hips where the joint has lost congruency that degeneration is rapid and inevitable.

Examination corner

Paeds oral 1: AP radiograph of the pelvis with severe (obvious) Perthes' disease

A spot diagnosis oral (learn to recognise pattern radiographs of particular conditions). Testing a candidate's ability to articulate the radiographic features of Perthes' disease.

CANDIDATE: This is an AP radiograph of a pelvis in a child. The most obvious features are at the right hip. There is fragmentation and lateral displacement of the femoral head, concentric widening of the joint space, areas of increased sclerosis and metaphyseal cysts. The appearances are very suggestive of Perthes' disease.

EXAMINER: How do you manage Perthes' disease?

CANDIDATE: My initial management of Perthes' disease would be conservative – Analgesia and NSAIDs for pain relief. Regular review in clinic. Admission to hospital for bed rest and traction. Physiotherapy. Avoidance of activities that provoke pain.

EXAMINER: If the condition is not settling down, what else would you do if pain is severe and movement is grossly restricted?

CANDIDATE: I would perform an arthrogram.

EXAMINER: This is what we did; what can you see?

CANDIDATE: This shows that there has been some lateral subluxation of the femoral head with pooling of the dye medially.

EXAMINER: So how will you proceed?

CANDIDATE: If the patient is still having a lot of flare-ups and pain I would consider either a femoral derotation varising osteotomy or pelvic osteotomy. But it is very rare to need to do this. Most children with Perthes' disease can be managed with supervised neglect.

EXAMINER: How many hospital admissions would it take before you would proceed towards surgery?

CANDIDATE: About three.

EXAMINER: Humph, I would probably have a lower threshold for surgery.

Paeds oral 2: Similar AP radiograph of the pelvis demonstrating severe Perthes' disease at the left hip

EXAMINER: Yes, this patient has severe Perthes' disease with gross flattening of the femoral head. You mentioned Gage's sign being a radiolucency of the lateral edge of the epiphysis, growth plate and metaphysis. What are the other Catterall's head-at-risk signs?

CANDIDATE: We can see also see metaphyseal cysts, lateral subluxation of the femoral head, a horizontal growth plate and calcification of the lateral epiphysis on this radiograph, indicating that the patient has severe Perthes' disease.

EXAMINER: What clinical features are associated with a poor prognosis?

CANDIDATE: A patient older than 6, who is female and has a marked restriction of hip movements with recurrent episodes of stiffness.

EXAMINER: So how do you treat Perthes' disease?

CANDIDATE: I would manage Perthes' disease initially conservatively, with so-called supervised neglect. Analgesia, regular follow-up in clinic, physiotherapy and hospital admission for severe exacerbations. In only very severe cases would I consider surgery, such as a femoral derotation osteotomy or pelvic osteotomy.

The management of Perthes' disease is somewhat controversial. The literature is rather confusing as different authors have different indications for surgical treatment. In fact, some surgeons are sceptical about whether surgical containment works at all. Other surgeons are much more aggressive. Walk the middle ground and stay away from controversy unless you know the subject very well. You are there to pass the examination, not get the gold

medal. Remember 50% of patients do well without treatment and the majority of the remaining 50% will do well into their fifth decade without treatment.

Paeds oral 3
Perthes' disease: Aetiology, classification and prognosis.

Paeds oral 4: Radiograph of advanced Perthes' disease of the hip
- Diagnosis
- Classifications: Catterall, Salter–Thompson, Herring
- Prognostic factors
- How would 'you' manage this patient?

Paeds oral 4

The examiner shows the candidate a pelvic x-ray of a 4-year-old child with fragmentation of the whole of the right femoral epiphysis.

EXAMINER: This is the x-ray of a 4-year-old boy who presented with a limp. What can you tell me about the x-ray?

CANDIDATE: This is an AP projection of the pelvis, on which the most obvious abnormality is fragmentation of the right hip. The hip itself appears to be well located and the acetabulum appears normal. I would want to exclude an infective process, but I think that the most likely diagnosis would be Perthes' disease.

EXAMINER: That is right, it is Perthes'. What can you tell me about the aetiology of Perthes' disease?

CANDIDATE: By definition it is an idiopathic avascular necrosis, so we don't really know! It is clearly a multifactorial process, with links to the following risk factors:

- Gender – Boys are affected about six times more frequently than girls
- Low socioeconomic class – There is a clear link to living in more deprived areas, being urban rather than rural, but particularly social class 4 and 5. It is not clear if this is due to other factors such as dietary deficiency, passive smoking, etc. or is an independent risk factor
- Passive smoking – This is thought to increase risk by anything up to five times
- Coagulation defects – Although these children do not have an established thrombotic tendency, there have been many studies showing them to have traits, or borderline deficiencies. Other studies have failed to show this link. It is likely that there is some kind of intermittent thrombotic tendency in relation to external triggers which gives repetitive ischaemia. Very controversial!!
- Vascularity of the femoral head – There has been work showing that there may be different vascular supply in these children making them more prone to ischaemia. Recent work from Liverpool has suggested that the vessels have a reduced response to vasodilators, making them less able to regulate femoral head blood flow
- In reality, it is most likely a combination of several of these factors which combine to cause critical ischaemia for a brief period.

EXAMINER: And once you have made the diagnosis of Perthes' disease, how would you classify it?

CANDIDATE: Well this child is in the fragmentation stage, and so I would use the Herring lateral pillar classification. That grades the severity of the disease and the likely prognosis dependent on the extent of involvement of the lateral pillar of the femoral head.

- A: No involvement of the lateral pillar
- B: <50% loss of height in comparison to the other hip
- C: >50% loss of height in comparison to the other hip

EXAMINER: How would you grade this child's x-ray?

CANDIDATE: I believe that this would be a group C hip with >50% loss of height. It is worth noting that this child also shows some of the 'head at risk' signs. The physis looks horizontal, there are clearly metaphyseal cysts and there is a suggestion that there may be some calcification appearing lateral to the physis.

EXAMINER: What would you tell the family regarding the prognosis and your management from here on?

CANDIDATE: I would tell the family that younger patients with Perthes' have a better prognosis than older children. Being 4, he would fit in to the best outcome group. However, even at this young age, some children do go on to get bad results. Bearing in mind that he has full head involvement, is a C on the lateral pillar classification and also has some head at risk signs, I would be concerned about this young man. In the first instance I would get him in as soon as possible to perform and EUA and arthrogram to establish the shape of the femoral head and whether or not it could be easily contained. If this showed that the head was containable and there was a good range of movement, then I would suggest a varising proximal femoral osteotomy.

EXAMINER: Why do you think containment surgery would be necessary for this child?

CANDIDATE: As I said, the fact that he is young goes in his favour, but does not mean that he cannot get a bad result. He has extensive head involvement and collapse of the lateral pillar. That is prognostic that the head will likely collapse and sublux out of the joint because it has lost its lateral buttress. He is still in the early stages of the disease and so containment surgery makes it more likely that the head will reform to be congruent within the acetabulum, giving him the best possible chance of avoiding THR in the future. If you wait until the head collapses further and subluxes then you have missed that window.

EXAMINER: OK, that sounds reasonable. Let's move on.

The candidate is shown a pelvic x-ray with bilateral symmetrical hip pathology. Both femoral heads are flattened with a slightly widened metaphysis.

EXAMINER: Take a look at this x-ray and tell me what you see.

CANDIDATE: This is an AP pelvic x-ray of a child of approximately 6 years of age. The most obvious abnormality is in the shape of both proximal femurs. The acetabulum looks relatively normal for both hips, but there is symmetrical femoral head deformity with flattening. The physis appears to be slightly irregular with a wide metaphysis bilaterally.

EXAMINER: The family were told that this was Perthes' disease, but have come to you for a second opinion. What do you think?

CANDIDATE: I think that it is extremely unlikely to be Perthes' disease. As much as 15% of cases are thought to be bilateral, they are never synchronous and absolutely symmetrical. If you add to that the slightly irregular appearance of the physis and metaphysis, I think it much more likely that this represents a skeletal dysplasia. The most likely candidates would be multiple epiphyseal dysplasia or spondyloepiphyseal dysplasia.

EXAMINER: How would you make that diagnosis?

CANDIDATE: I would examine the child looking for any other signs or features of skeletal dysplasia (short stature, short limb segments, etc) and would get x-rays of other joints as indicated to show changes in other areas. After this I would tell the parents a small amount about skeletal dysplasia and then refer on to my colleagues in the skeletal dysplasia clinic for them to make a formal diagnosis and counsel the parents accordingly.

The candidate is shown an x-ray of the pelvis showing Perthes' disease of the left hip in the fragmentation stage. The femoral head has extensive involvement, has already started to collapse and has subluxed partially out of the acetabulum.

EXAMINER: This is the x-ray of a 7-year-old boy with Perthes' disease. Can you tell me what it shows?

CANDIDATE: This is an AP x-ray of the pelvis. The most obvious abnormality is fragmentation and partial collapse of the proximal femoral epiphysis of the left hip. There appears to be total hip involvement and this would be a Herring C on the lateral pillar classification as there has been >50% loss of height in comparison to the other side. The head has started to sublux laterally and is no longer fully contained within the acetabulum. He also displays several of the 'head at risk' signs with a horizontal physis, gage sign, metaphyseal cysts and calcification lateral to the physis.

EXAMINER: What would be your plan of management for this young man?

CANDIDATE: Well, I think that given the degree of involvement and collapse in combination with him being 7, the outlook for this hip is poor. Surgical intervention will likely improve his chance of developing a congruent hip joint for skeletal maturity. I would take him for an EUA and arthrogram to plan any further management. I would assess at this stage whether there would be any chance of containing the head or whether we would have to consider salvage procedures.

EXAMINER: What salvage procedures would you be considering?

CANDIDATE: If the hip was largely round but there was a bump preventing containment, the first option would be cheilectomy to allow you to perform routine containment surgery. This cheilectomy could be performed either open or arthroscopically with containment performed after that.

If it was felt that this would not be possible or ill-advised, then you could perform an abduction/extension osteotomy. What this does is bring the round portion of the femoral head up in to the weight-bearing region, and distalises the bump to prevent lateral impringement.

Alternatively you could perform a pelvic osteotomy such as a Shelf. This recovers the hip, spreads your contact pressures and reduces load, and stabilises the hip preventing hinge abduction. This is accepting that the hip will remain aspherical, but the hope is that it remains congruent giving the next best outcome.

A newer possibility would be to place a hinged hip distractor on to prevent any further collapse. This would not change the shape of the head, but would hopefully prevent any further degeneration. Then after the disease has resolved you could do a cheilectomy to try and restore the shape of the head.

EXAMINER: What would you tell the family regarding the prognosis for the future?

CANDIDATE: I would tell them that the prognosis is based on the shape of the hip at skeletal maturity and many things can happen between now and then to change the outcome.

EXAMINER: How would you assess the hip at maturity?

CANDIDATE: The Stuhlberg classification gives prognostic information at skeletal maturity. It divides hips into five categories:

- Types 1 and 2 are largely spherical and congruent
- Type 3 is aspherical but congruent
- Types 4 and 5 have misshapen femoral heads and the joint in non-congruent

For types 1 and 2, the prognosis is not that much worse than for the general population. Type 3 hips may require a total hip replacement approximately 10 years earlier than controls. Those with type 4 and 5 hips degenerate quickly and will likely need a hip replacement before 50.

Coxa vara

Definition

Localized bone dysplasia characterized by decreased neck-shaft angle (<110°) owing to a defect in ossification of the infero-medial femoral neck (Fairbank's triangle).

Epidemiology

- Incidence 1 : 25 000
- Bilateral in one-third to one-half of cases
- No clear pattern of inheritance has been established, but there are reports of positive family histories and of identical twins being affected

Aetiology

- Congenital (noted at birth). Often associated with a short femur or skeletal dysplasia. Nearly always unilateral
- Developmental (AD, progressive). Historically has been called infantile, develops over time
- Acquired (trauma, rickets, Perthes', SUFE). A defect of enchondral ossification in a metaphyseal triangular fragment of the inferior femoral neck, where physiological shearing stresses cause fatigue of the local dystrophic bone, resulting in a progressive varus deformity

Weinstein classification

- Coxa vara associated with hypoplastic femur or proximal focal femoral deficiency (PFFD)
- Coxa vara associated with congenital skeletal dysplasia
- Acquired coxa vara (trauma, metabolic diseases such as rickets and Perthes)
- Adolescent coxa vara associated with SUFE
- Idiopathic infantile coxa vara

Clinical features

- In unilateral cases children present with a painless progressive limp. The limp is not antalgic, it is painless and the weight-bearing phase is not shortened. In bilateral cases a waddling gait is noted
- Examination reveals a prominent greater trochanter on the affected side and weakness of hip abductors
- Positive Trendelenburg's test and gait
- In unilateral cases there will be a leg length discrepancy (2–3 cm) and the thigh and popliteal creases are uneven

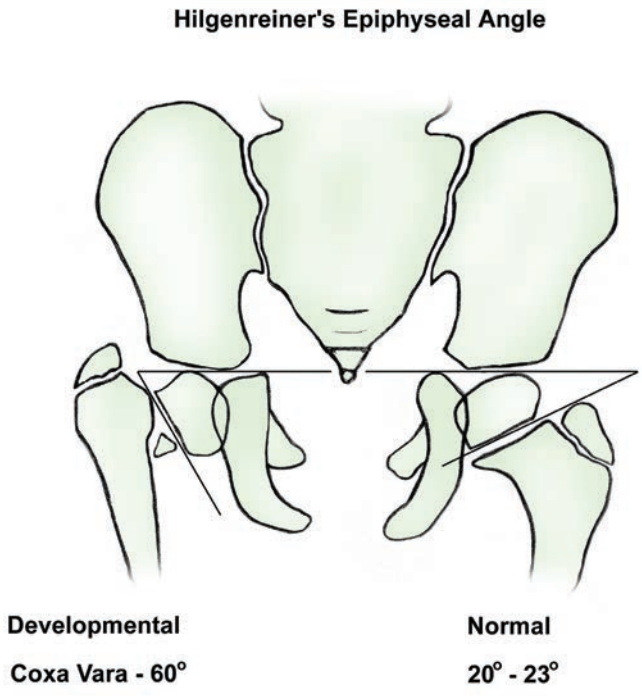

Figure 25.21 Measurement of Hilgenreiner's epiphyseal angle (HEA)

- Decreased internal rotation of the hip is often present owing to decreased femoral anteversion or true retroversion

Differential diagnosis

Congenital

- Proximal focal femoral deficiency
- DDH
- Achondroplasia
- Associated with fibula hemimelia

Acquired

- Rickets
- SUFE

Radiographic assessment

Hilgenreiner's epiphyseal angle (HEA): The angle between Hilgenreiner's line and a line drawn along the femoral capital physis. The normal angle is <25° (Figure 25.21).

Weinstein[26] found that if the HEA was:

- <45°, deformity corrects spontaneously
- 45–60°, outcome uncertain – Observe
- >60°, all patients will progress and, therefore, require corrective surgery

Management

The aim of surgery is to correct the deformity such that the neck-shaft angle is restored to 140° and the HEA to <35–40°. Pauwel's Y-shape and Langenskiöld intertrochanteric corrective valgus osteotomies are used for a neck-shaft angle <90°.

Leg length discrepancy

Definition

Leg length discrepancy is a measurable difference in the overall length of the two legs, which can be true, apparent or functional:

True – An absolute difference in leg lengths, clinically measured from ASIS to medial malleolus

Apparent – Where there is a measurable difference owing to positioning but the actual limb lengths may be the same. Clinically measured from xiphisternum or umbilicus to the medial malleolus

Functional – The difference the patient perceives (corrected clinically by blocks under the short limb)

Causes of leg length inequality (eight surgical sieves)

Congenital (small number but major difference in leg length discrepancy)

- PFFD (inequality remains proportional to the length of the opposite limb)
- Congenital short femur
- Tibia/fibula hemimelia
- DDH
- Vascular malformations (including Klippel–Trenaunay–Weber syndrome)
- AV fistula
- Diffuse haemangioma
- Seldom mentioned, but CTEV can be associated with a short limb as well as a short foot
- Hemihypertrophy

Trauma

- Diaphyseal fractures may lead to overlap and mal-union. This is usually a static, non-progressive, small leg length discrepancy
- Epiphyseal injuries can damage the growth plate. This may lead to partial (particularly Salter–Harris type III and IV) or complete growth plate arrest. If partial and the arrest is peripheral, it can cause a progressive angular deformity in addition to leg length discrepancy

Infection

Growth plate arrest in septic dislocation (Tom Smith's disease). Diaphyseal osteomyelitis can cause overgrowth owing to bone hyperaemia.

Neurological

- Cerebral palsy, polio and spinal dysraphism

Neoplasms

- Neurofibromatosis, haemangioma, Wilm's tumour

Skeletal dysplasia syndromes

- Hemihypertrophy and hemiatrophy syndromes (and knowing which it is can be a challenge)
- Russell–Silver syndrome (shorter lower limb)
- Klippel–Trenaunay–Weber syndrome (asymmetrical limb hypertrophy)

Inflammatory conditions

- Juvenile chronic arthritis (overgrowth)

Radiotherapy

Causes physeal damage and may lead to premature fusion of the growth plate.

Clinical evaluation

Standing

- Look for scoliosis, pelvic obliquity and joint contractures
- Stand on pre-measured blocks and reassess any scoliosis or pelvic obliquity
- Gait – Short-leg gait. On the short side, stance stride is shorter and push off reduced

Sitting

- Does the scoliosis correct? (If yes, then it is functional.)

Supine

- Get a tape measure. True leg length discrepancy measures the overall difference between the ASIS and the medial malleolus. Apparent leg length discrepancy is measured between the umbilicus and xiphisternum to medial malleolus
- Galeazzi's test – Look for flexed knee height with heels together
- Thomas' test to rule out flexion deformity of the hip
- Ankle – Rule out equinus deformity
- Knee – Flexion or hyperextension deformity
- Skin – Previous operative scars, café-au-lait spots
- Temperature variation in the limb may indicate a haemangioma

Prediction of leg length discrepancy

It is not unheard of to be presented with a growth chart in the oral. You should take time to familiarize yourself with them so that you can interpret them with confidence.

Green–Anderson tables

- Predict the remaining growth for the distal femur and proximal tibia according to skeletal age

Moseley straight-line method

- Moseley converted the Green–Anderson tables into a straight-line graph
- A logarithmic scale of predicting remaining limb growth along with expected discrepancy at maturity
- It assumes growth inhibition is constant and requires at least three scanogram measurements

White–Menelaus rule of thumb

- Used in the last few years of remaining growth (>10 years). This method assumes that
  - Distal femoral physis grows 9 mm/year (3/8 inch) (contributes 70% of femoral growth)
  - Proximal tibia physis grows 6 mm/year (1/4 inch) (contributes 60% of tibial growth)
  - Proximal femur grows 3 mm/year (1/8 inch)
- It further assumes that these physes fuse at the age of 16 in boys and 14 in girls
- Allows calculation of the discrepancy at maturity and the effect of epiphysiodesis. Reliable method as long as skeletal age is the same as chronological age

Eastwood and Cole method

- Leg length discrepancy measured using blocks or tape measure and the total discrepancy is plotted against chronological age. The points on the graph represent directly the pattern of increase in discrepancy
- Epiphysiodesis reference slopes are placed on the same graph

Bone age determinants (Greulich and Pyle atlas[27])

- AP films of the left wrist and hand are compared to radiographs in Greulich and Pyle atlas to determine skeletal age

Radiographic evaluation

Teleroentgenogram (grid films)

A single 3-ft radiograph of the entire lower limbs. Magnification distortion is minimal in small children, but it increases as the child grows bigger. Used in infants and young children.

Orthoroentgenogram

Also a 3-ft radiograph, but the radiographs are taken in 3 separate exposures centred exactly over the hip, knee and ankle to reduce magnification distortion.

Scanograms

Designed to avoid inaccuracies owing to projectional errors. A series of radiographs of the hips, knees and ankles exposed separately to avoid magnification errors is taken with the child in the supine position with a metal ruler in between the extremities.

CT scanogram

This is the preferred method in patients with angular deformities or joint contractures. It measures the distance from the top of the femoral head to the medial malleolus. Allows a more accurate measurement of the whole limb length, and involves less exposure to radiation.

Management

It is generally accepted that leg length discrepancy:

- <2 cm: Managed conservatively (shoe lifts up to 2 cm can be accommodated inside a shoe but thereafter must be attached to the sole)
- 2–4 cm: Epiphysiodesis is indicated in the longer limb
- >5 cm: Lengthening ± epiphysiodesis is indicated

Epiphysiodesis (surgical growth arrest)

Parents (particularly of children who are predicted to have a relatively short adult height) may struggle to accept a procedure that will limit the height of their child. This needs to be explained in the context of the greater investment required by everyone in the alternative – Limb lengthening on the shorter side.

Open growth plate arrest (Phemister technique 1933) has been replaced by percutaneous epiphysiodesis. Under radiographic control, a small window is cut in the peripheral part of the bone and the physis is curetted. This is usually performed 2–3 years prior to maturity on the distal femur or proximal tibial physis.

Staple epiphysiodesis is achieved by using medial and lateral staples. It is potentially reversible by removing the staples. It is less reliable than percutaneous epiphysiodesis, growth may not be retarded immediately and uneven inhibition may lead to condylar deformity. Attempts to correct a discrepancy of >5 cm may lead to miscalculation of limb growth potential or development of deformity owing to uneven retardation of growth. '8' plates have made this technically less demanding. In addition to their use for angular growth correction, they can be inserted medially and laterally to cause temporary physeal arrest. They must be removed by 2 years after insertion or permanent physeal arrest may occur.

Lengthening procedures

Periosteal release

This is a useful adjuvant procedure, particularly if a large limb length discrepancy (LLD) is anticipated. It can be repeated 4–5 years later although it is less effective. The procedure is performed by elevating and stripping the periosteal attachment adjacent to the growth plate.

Chondrodiastasis (physeal distraction)

Distraction force is applied progressively across the epiphyseal plate. The growth plate fractures, following which the bone is lengthened gradually and the distracted segment heals spontaneously. The operation is no longer widely used because initial lengthening is often followed by growth plate fusion. This has limited application and unpredictable results. Occasionally it is indicated for the correction of deformity sited at the level of the physis.

Diaphyseal lengthening

The diaphysis is divided and acutely lengthened by up to 3–5 cm. The lengthened bone is stabilised with a locked intramedullary nail. Supplementary bone graft is needed. The procedure remains unpopular because of a significant complication rate.

Diaphyseal osteotomy

The diaphysis is divided and followed by progressive distraction of 1.5 mm/day through an external skeletal fixation frame. Little new bone is formed within the gap and, once the desired length is achieved, the bone is fixed with a large plate with bone grafting across the callous bridge.

Ilizarov frame (circular)

The frame is formed by a series of full or half ring distractors and multiple small-diameter pins. The frame allows simultaneous correction of rotational and angular deformities as well as leg length discrepancy. Corticotomy is performed at the lower metaphyseal level. Internal fixation and bone grafting are rarely required. The disadvantages of this frame are the learning curve, a long initial operating time and tethering of muscles.

De Bastiani orthofix

A unilateral frame, which allows only distraction. Once attached it does not allow angular or rotational correction.

Complications

Serious complications can occur with leg lengthening:

- Pin tract infection, loosening of pins
- Osteomyelitis
- Deformity of adjacent joints
- Nerve palsies
- Vascular injuries
- Muscle contractures and weakness
- Premature consolidation
- Mal-union, delayed union and non-union
- Re-fracture after fixation removal
- Angular and rotational deformity

The safe limit of lengthening is 15% of the original bone length, although more has been attempted successfully.

Examination corner

Paeds oral 1: Clinical picture of a child standing with one leg on a wooden block

- Causes of leg length discrepancy
- Causes of undergrowth

- Causes of overgrowth
- Pathology of osteomyelitis

Picture of CT scanogram
Discuss the potential pitfalls with this technique.

Cerebral palsy (CP)

Definition

A permanent and non-progressive motor disorder owing to brain damage before birth or during the first 2 years of life. The lesion is static but (because a child is growing) the clinical picture is not.

Incidence

- Two per 1000. This is increased by resource-poor prenatal and postnatal care. It is also inadvertently increased where excellent care enables profoundly disabled children to survive where otherwise they would have died

Aetiology

This is not known in up to one-third of cases. The following risk factors have been identified:

- Prenatal: Placental insufficiency, toxaemia, smoking, alcohol, drugs, infection such as toxoplasmosis, rubella, CMV and herpes type II (TORCH), epilepsy, third trimester bleeding
- Perinatal: Prematurity (most common), anoxic injuries, infections, kernicterus, erythroblastosis fetalis, multiple births, trauma, placental abruption
- Postnatal: Infection (CMV, rubella), head trauma

In a discussion about aetiology it is important to recognise that, whilst low birth weight (often manifested in prematurity) is a strong risk factor for cerebral palsy, up to two-thirds of cases are born at term.

The influence of perinatal hypoxia is easily overplayed – It has only been demonstrated in 1 in 10 cases.

It is often impossible to give a definite cause in a given case; often the diagnosis is not apparent until several months and sometimes years after birth.

Classification

There is no universally accepted and satisfactory classification system for CP. It is best considered in terms of either physiology or anatomy.

Physiological classification

- Spastic (pyramidal system, motor cortex) – By far the most common
- Athetoid (extrapyramidal system, basal ganglia)
  - Ballismus – Uncontrolled proximal movements
  - Chorea – Uncontrolled distal (e.g. finger) movements
  - Rigid – Resist passive movement
  - Dystonic – Intermittent posturing
- Ataxia (cerebellum and brainstem) lack balance
- Mixed (combination of spasticity and athetosis)

Anatomical classification

- Monoplegia (one limb involved)
- Hemiplegia (one side of the body)
- Diplegia (lower limbs) with mild upper limb involvement – May be asymmetrical
- Triplegia – Three-limb involvement
- Quadriplegia or total body involvement

It is important to remember that epilepsy is coincident in one-third of cases, visual problems in 50% and mental retardation in 50%, with these complications being more common in more severely involved children.

Orthopaedic evaluation

The persistence of two or more primitive reflexes (Moro startle reflex, parachute reflex, tonic neck reflex, neck righting reflex and extensor thrust) usually means the child will be non-ambulatory.

Main problems with the musculoskeletal system are:

- Spasticity
- Lack of voluntary control
- Weakness
- Poor coordination
- Sensory impairment

Spasticity causes deformities that follow a staged pattern:

1. Dynamic contractures
   - Increased muscle tone and hyperreflexia
   - No fixed deformity of joints
   - Deformity is overcome during examination
2. Fixed muscle contractures
   - Persistent spasticity and contracture
   - Shortened muscle tendon units
   - Fixed deformity of joints: Cannot be overcome
3. Fixed contractures with joint subluxation/dislocation and secondary bone changes

Gait disorders are the most common problem. The use of three-dimensional computerized gait analysis and force plate studies assists in the development of and subsequent evaluation of an individualized management plan.

Hoffer classification of ambulation potential

Ambulation is classified to four grades:

Grade 1: Community ambulator
Grade 2: Household ambulator
Grade 3: Therapeutic ambulator
Grade 4: Non-ambulators

Gross Motor Function Classification System (GMFCS)

This is predictive for hip subluxation:

Level I: Walks without restrictions; limitations in more advanced gross motor skills

Level II: Walks without devices; limitations in walking outdoors and in the community

Level III: Walks with mobility devices; limitations in walking outdoors and in the community

Level IV: Self-mobility with limitations; children are transported or use power mobility outdoors and in the community

Level V: Self-mobility is severely limited even with the use of supporting technology

General management

- A comprehensive assessment of a child with CP is essential to plan appropriate management. Because of the multiplicity of problems, a *multidisciplinary team* is required
- Evaluation and management plans should be organized for motor, sensory and cognitive problems such as: Epilepsy, speech and hearing difficulties, visual defects, feeding difficulties, learning and behavioural problems
- Orthopaedics can only address spasticity problems and the deformity caused by the spasticity

The common sites of involvement are:

- Spine deformity
- Hip joint subluxation/dislocation
- Flexion deformity of the knee
- Foot and ankle abnormalities
- Flexion deformity of the hand

There are several schools of thought in respect of timing of orthopaedic interventions.

Traditionally, soft-tissue releases were undertaken successively with bony surgery reserved for difficult cases – The common practice of a surgery every year after a clinic visit led to the term 'birthday surgery'.

More recently, the advent of botulinum toxin has enabled some surgeries to be postponed so that single event multiple level surgery (SEMLS, also known as 'shark attack') can take place in the hope of delivering improved musculoskeletal function on a one-off basis. The risks (to ambulation) are high but so are the potential benefits.

Work in Sweden[28] has convincingly demonstrated that a surveillance programme with early intervention can deliver a much improved clinical picture for these children, with hip dislocation effectively eradicated.

Management options

- Dynamic contractures. Physiotherapy (stretching and casting), orthotic use, selective dorsal rhizotomy (this involves division of afferent sensory neurons to reduce spasticity. It is highly dependent on good patient selection and remains somewhat controversial. NICE published equivocal guidance in 2006 (reviewed in 2010), intramuscular botulinum injection and intrathecal baclofen
- Fixed deformity. Tendon release or lengthening, muscle transfer, split tendon transfers
- Bony abnormalities. Derotation osteotomy or joint arthrodesis

Clinical features

Spine

Scoliosis is the most common presentation. Surgical correction is usually considered when curves progress beyond 40° or there is worsening pelvic tilt. Custom-moulded seat inserts allow better positioning but do not prevent curve progression. Bracing is controversial and does not stop curve progression but may be able to delay it.

Scoliosis curves are divided into groups I (ambulators) and II (non-ambulators):

- Group I (double small curves with thoracic and lumbar involvement): Managed with posterior fusion
- Group II (large lumbar or thoracolumbar curves): Requires anterior and posterior fusion. If there is a significant pre-existing pelvic obliquity, then fusion to the pelvis is also needed to achieve adequate curve correction

Hip subluxation/dislocation

If hips dislocate they can be painful and make sitting and nursing difficult. Dislocation can contribute to pelvic obliquity and scoliosis.

Hip at risk

- Abduction $<45°$. Femoral head uncovered $>30\%$ (using Reimer's index on AP radiographs to give the migration percentage; Figure 25.22)
- Managed with abductor tenotomy. Iliopsoas tenotomy can be performed at the same time, but avoid in patients who can walk

Hip subluxation

- Head uncovered $>>50\%$
- Femoral varus osteotomy (derotation and shortening)
- Additional pelvic (Dega's in a growing child and Chiari post-maturity) osteotomy is occasionally necessary

Hip dislocation

- Early: Open reduction, femoral shortening and varus derotation osteotomy
- Late: Proximal femoral resection (*not* 'Girdlestone'), excision with interposition where symptomatic. Bone spikes at the resected proximal femoral end are a common complication

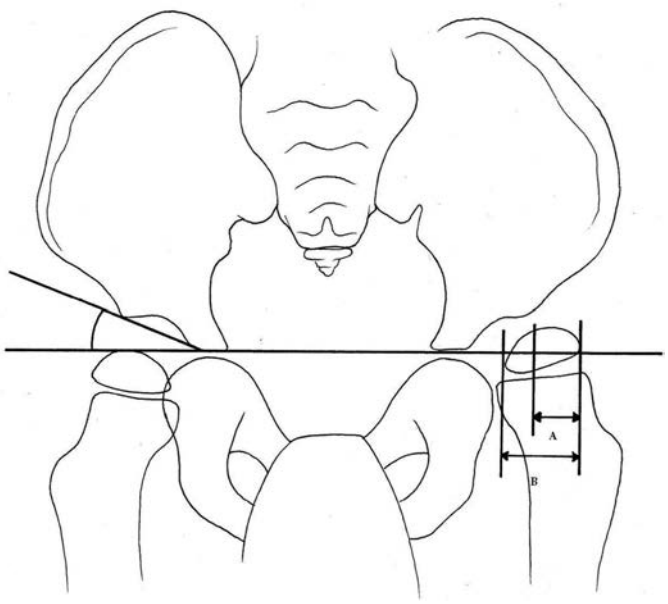

Figure 25.22 Hip migration index. The percentage of the femoral head that falls outside the acetabulum

Windswept hips

- Characterized by abduction of one hip and adduction of the contralateral hip
- Adductor release
- Fixed deformity in an older child may need to be managed by a combination of varus osteotomy on the abducted side and valgus osteotomy with shortening on the adducted side to create symmetry about the pelvis

Flexion contracture knee

- Knee flexion contracture with decreased range of movement may develop secondary to hamstring spasticity
- Spasticity of the quadriceps (cospasticity) is often associated with this deformity
- If hamstrings alone are lengthened, a stiff, flexed knee gait becomes a stiff, extended knee gait
- Indications for hamstring lengthening vary, but guidelines are a popliteal angle of 90–100° in non-ambulators and of 135° in ambulators
- Distal lengthening is preferred in ambulatory patients

Spastic crouch contracture

- Gait with flexed knees and hips, ankle dorsiflexion which commonly develops in spastic diplegia (weight of the child increases in a cubic fashion whilst the muscle strength only increases in proportion to cross-sectional area of the muscle)
- Either psoas or hamstrings or both are responsible
- May be iatrogenically precipitated by lengthening the Achilles tendon
- Is often progressive – With knee pain often leading to a late presentation to orthopaedic services

Foot and ankle

Ankle equinus

Caused by triceps surae contracture. A Silfverskiöld's test differentiates between tight gastrocnemeii alone and accompanying tightness of the soleus by assessing dorsiflexion with a flexed and extended knee, respectively. Various surgical procedures are described for the correction of the equinus deformity.

1. *Baker's procedure*
   - An inverted U incision of the gastrocnemius aponeurosis is performed through a vertical midline incision in the middle one-third of the leg
   - Aponeurosis slides apart in a controlled and stable fashion
   - There is a high recurrence rate that makes relengthening virtually impossible
2. *Strayer's procedure*
   - Via a mid posteromedial calf incision the gastrocnemius is 'peeled' from the underlying soleus, which is not damaged
3. *Percutaneous Achilles tendon lengthening*
   - A triple hemisection technique, taking care to dorsiflex the foot only to the plantigrade position
   - Minimal scarring: The incisions are two lateral and one medial
   - The most unpredictable procedure, especially in young children, as it does not respect soleus
4. *Slide technique (White) of Achilles tendon lengthening*
   - DAMP: **D**istal **a**nterior two-thirds, **m**edial two-thirds **p**roximal
   - Small risk of over-lengthening
5. *Open Z-lengthening of Achilles tendon*
   - Neglected cases, severe deformity
   - Risk of over-lengthening and calcaneus deformity

Those procedures which preserve soleus are generally preferred as the soleus is the primary 'antigravity' muscle of the ankle and its function cannot be easily recovered. If Achilles tendon lengthening is performed with tight hamstrings, then a crouch gait occurs, and the child walks with their ankles maximally dorsiflexed and their knees flexed. In this situation a GRAFO (ground reaction ankle foot orthosis) is required to try to maintain ambulation. Hamstring tightness should be corrected at the same time as Achilles tendon lengthening.

Equinovarus

- Most often caused by tibialis posterior spasticity, although occasionally the tibialis anterior tendon is at fault
- Split tibialis posterior transfer involves rerouting half of the tendon dorsally to the peroneus brevis

- Split tibialis anterior tendon transfer to the cuboid is often combined with Achilles tendon and tibialis posterior lengthening to manage a fixed equinovarus

Equinovalgus

- Spastic peroneal muscles pull the forefoot laterally
- Excessive valgus, external rotation and dorsiflexion of the calcaneus in relationship to the talus

Management

- Ankle foot orthoses. Grice arthrodesis. Subtalar extra-articular arthrodesis performed through a lateral approach using a corticocancellous bone graft. Especially indicated in a growing child as it allows for full growth of the hindfoot
- Subtalar fusion
- Triple arthrodesis

Upper limb cerebral palsy

- Surgery can achieve good cosmetic results but functional gains may be small (or non-existent)
- Thumb in palm deformity is common but difficult to manage. Correction entails release of the adductor pollicis and first dorsal interosseous muscle, fusion of the MCP joint and rerouting of EPL
- Finger flexion contractures can be released by selective myotendinous lengthening in the forearm

Examination corner

Paeds oral 1: CP clinical photograph

Describe the diagnosis, classification, definition and management for hip problems in CP.

Paeds oral 2: Video clip of gait analysis in a child with scissors gait

The prerequisites of normal gait (gage) are:

1. Stability in stance phase
2. Foot clearance in swing
3. Normal initial contact
4. Step length
5. Energy conservation

Paeds oral 3: Video clip of scissors gait

Describe the gait.

Scissoring during gait is caused by adductor spasticity. Legs are flexed slightly at the hips and knees, giving the appearance of crouching, with the knees and thighs hitting or crossing in a scissors-like movement. The typical features include:

- Rigidity and excessive adduction of the leg in swing phase
- Plantarflexion of the ankle
- Flexion at the knee
- Adduction and internal rotation at the hip
- Contractures of all spastic muscles
- Complicated assisting movements of the upper limbs when walking

Paeds oral 4

EXAMINER: What is cerebral palsy?

CANDIDATE: CP is a permanent and non-progressive motor disorder caused by brain damage before birth or during the first 2 years of life. The lesion is static but the clinical picture is not.

EXAMINER: How do you classify CP?

CANDIDATE: CP can be classified either anatomically or physiologically. Physiological categories include spastic, athetoid, ataxia and rigid or mixed varieties. The anatomical types include monoplegia, hemiplegia, diplegia and quadriplegia.

EXAMINER: What part of the brain is affected with athetoid CP?

CANDIDATE: The extrapyramidal system, basal ganglia.

Paeds oral 5

Exactly the same questions as oral 4 (I think it was the same examiner). However, the examiner did not like the definition of CP, the sticking point being 'non-progressive'. The examiner considered the disorder a progressive one. The candidate stuck to their guns and mentioned that although the lesion is static the clinical picture is not. There was a bit of to-ing and fro-ing between the candidate and examiner about this point. *(Pass)*

CANDIDATE: The examiner did not know the definition of CP particularly well and we spent a while arguing about whether the condition was progressive or non-progressive. It was a bit off-putting and was not the best way to start the oral.

Key advice here is stick to what you know but don't be argumentative!

Paeds oral 6: Clinical photograph of child with total body involvement CP

- Define CP
- Classify CP

Paeds oral 6

The candidate is shown an x-ray of a 10-year-old girl with a left hip subluxation, shallow acetabular index and pelvic tilt. At the top of the x-ray you can see the start of a scoliosis.

EXAMINER: A 10-year-old girl is transferred to your care after moving in to the area. She has got cerebral palsy and she is wheelchair bound. This is the x-ray taken of her pelvis on your first consultation.

CANDIDATE: Well, this is an AP pelvis x-ray of a 10-year-old girl. Both of the legs appear to be in adduction, however, the left leg is more adducted than the right. There is an obvious pelvic tilt, with the left side tilted up and there appears to be a scoliosis although we would need full spinal x-rays to ascertain this. With regards to the hips, the right hip looks to fairly well centered and covered. The left hip is starting to sublux. I would estimate the migration percentage to be 50% on this x-ray although I would need to formally measure it to be sure. The acetabulum also appears shallow on this side, with an acetabular index of approximately 35° following erosion of the superior lip of the acetabulum as the head has migrated out.

EXAMINER: How would you measure the acetabular index and the migration percentage?

CANDIDATE: I would draw Hilgenreiner's line through the triradiate cartilages and then Perkin's line, perpendicular to this at the lateral extent of the acetabulum. The acetabular index is calculated by drawing the angle between Hilgenreiner's line and the line from this line to the superolateral aspect of the acetabulum at Perkin's line.

The migration percentage is calculated by measuring what proportion of the femoral epiphysis lies lateral to Perkin's line in comparison to the entire diameter, ie, what percentage lies outside of the acetabulum.

EXAMINER: How would you use the migration percentage to guide your management?

CANDIDATE: The natural history of hip subluxation in cerebral palsy is well known. A migration percentage (MP) of <15% is normal. Anything <40% can be monitored for progression as per NICE guidance. Once the MP exceeds 40% then the hip is likely to proceed to complete dislocation if left unattended. Once the MP hits 60%, then urgent intervention is required to keep the hip in joint.

EXAMINER: So you would plan to intervene for any child with a MP of 40%?

CANDIDATE: No, every child is different and cerebral palsy can be an extremely complex condition to treat. Assuming that the child is fit enough for surgery and there are no contraindications, then I would discuss hip reconstruction at that point. If they were very opposed to surgery, then we could wait, monitoring the situation closely and intervene when the hip shows further signs of subluxation. I would, however, point out to the family, that the longer that we leave the hip to displace it is likely to undergo femoral head deformation and erode the acetabulum. I would also consider hip reconstruction earlier than 40% if there were other factors such as seating concerns, perineal hygiene, etc.

EXAMINER: For this child what would you recommend?

CANDIDATE: I would just like to clarify the GMFCS of the child, and know if there were any confounding factors which may affect the decision.

EXAMINER: She is GMFCS IV and there is nothing else that would affect your decision.

CANDIDATE: In that case, my treatment of choice would be bilateral hip reconstructions to level the pelvis. I would perform a shortening, varising, derotational osteotomy of the left femur using a proximal femoral locking plate. After this I would assess the congruency of the joint and proceed to a Dega acetabuloplasty as necessary. Assuming that the child was managing well after the first side, then I would go on to do a similar femoral osteotomy on the other leg to level the pelvis.

EXAMINER: You would operate on the right hip even though it is well contained?

CANDIDATE: Yes, if the parents consent. For children with GMFCS IV or V cerebral palsy the entire body is affected. They typically have asymmetrical tone issues making one leg worse than the other. In this situation, the more affected leg (left) is acting as an abduction splint for the right leg keeping it in joint. If you effectively treat the left leg you remove the splint and then the other hip migrates as the first side did. Also for children with scoliosis, if you only address one side, then one hip is varus, the other is valgus giving pelvic tilt and driving the scoliosis to progress.

I appreciate that this is very controversial. However, the birthday syndrome is well-known CP and we try very hard to avoid this. This was the basis for the multilevel CP surgery in walkers. If you only do one bit at a time, the disease process invariably leads to a new problem. This means that traditionally these children ended up coming in for surgery on an annual basis throughout their entire childhood, never really getting the full benefit of any of your interventions. It is much better for them to get the surgery all done in one go.

EXAMINER: How much varus would you put the hip in to?

CANDIDATE: In this child I would take it down to 100° as there is no walking potential and she is still relatively young. We know that the bone continues to remodel as long as they grow and so the best chance of avoiding a revision procedure is to deeply seat the hips. For a child who was walking then I would bring the hip to 120°.

EXAMINER: OK, let's move on.

The candidate is shown a picture of a child in a pushchair with legs adducted and internally rotated, hips and knees flexed in AFOs.

EXAMINER: This child is 3 years old and has been brought to your clinic for an initial assessment. He was originally diagnosed with CP after a traumatic birth and resulting brain injury. How would you assess him?

CANDIDATE: I would want to establish his level of function and potential, any learning difficulties or associated medical conditions from his family. I would do a thorough assessment to establish the pattern of involvement, type of CP and walking potential. I would also carefully assess his tone and the presence of any contractures.

EXAMINER: What patterns of involvement are you aware of for this condition?

CANDIDATE: The main patterns include hemiplegia (just one side of the body affected), diplegia (legs more affected than arms) and quadriplegia (where there is total body involvement).

EXAMINER: And what types are there?

CANDIDATE: The main types would be spastic and choreoathetotic. The spastic type just has constant increased tone, whereas the other type is much more difficult to treat. Patients experience great fluctuations in condition and it is more about writhing movements as opposed to contractures.

EXAMINER: And how would you assess walking potential?

CANDIDATE: I would use the GMFCS, which scores them I to V based on ambulatory capacity. I is virtually normal; II would probably require a walking aid for longer distances; III can walk short

distances with aids but would require a wheelchair for long distances; IV is wheelchair bound but has got head control; and V is wheelchair-bound requiring head support.

EXAMINER: What would you tell the family about his likely outcome and management if he was felt to be GMFCS IV spastic quadriplegia?

CANDIDATE: For a child who is GMFCS IV then he would not really walk unassisted and would be wheelchair bound life-long. These children can usually have some sort of walking in a special frame system with assistance and splintage as required.

As long as he grows then his musculoskeletal system will be constantly changing and we will have to monitor him and address problems as they arise. With regards to his hips we know that the majority of children with a GMFCS of IV will develop hip subluxation. The NICE guidance for these kids is that we should be performing an initial pelvic x-ray by the age of 3 and then screening the hips annually for any signs of subluxation.

EXAMINER: What categories of hip surgery are you aware of in CP?

CANDIDATE: The three main categories are: Preventive, reconstructive and salvage. Preventive surgery is largely soft-tissue work such as adductor and psoas tendon releases to attempt to slow the progression of hip subluxation. These are typically reserved for younger children or older children who are very mildly affected. Reconstruction is in the form of proximal femoral ± acetabular osteotomy to restore congruency of the hip joint. Salvage procedures are those when the hip is fully out and it is decided that a reconstruction cannot be performed. These would typically involve a pelvic support osteotomy or a proximal femoral excision.

OK.

The candidate is shown an AP x-ray of the pelvis of a 14-year-old child with both hips dislocated.

EXAMINER: This child is 14, has CP with a GMFCS of V. He has just moved in to your area and you are seeing him for the first time. This is the x-ray that you take on that initial consultation.

CANDIDATE: OK, this is an AP pelvis x-ray of a 14-year-old boy. Both hips appear to be completely dislocated with shallow acetabulae bilaterally. The femoral heads appear to be deformed bilaterally. There does not appear to be significant pelvic tilt or an obvious scoliosis.

EXAMINER: What do you think the appropriate management for this child would be?

CANDIDATE: It depends very much on his symptoms and medical condition. If he is wheelchair bound, his walking potential is irrelevant. If he has a bilateral symmetrical hip dislocation this rarely goes on to cause problems with scoliosis or other posture related issues. The fact that he is older as well means that his remodelling potential is not good. If he was completely pain free or only marginally affected then I would not suggest surgical intervention.

EXAMINER: It appears on discussion with the family that he is medically quite good. They feel that he is getting a significant amount of pain from his hips, and when you examine him he grimaces on hip movement.

CANDIDATE: Well in this case it is a discussion regarding reconstruction vs salvage procedures. I think that this looks like it is a long-standing dislocation with significant deformation of the femoral head. If we were to do a reconstruction it would likely lead to a painful joint. I believe that I would advise a salvage procedure. My preference would be a pelvic support osteotomy (PSO).

EXAMINER: Why would you choose a PSO over a proximal femoral excision?

CANDIDATE: The problem with a proximal femoral excision is that no matter how much you excise it tends to rise up afterwards and can cause pain. If we are doing a salvage then I would just like to do the operation once and be fairly sure that it will do the job.

EXAMINER: OK, let's move on.

Neurofibromatosis (Nf)

Definition

This is an autosomal dominant (AD) disorder of neural crest origin, which is often associated with neoplastic and skeletal abnormalities. There are two major types:

- Peripheral (Nf-1)
- Central (Nf-2) – Rare, 1/100 000. Eighth cranial nerve schwannomas are pathognomonic

Neurofibromatosis type 1 (peripheral neurofibromatosis or von Recklinghausen's disease)

- Incidence 1 : 4500
- AD gene mutation at chromosome 17: One in two are new mutations
- The manifestations vary but all carriers will have some clinical features (100% penetrance)
- Neurofibromas are Schwann cell tumours

Diagnosis

Two or more of the following criteria are diagnostic:

1. At least 6 café-au-lait spots (5 mm in children, 15 mm in adults)
2. More than two neurofibromas or one plexiform neurofibroma
3. Axillary, groin and base of neck freckles
4. Optic gliomaz
5. Two or more Lisch nodules (benign iris hamartomas)
6. Osseous lesions: Long bone cortex thinning with or without pseudoarthrosis, dystrophic scoliosis
7. Positive family history: First-degree relative with Nf-1

Musculoskeletal manifestations

- Scoliosis
- Extremity hypertrophy
- Pseudoarthrosis of long bones (tibia, ulna, humerus)
- Peripheral or spinal nerve tumour

Radiology

- Cortical bone defects: Usually caused by neurofibromatosis tissue irritating the periosteum
- Bone cyst formation: Caused by proliferation of tissue within the medullary canal
- Bowing of long bones
- Pseudoarthrosis of long bones

Tibial bowing

This occurs in 2% of Nf-1 and is always anterolateral. It is usually unilateral and the foot is spared. Fracture is common by age 3. Treatment is extremely challenging – Bracing, excision and bone grafting, Ilizarov callotasis and amputation all have their advocates. In contrast, posteromedial bowing is usually benign ±LLD.

Scoliosis

The spine is the most common site of skeletal involvement. There are two types: Dystrophic and non-dystrophic.

Features of **non-dystrophic** scoliosis are similar to those of idiopathic scoliosis.

Features of dystrophic scoliosis include:

- Posterior vertebral body scalloping (saccular dilatation of the dura)
- Enlarged neural foramina (dumb-bell tumour)
- Rib or transverse process pencilling
- Short, tight, sharply angulated curves that involve only a few vertebrae with severe apical rotation and wedging
- Soft-tissue masses
- Defective pedicles

Management

Non-dystrophic scoliosis is managed as idiopathic scoliosis. A dystrophic curve is relentlessly progressive even when growth has finished, cannot be controlled by bracing and requires early fusion. There can be associated kyphosis, and correction deformity carries a high risk of paraplegia, pseudoarthrosis and loss of correction after anterior and posterior fusion. Neurological involvement is common and may be caused by the deformity itself, soft-tissue mass, intraspinal tumour or dural ectasia (saccular dilatation of the dura). MRI is, therefore, mandatory preoperatively. It is not the condition itself that is the problem, but the extent of expression of the disease.

Examination corner

Paeds oral 1: Clinical photographs

- Severe scoliosis with cafè-au-lait spots (distinguishing feature)
- Thoracolumbar radiograph demonstrating short dystrophic curve

Paeds oral 2

- Neurofibromatosis diagnostic criteria
- Inheritance and chromosome defect

Paeds oral 2

A child who is 10 years old attends your fracture clinic with a displaced olecranon fracture after a very minor fall. Mum is extremely concerned sue to the fact that she has had four previous fractures, all of which occurred after minimal trauma. What would be your differential diagnosis?

In any child who attends with repeated injuries there must be a thought regarding the possibility of non-accidental injury. This would need to be excluded first and foremost. I would then consider underlying causes for recurrent fractures such as osteogenesis imperfect, metabolic bone problems such as vitamin D deficiency or just bad luck! We all know that injuries can occur with very minimal trauma if the mechanism is correct and we do see many children who have multiple injuries throughout childhood because they are very active.

However, the recurrent injuries with minimal trauma and particularly the olecranon fracture would make me suspicious of osteogenesis imperfecta (OI). That injury is particularly common in OI.

EXAMINER: What would you do to confirm a diagnosis of OI?

CANDIDATE: In the first instance I would take a history, including family history to establish any inheritance. Then I would examine the patient looking particularly for blue sclerae or dentinogenesis imperfecta. Ultimately, however, the diagnosis will come from genetic testing usually from a skin sample looking for the *COL1A1* and *COL1A2* genes. Mutations in these genes are responsible for the vast majority of cases of OI. I would also take some blood tests to exclude other causes such as vitamin D deficiency.

EXAMINER: How would you classify this child?

CANDIDATE: The Sillence classification is the most commonly used classification:

- Type I – Mildest and most common form, which may present with fractures in childhood. Typically fractures stop when they reach skeletal maturity. They have blue sclerae and may or may not have dentinogenesis imperfect
- Type II – Fatal in the perinatal period
- Type III – The most severe of the survivable forms of OI. They have white sclerae and typically have tooth involvement. They present with multiple fractures, progressive deformity of long bones and scoliosis. Their ambulatory potential is limited by their fragility and deformity, traditionally ending up in a wheelchair

- Type IV – Lies between types I and III. They will have multiple fractures but do not necessarily develop any deformity. They have white sclerae and may or may not have tooth involvement

This was the original classification but there have been many additions since this point. There have been nine subsets identified, but only type V is clinically distinct. These children develop ossification of the forearm interosseous membrane and get dislocation of the radial head. They are also prone to developing hypertrophic callus formation following minor injuries which can easily be confused with an osteosarcoma. The other four groups have different genetic mutations, histological appearances or are seen in specific populations.

For this child, I think it is most likely that she would be a type IV; however, if she had blue sclerae I would revise that to type I.

EXAMINER: How would you manage her olecranon fracture?

CANDIDATE: If it was displaced then I would treat it with a tension-band wire as I would with any other child. Children with OI will get fractures easily but they heal normally. The problem is that they heal with OI bone and so are just as prone to develop further fractures in future.

EXAMINER: What is the underlying defect in OI?

CANDIDATE: It is a defect in the production of type I collagen. That defect may be quantitative and qualitative. For instance in type I, the disease is quite mild because the defect in quantitative. The collagen produced is normal it is just that the levels produced are subnormal. Whereas in type III, there are reduced levels of collagen but they themselves are also abnormal giving limited function.

EXAMINER: Indeed. Let's move on.

The candidate is shown an x-ray of a lower limb in a child of 2 with severe bowing in the sagittal plane and multiple transverse sclerotic lines running parallel to the physis at either end of the long bones.

EXAMINER: Tell me what you see on this x-ray?

CANDIDATE: These are AP and lateral projections of the right lower limb of a small child, approximately 2 years of age. The most obvious abnormality is significant bowing of the femur and tibia in the sagittal plane. There does not appear to be any deformity in the coronal plane but this is difficult to say for sure due to the angulation. There are multiple transverse sclerotic lines related to each physis.

EXAMINER: Yes. What would your guess be regarding the diagnosis?

CANDIDATE: I would guess that it would be OI due to the severe bowing only in the sagittal plane, the deformity being mainly diaphyseal and the multiple sclerotic lines look like they have probably been caused by bisphosphonates.

EXAMINER: Yes they are bisphosphonate-related sclerosis. What is the role of bisphosphonate treatment in OI?

CANDIDATE: Bisphosphonates reduce osteoclastic activity and so prevent bone resorption. In children with OI where the bone density is reduced, preventing resorption can increase bone density and help to reduce the risk of fractures and progressive deformity.

EXAMINER: From those x-rays, what type of OI do you think that this child has got?

CANDIDATE: According to the Sillence classification I would put her as a type III. There is significant deformity in a young child putting her in the most severe survivable type.

EXAMINER: Indeed. What are the other types in that classification?

CANDIDATE: Type I is the mildest, with blue sclerae and occasional fractures in childhood. Type II is lethal in the perinatal period. Type III is the most severe type with progressive deformity, scoliosis and multiple fractures. Type IV is between types I and III with moderate numbers of fractures, but deformity is relatively unusual, they have white sclerae. I know that they have added several other types of OI, but I think that type V is the only one which is clinically distinct. They get ossification of the interosseous membrane of the forearm and hypertrophic callus formation.

EXAMINER: So, if we assume that this child has got type III OI with this deformity, what management would you advise?

CANDIDATE: Assuming that the child has got ambulatory potential then I would advise osteotomy and intramedullary rodding with a growing rod. This will provide them with straight limbs to support weight-bearing, reduce the risk of fractures and prevent further deformity.

EXAMINER: What age would you recommend the surgery?

CANDIDATE: As soon as they show ambulatory potential. We know that weight-bearing is good for increasing bone density and reducing fractures. Getting them up and mobile is extremely helpful, and correcting their mechanical axis helps reduce further injuries.

EXAMINER: What would you warn the family about before the surgery?

CANDIDATE: I would tell them that there is a bleeding problem in OI and that we may need to give a blood transfusion. I would warn them that there is a risk of fracture in doing the rodding itself, and in particular trying to splint them for healing before you manage to rod all four bones. I would tell the family that they will definitely require repeat surgery through life as they will grow and need the rods exchanging, hopefully without osteotomy. When they reach skeletal maturity then it is advisable to change the growing rods to a more rigid locked nail.

EXAMINER: Okay – Let's move on.

Pes cavus

Definition

A high-arched foot deformity where the longitudinal arch fails to flatten with weight-bearing. There is fixed plantar flexion of the forefoot relative to the hindfoot. Clawing toes are almost always present and the hindfoot is generally in varus.

Classification

Congenital

- Idiopathic
- Arthrogryposis
- Residual congenital talipes equinovarus

Acquired

- Neuromuscular disorders
- Muscular: Muscular dystrophies
- Peripheral nerves: hereditary motor sensory neuropathies (HMSN), polyneuritis
- Spinal cord: Spinal dysraphism, polio, spinal tumours, tethered cord, spina bifida
- Central: Cerebral palsy, Friedreich's ataxia, Charcot–Marie–Tooth disease
- Trauma: Compartment syndrome, crush injuries

Most idiopathic cases are simple cavus, whereas neurological cases are usually cavovarus. Two-thirds of cases have a neurological cause and most of these are HMSN. The cause of pes cavus is neurological until proven otherwise. Therefore, a thorough neurological examination is mandatory. Unilateral cases are less likely to have a neurological cause.

Coleman's lateral block test assesses hindfoot flexibility in the cavovarus foot. A flexible hindfoot corrects to neutral when a block is placed under the lateral aspect of the forefoot. This test relies on the 'tripod' of the first and fifth metatarsals and the calcaneus. Hindfoot mobility is an important factor in surgical planning.

Idiopathic pes cavus

This is a diagnosis of exclusion.

- Presents in adolescence/adult life
- Pressure effects on the deformed foot
- Painful calluses are present under prominent metatarsal heads
- ±Associated claw toes – Callosities over the dorsum of IP joints

Neuromuscular pes cavus

- Presents earlier with concern about the appearance of the foot, difficulty with shoe-fitting, excessive uneven shoewear (lateral aspect of the forefoot) and recurrent ankle giving way into inversion
- Loss of sensation can lead to ulcers over prominent bones (fifth metatarsal)

Radiology

Standing AP and true lateral views. The first metatarsal and talus long axes should be in line. The 'Meary' angle is measured between them.

Management

Non-operative management has met with limited success, and includes stretching programmes, arch supports, ankle foot orthoses and special shoes.

Operative options for supple deformities

Plantar release with or without tendon transfers

- Indicated in children <10 years old
- Fascia is cut while applying tension by dorsiflexion to the metatarsal joints
- Release of the abductor hallucis fascia is usually included. The neurovascular bundle is traced distally from beneath the abductor hallucis fascia and is thereby preserved
- Medial release may be indicated in fixed varus angulations. This involves releasing the medial structures such as the talonavicular joint capsule, the superficial deltoid ligament and possibly the long toe flexors
- Consider transfer of tibialis anterior into the midtarsal region for flexible inversion deformity

Jones procedure

- Performed for clawing of the hallux with associated weakness of tibialis anterior muscle
- This procedure involves transferring the extensor hallucis longus to the neck of the first metatarsal with arthrodesis of the IP joint
- Improves dorsiflexion and removes deforming forces at MTP joint
- The most common complication is non-union of the IP joint

Operative options for rigid deformities

Calcaneal osteotomy

- Dwyer medial opening wedge osteotomy
- Performed for hindfoot involvement
- Usually combined with plantar fascia release
- Translate the distal and posterior calcaneal fragment laterally

Beak triple arthrodesis

- Indicated in rigid deformity once growth has ceased
- The technique involves mortising the navicular into the head of the talus and depressing the navicular, cuboid and cuneiforms to improve the forefoot cavus deformity
- Lengthening of tendo-Achilles may be required
- This procedure is complex and technically demanding

Examination corner

Paeds oral

- Clinical photograph of pes cavus deformity
- Discussion of the Jones procedure

Congenital talipes equinovarus (CTEV)

Definition

A deformity in which the forefoot is in adduction and supination, and the hindfoot is in equinus and varus.

Epidemiology

- 1/1000 Caucasians, 3/1000 Polynesians
- Female : Male 2 : 1, bilateral 50%

Syndromic associations

- Arthrogryposis
- Streeter's syndrome (constriction bands)
- Möbius syndrome
- Larsen syndrome (autosomal recessive)

- Pierre Robin syndrome (X-linked recessive)
- Diastrophic dwarfism (autosomal recessive)
- Freeman–Sheldon syndrome (autosomal dominant)

Aetiology

Despite much research, the exact pathogenesis and aetiology remain obscure. Most infants who have clubfoot have no identifiable cause. In this idiopathic group the cause is considered to be multifactorial, including genetic and environmental components. Theories include:

- Primary germ plasm defect
- Mechanical moulding theory: Fallen out of favour in recent years
- Neurogenic theory: Histochemical abnormalities secondary to denervation changes in various muscle groups of the leg/foot
- Neurogenic imbalance deformity: Defect in nerve supply (the incidence of varus and equinovarus deformity in spina bifida is approximately 35%)
- Myogenic theory: Primary muscle defect. Predominance in type I muscle fibres, fibre type IIB deficiency
- Arrest of normal development of the growing limb bud
- Congenital constriction bands/rings
- Retracting fibrosis: Increased fibrous tissue in muscles and ligaments
- Viral infection aetiology (variably demonstrated through seasonality)
- Heritability is well established. A polygenic model is most likely, although an autosomal dominant gene with variable penetrance is also a possibility

Pathology

- **Mal-alignment** of the talocalcaneal, talonavicular and calcaneocuboid joints fixed by contracted joint capsules, ligaments and foot/ankle tendons
- **Tendon contractures** include tibialis anterior, extensor hallucis longus, Achilles tendon, tibialis posterior, plantar aponeurosis, abductor hallucis, flexor digitorum brevis, extensor digitorum longus
- **Ligament contractures** include spring, bifurcate, deltoid, calcaneofibular, talofibular and calcaneonavicular (spring) ligaments

Joint pathology

- Ankle and subtalar joints: Are in fixed equinus
- Hindfoot
  - Heel inverted (varus)
  - Talus lies in equinus with its head palpable at the sinus tarsi and marked medial angulation of the head and neck talus
  - Calcaneus is in equinus, varus and internal rotation
- Midfoot: Navicular and cuboid are medially displaced
- Forefoot: Inverted, adducted with forefoot supination relative to the hindfoot (forefoot varus)

Clinical assessment

- Examine the whole child to exclude associated abnormalities: Myelomeningocele, intraspinal tumour, diastematomyelia, polio, CP
- Also look for any associated developmental syndrome: Arthrogryposis, diastrophic dysplasia
- Look for other moulding conditions
- Examine the spine (neurological cause)
- Pulses: Usually present but vascular dysgenesis is possible. Dorsalis pedis artery may be absent
- Examine foot creases: Medial, plantar, posterior
- Affected limb may be shortened, calf muscle is atrophic, and foot is short compared to opposite side

Scoring systems

The Pirani scoring system is a widely adopted scoring system based on three midfoot and three hindfoot features. Each is considered on a range of normal (0), moderately abnormal (0.5) or severely abnormal (1). Maximum score is 6 points:

- Hindfoot contracture score (maximum score = 3 points)
  - Posterior crease
  - Equinus rigidity
  - Heel configuration
- Midfoot contracture score (maximum score = 3 points)
  - Medial crease
  - Talar head coverage
  - Curvature of the lateral border

The Clubfoot Assessment Protocol (CAP) is a more complicated system based on the degree of joint mobility.

Investigations

- Radiographs are not routinely taken in a newly presenting infant but may be of value if the case is resistant to therapy or other pathologies (e.g. congenital vertical talus) are suspected
- MRI scan of the spine (if a neurological cause is suspected)

Radiographic assessment

Weight-bearing AP view (kite's)

- On AP view the talocalcaneal (kite's) angle is normally 20–40° (<20° is seen in clubfoot; Figure 25.23)
- The first metatarsal talus angle is between the longitudinal axis of the first metatarsal and that of the talus and is normally 0–20° (a negative angle is seen in CTEV)

Forced dorsiflexion lateral view (Turco's)

- Turco's talocalcaneal angle is normally >35°. In CTEV, the angle is decreased and parallelism of calcaneus and talus is often seen

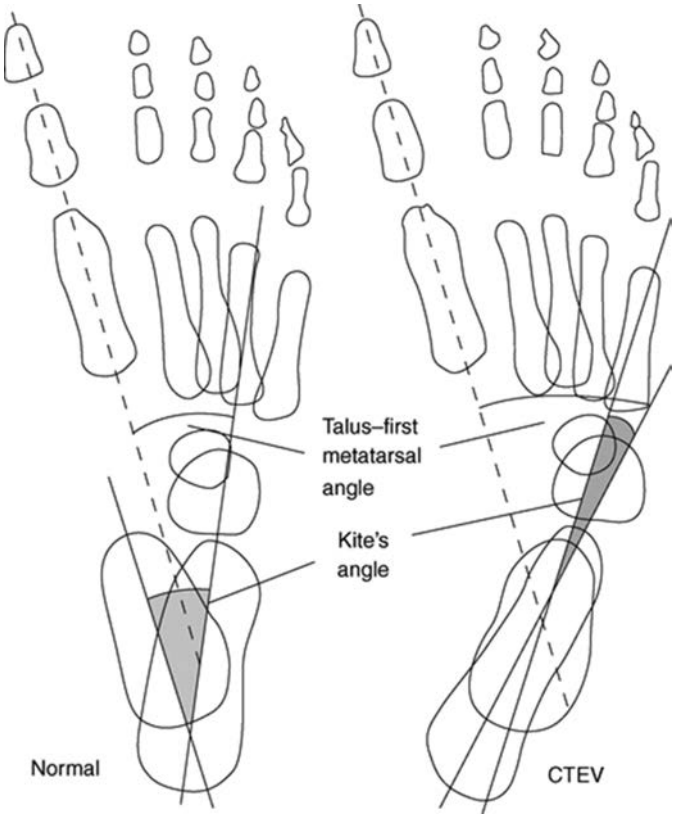

Figure 25.23 Radiographic evaluation of clubfoot

Management

The aim is to achieve a plantigrade, pliable, cosmetically acceptable, pain-free foot (Ponseti). Up until the mid 1990s, soft-tissue surgery was often undertaken at around 1 year of age. Strapping and various splints were employed. The Ponseti technique is long established but has taken time to gain wide acceptance From the mid 1990s, it has become the treatment of choice in all new cases. Surgery is now reserved for the very small proportion of cases that do not respond.

Ponseti casting technique

Serial casts are applied weekly for approximately the first 6 weeks of life. The sequence of correction is:

- Correction of cavus – Always the sole aim of the first cast
- Correction of adduction and heel varus
- Correction of equinus
- Percutaneous Achilles tenotomy is required in 90% of cases (under local or general anaesthetic). Where this is required, a further cast is applied for 3 weeks post-tenotomy. Post-reduction abduction splinting is required to maintain the position – For 23 hours per day for 3 months and then 12 hours per day until age 5 (or as close to this as can realistically be achieved). Traditionally, this involved Denis Browne boots and bar; however, Mitchell boots are gaining in popularity as they are well-tolerated by infants (and, therefore, their parents)

There is now very good evidence that most idiopathic CTEV can be successfully treated this way. Critics note that this procedure is not a non-surgical technique in the strictest sense – The Achilles' tenotomy is commonly required and later tibialis anterior tendon transfer is advocated in a significant proportion.

Surgery

Posteromedial release

- Incisions vary; however, the standard posteromedial release has been performed through a medial curvilinear incision, tracking the tibial neurovascular bundle from the calf behind the medial malleolus and into the foot. An alternative is the Cincinnati incision, which is in the axial plane hemicircumferentially round the hindfoot
- Whatever the incision, it is mandatory to identify and preserve the posterior tibial neurovascular bundle and, where appropriate, the sural nerve
- The release is generally undertaken on an '*a la carte*' basis (i.e. incorporating release of structures in a serial fashion until satisfactory correction has been achieved). It can include: Z-lengthening of tendo-Achilles
- Divide and lengthen tibialis posterior, flexor hallucis longus (FHL) and flexor digitorum longus (FDL)
- Capsulotomies: Ankle posteriorly, subtalar joint, calcaneocuboid joint
- Release plantar ligament, abductor hallucis, flexor digitorum brevis (FDB)
- Repair of tendons and insert K-wires into the talus and calcaneus to hold reduction

Residual deformity

Consider spinal cord MRI to rule out spinal lesion. Must exclude a neurological cause. Residual deformity may be either:

- Dynamic
- Fixed

If it is dynamic, SPLATT (split anterior tibialis transfer) or, indeed, whole tibialis anterior tendon transfer is considered if the patient is unable to evert their foot actively. A three-incision technique allows harvesting, proximal pull-through and distal reimplantation (classically into the ossified intermediate or lateral cuneiform). The foot is typically held in cast for 6 weeks postoperatively.

When the deformity is fixed, consider a repeat release if there is not too much scarring and the patient is <5 years old. This is difficult, and in general poor results are reported. If patients are older than 5 years they may need bony procedures to straighten the lateral border of the foot. The envelope for successful treatment with Ponseti casting has been extending, and it can be attempted even in late presenting or relapsing cases before surgery.

Bony surgery adduction deformity

- Calcaneocuboid fusion (Dillwyn–Evans procedure)
- Metatarsal osteotomy

These two procedures allow for lateral border shortening. Lengthening the medial border of the foot is technically more challenging.

Hindfoot deformity

- Varus heel
- Opening medial wedge or laterally based closing wedge osteotomy of the calcaneum
- Residual cavus and adductus
- Wedge tarsectomy
- Triple arthrodesis salvage procedure for stiff, painful foot in patients >12 years old

Ilizarov multiplanar external fixator

- Can be used as a primary procedure but is generally reserved for recurrent CTEV

Complications of surgery

- Overcorrection – A planovalgus overcorrected foot is a real problem that cannot be readily addressed and may require hindfoot fusion
- Infection, wound breakdown
- Stiffness/restricted range of movement
- AVN of the talus
- Scarring
- Rocker bottom deformity
- Residual deformity (undercorrection)

Examination corner

Paeds oral 1: Clinical photograph of bilateral clubfeet

- Discussion about causes: Arthrogryposis, dysraphism
- Association with DDH

Congenital vertical talus

This rare condition is an important exclusion when evaluating a paediatric foot deformity. Its principal features are:

- The navicular is dislocated dorsally off the talar head
- There is equinus of the hindfoot
- The cuboid is displaced dorsally
- The dorsal soft tissues are tight

The plantar convexity of the foot gives a characteristic 'rocker bottom' appearance.

A lateral forced plantarflexion radiograph gives a pathognomonic appearance of the forefoot remaining dorsal to and misaligned with the long axis of the talus.

Effective treatment is elusive. Serial casts to stretch dorsal structures must be supplemented by surgery. Traditionally this has involved single or two-stage releases; however, K-wire fixation of an aligned plantarflexed foot followed by further serial casting has recently been described and is a promising alternative approach.

Metatarsus adductus

- A common (1/1000 live births) neonatal presentation
- Involves adduction of forefoot at tarsometatarsal joints
- Uncertain aetiology – Thought to arise by uterine close-packing
- Historical association with DDH *now disproved*
- Hindfoot and midfoot are normal
- Bleck classified by degree of passive correctability
- Most spontaneously resolve; the efficacy of passive stretching is uncertain. Serial corrective casting for residual deformity beyond 6 months of age. Surgery rarely required

This condition needs to be differentiated from skewfoot:

- Also known as metatarsus varus, serpentine foot
- Very rare
- Medial forefoot, lateral translation of midfoot with heel in valgus
- Natural history uncertain – May resolve spontaneously, response to casting uncertain owing to multiplanar deformity

Calcaneovalgus

- Dorsiflexion positioning of the feet is a common (1/1000 live births) result of uterine close-packing
- May be associated with posteromedial bowing of tibia – Both are benign
- Foot generally spontaneously resolves over a period of weeks after birth
- Serial casting may be considered if fails to correct to beyond plantigrade
- It is important to differentiate from congenital vertical talus
  - Congenital vertical talus is less flexible than calcaneovalgus
  - Congenital vertical talus navicular has fixed dorsal dislocation on talus
  - A plantarflexion radiograph is diagnostic (see section on congenital vertical talus)

Curly toes

- Common disorder in children
- Frequently runs in families
- Often bilateral – Look for symmetrical deformity in the opposite foot
- There is malrotation of one or more toes along with a digit flexion deformity (contracture of FDL and FDB)
- Noticed when child walks

- Almost always asymptomatic in the child, but highly symptomatic for the parent!
- Child may occasionally complain of discomfort; their toe may catch when putting their socks on, callosity of the dorsum of the toe with footwear

Management

- Reassurance is the mainstay of management
- Surgical management involves FDL tenotomy at age 4 years
- Girdlestone procedure is a flexor to extensor tenotomy that has gone out of favour. It is technically difficult and often produces stiff toes in extension with a rotational element

Examination corner

Paeds oral 1: Clinical picture of child with curly toes

- Management is conservative and operative

Juvenile hallux valgus

This is a lateral deviation of the great toe with apex at the MTP joint (where associated with medial prominence, it is a bunion).

Epidemiology

- Variable prevalence – Approximately 25% of adolescents
- 80% female
- Strong family history – X-linked dominant/autosomal dominant with variable penetrance
- Metatarsus primus varus is a risk factor (first to second intermetatarsal ray angle of >10°)

Assessment

- Usually asymptomatic – Check skin, mobility of foot joints, shoewear
- Be aware of central causes of muscle imbalance (CP, spinal cord abnormality)
- Standing AP and lateral radiographs of foot
  - First to second intermetatarsal angle (<9° normal)
  - Hallux valgus angle (long axis of first MT and long axis of PP, <15° normal)
  - Distal metatarsal articular angle (DMAA = first metatarsal shaft and perpendicular to first metatarsal articular surface)
  - Lengths of metatarsals

Management

- In contrast to many conditions, early intervention has not been shown to positively influence outcome or reduce subsequent need for surgery
- Surgery may be indicated when *pain* cannot be accommodated by footwear adaptations and/or significant lifestyle modification
- Metatarsal osteotomies tend to deliver better results, with more proximal procedures, including cuneiform osteotomy, being considered where metatarsus primus varus is an issue

Pes planus

Pes planus (flat foot) describes depression of the medial longitudinal arch of the foot. There is associated valgus hindfoot and supination/abduction of the forefoot on the hindfoot.

- This is a very common presenting 'problem' to the paediatric orthopaedic clinic
- The crucial issue here is to differentiate between *flexible* and *rigid* pes planus. In a compliant child this is done by observing the feet from behind and asking them to stand on tiptoe. A flexible flat foot will then demonstrate an arch (the great toe MTP joint acts as a windlass, 'winding up' the plantar structures). At the same time the heel will correct from valgus to neutral or even into varus (demonstrating normal subtalar function). If compliance is an issue, the great toe can be manually wound into dorsiflexion and the same features observed
- It is important to check that the Achilles tendon is not too tight (and that dorsiflexion is possible beyond a plantigrade position – If not, perhaps the flat foot is an 'escape' mechanism to allow an equinus foot to weightbear)
- Examination should include attention to the rest of the foot and the lower limb
- Radiographs are not indicated unless there are particular indications (such as unexplained pain)

Flexible pes planus

- This is a normal variant in children and is almost universally present in infants. Spontaneous elevation of the longitudinal arch of the foot is the norm in the first 10 years of life
- Is a benign 'problem' (and arguably part of the normal range!) in older children and adults and is almost never responsible for symptoms
- There is evidence to suggest that expensive orthotic insoles are **not** indicated and are **not** of any value in modifying the natural history of this condition. A recent study suggests there is a psychological cost in their indiscriminate use in children
- Reassurance is the mainstay of treatment. When exercise-related fatigue pain is an issue, orthoses can be considered

Rigid pes planus

- Is a rare problem, but when it occurs is generally caused by a tarsal coalition (also known as peroneal spastic flat foot), but important differentials include

- Congenital vertical talus
- Juvenile idiopathic arthritis
- Trauma to subtalar joint
- Requires treatment when symptomatic. Prophylactic treatment to reduce abnormal joint loading is controversial

Tarsal coalition

Definition

An autosomal dominant disorder of primitive mesenchymal segmentation and differentiation leading to fusion of tarsal bones and rigid flat foot. The coalitions can develop from fibrous (syndesmosis), through cartilaginous (synchondrosis), to osseous (synostosis).

Epidemiology

- Prevalence between 2% and 6% depending on diagnostic method (clinical, radiographic)
- Bilateral in 50%
- Multiple coalitions occur in one in five cases
- Three in four are said to be asymptomatic

Calcaneonavicular (C-N) coalition

- Most common tarsal coalition, occurs in two-thirds of cases
- Rigid flat foot with contracture of the peroneal tendons, lateral foot pain and limited subtalar movement
- Radiographs: Blunting of the subtalar process, narrowing of the posterior subtalar joint, elongated anterior calcaneal process, talar beaking
- Calcaneonavicular bony bridges are seen on lateral radiographs with the classic anteater nose sign arising from the calcaneus
- Presents between 8 and 12 years of age when ossification of the coalition occurs

Talocalcaneal (T-C) coalition

- Coalition between the calcaneus and talus may occur in any of the three facets. Usually involves the middle facet of the subtalar joint
- T-C coalitions account for approximately one-third of tarsal coalitions
- Pain in the medial side of the subtalar joint, repeated ankle sprains. The patient is not able to take part in sports
- Contraction and spasm of the peroneal tendons with forced inversion, reduced subtalar movements
- T-C coalitions tend to ossify at 12–15 years of age
- T-C coalitions may be difficult to see on radiographs, which can often be normal. May see irregularity of the talus and calcaneus joint surfaces and occasionally the C sign of Lateur may be present
- Harris view – Axial radiograph to visualize posterior and middle facet
- Coalitions between calcaneum and cuboid and navicular and cuboid are rare

Clinical features

- Tenderness at the location of the coalition
- Antalgic gait
- Valgus hindfoot which, on attempted correction, induces peroneal spasm (peroneal spastic flat foot) and discomfort
- Calf pain
- Limited subtalar motion and peroneal tendon shortening
- Increased laxity of the ankle joint

Investigations

- AP, lateral and oblique at 45° hindfoot radiographs, Harris view
- CT or MRI scan to rule out subtalar coalition. Coronal cuts are helpful in evaluating talocalcaneal bony bridges while transverse cuts are used for calcaneonavicular bars

Management

Conservative

- In children with mild symptoms
- Natural history unclear but only 25% are thought to develop pain
- Supportive insoles or below-knee plaster of Paris (POP) cast can be used

However, many children who present with pain have evidence of degenerative changes in the hindfoot. Opinion is divided concerning timing and indications for surgery. Some consider activity modification, NSAIDs, immobilization in cast and joint injection to be sensible first steps with surgery reserved for persistent cases. Others opt for surgery earlier.

Surgery options

Calcaneonavicular coalition

Ollier's approach: Wide bar excision such that one should be able to see across to soft tissues on the medial side of the foot through the excised bar. To prevent recurrence, all cartilage must be removed from both the calcaneus and navicular. Interposition of extensor digitorum brevis (EDB) into the defect reduces the risk of refusion

Talocalcaneal coalition

Medial limb Cincinnati incision: flexor hallucis longus (FHL) lies just plantar to the sustentaculum tali and the tendon can be used for orientation to the coalition anomaly. The FHL tendon sheath is incised, and the tendon is retracted inferiorly. The sustentaculum tali and its associated coalition are identified. Once the coalition is resected,

interposition of one half of the FHL tendon will decrease the chance of recurrence

Subtalar arthrodesis: Performed when >50% of the middle facet is involved, with recurrence (failed resection), or if significant degenerative changes in the tarsal joints exist (talar beaking is not considered a degenerative change)

Triple arthrodesis may be indicated for severe symptoms with significant degenerative changes

Arthrogrypotic syndromes

Definition

Congenital non-progressive limitation of joint movement owing to soft-tissue contractures affecting two or more joints. Many different subgroups exist but it is easier to group them into three major categories:

- Arthrogryposis multiplex congenita (classic form)
- In association with major neurogenic or myopathic dysfunction
- In association with other major anomalies and specific syndromes

Aetiology

- Exact cause is unknown; multifactorial, reflecting such a heterogeneous group, but factors likely to limit fetal movement in utero appear important, together with, possibly, intrauterine viral infection, teratogenic or metabolic causes

Arthrogryposis multiplex congenita (amyoplasia)

- Non-progressive congenital disorder with multiple congenitally rigid joints
- It is a sporadic disorder with no known hereditary pattern
- Incidence is variably quoted to be from 1 in 3000 to 1 in 50 000
- Joints develop normally in arthrogryposis multiplex congenita, but periarticular soft-tissue structures become fibrotic, leading to development of an incomplete fibrous ankylosis and muscle atrophy
- Associated with a decrease in anterior horn cells and other neural elements of the spinal cord
- Sensory function is maintained whilst motor function is lost

Clinical features

- Normal facies and normal intelligence. Head and neck movements are normal
- Skin creases are absent and there is tense, shiny skin with underlying muscle wasting
- Shapeless featureless cylindrical limbs
- Upper limbs: Adduction and internal rotation of the shoulder, extension of the elbow, flexion and ulnar deviation of the wrist, fingers flexed at the MCP joints and IP joints, thumb adducted (similar to Erb's palsy deformity)
- Lower limbs: Flexion, abduction and external rotation hip, teratological hip dislocations, knee contractures (flexion), equinovarus (clubfoot), vertical talus
- Spine: C-shaped neuromuscular scoliosis (33%)

Investigations

To establish the underlying diagnosis. Consider paediatrician, neurologist and clinical geneticist input.

- Nerve conduction studies, enzyme studies, muscle biopsy, chromosome analysis, collagen biochemistry, head scan (CT/MRI) and radiographs of the whole spine, anteroposterior pelvis and the involved limb

Management

Physiotherapy is an absolutely essential part of the management plan. The aim of management is to obtain maximum function, independent mobility and self-care.

- **Elbow:** Passive manipulation, serial casts, tendon transfer, posterior elbow capsulotomy, possibly osteotomies after the age of 4 years. One elbow should be left in extension for use of crutches when walking and the other in flexion for feeding
- **Wrist:** Flexion deformity common. FCU to extensor carpi radialis transfer and volar capsulotomy may be beneficial
- **Hand:** Release of thumb and palmar deformity by adductor pollicis lengthening; MCP joint fusion can be considered
- **Hips:** Two-thirds have hip dysplasia or dislocation. Surgery is nearly always associated with stiffness, which can be more disabling than a dislocated, but mobile, hip. In general, unilateral dislocation is managed surgically because of concerns over LLD and asymmetry. Management of bilateral hip dislocation is controversial and there are two schools of thought: Either medial open reduction without risking disabling stiffness or leave it alone. For a stiff, located hip following surgery, excision of the upper end of the femur may be required
- **Knees:** Both fixed flexion and fixed extension are common, the former being most common with associated pterygium possible. Fixed extension responds well to stretching and serial casting, although occasionally quadricepsplasty is required. Fixed flexion is difficult to manage and often requires extensive posterior soft-tissue release with prolonged splintage. Femoral osteotomy with or without shortening (avoids stretching the neurovascular bundle) is indicated for recurrence towards the end of maturity
- **Foot:** The most common deformity is equinovarus; more rarely, vertical talus is seen. Severe equinovarus is traditionally managed with extended soft-tissue release; however, serial casting has been a successful alternative. Recurrences may need talectomy. Congenital vertical talus

does not prevent the patient from standing and walking, but it may cause problems with shoewear. Surgical correction is only carried out if absolutely necessary
- **Scoliosis:** Early surgical intervention is recommended – Either posterior spinal fusion alone or combined with anterior spinal fusion

The aim is to have finished surgery by the time the patient is 7 years old, if possible.

Examination corner

Paeds oral 1: Clinical photograph of a child with congenital arthrogryposis multiplex
- Spot diagnosis
- Discussion about hip dislocation and other associated syndromes

Bone and joint infection

Epidemiology

- Common organisms remain *Staphylococcus aureus,* coagulase-negative staphylococci, group A β-haemolytic streptococci, *Streptococcus pneumoniae* and group B streptococci. The advent of vaccination has reduced *Haemophilus influenzae* infection dramatically
- Usually occurs in under 10s
- The possibility of contiguous septic arthritis and osteomyelitis must be considered – The blood supply to the epiphysis in those under 18 months old predisposes to this

Aetiopathogenesis

- Requires the presence of a virulent organism in sufficient numbers to overwhelm (possibly suppressed or sometimes immature) host defences
- Metaphyseal predilection for osteomyelitis may reflect acute angle of vascular hoops described by Hobo
- Localized trauma has a proven association
- Varicella predisposes to bacterial infection by lowering host immunity

Clinical presentation

- Fever, malaise, anorexia and pseudoparalysis are common presenting features
- Antibiotics may blunt the symptoms
- Bone pain/joint pain + fever = osteomyelitis/septic arthritis until proven otherwise
- Differential diagnosis – Osteomyelitis
- Neoplasm (in leukaemia 30% have bone pain)
- Trauma (but not normally with raised ESR)
- Eosinophilic granuloma
- Bone infarction
- Differential diagnosis – Septic arthritis
- Transient synovitis, irritable hip
- Juvenile idiopathic arthritis

Kocher's criteria

- Offer good discrimination for septic arthritis of hip
- Fever + non-weight-bearing + ESR >40 + WCC >12 = septic hip (where all 4 have a positive predictive value (PPV) of 93%, 3/4 have a PPV of 73% and 2/4 have a PPV of 35%)

Investigations

- ESR, CRP, FBC, blood cultures (especially if pyrexial)
- Plain radiographs (accepting bony changes may take 7 days)
- Tc–99m bone scan – Sensitivity 89%, specificity 94%
- MRI – Sensitivity 88–100%, specificity 75–100%
- (CT – Occasionally to assess bony destruction)
- USS – Highly operator-dependent – Used to assess hip effusion
- Aspiration – To obtain fluid for culture advocated where possible by some; *however,* risk of false-negative aspirate (thick fluid or inaccurate needle placement) leads to elective exploration by others

Synovial aspirate

- Normal WCC <200/ml
- Juvenile idiopathic arthritis WCC 15–80 000/ml
- Sepsis >50 000/ml

Septic arthritis

- Management is not universally similar in all centres, and you could not be reasonably criticized for answering that where clinically there is a high index of suspicion of a septic hip it is a *surgical emergency* requiring formal open lavage. This would usually be via a Smith–Peterson approach with a small anterior capsulotomy to gain access to the joint and with samples sent to microbiology
- In Glasgow, practice differs with aspiration (repeated if necessary) being the mainstay of treatment with very comparable results
- The important principle is that pus under pressure in the hip joint is highly destructive to the joint surfaces and needs to be removed as soon as possible
- Empirical IV antibiotic therapy should be started as soon as samples have been sent
- The duration of IV therapy has been the subject of discussion and it is now generally accepted that it does not need to be continued for 6 weeks but rather can be converted to oral therapy when a good response is reflected in improving clinical picture and inflammatory markers

Osteomyelitis

- Acute – Without abscess formation
  - Can be managed expectantly with antibiotics alone and monitor response
- Acute – With abscess formation (subperiosteal, etc)
  - Abscess may be at 3–5 days following infection
  - Requires surgical drainage
- Chronic – Poorly defined transition from acute –c. >3 weeks
  - Abscess within bone generally indicates a need for surgical drainage
  - Samples for microbiology and histopathology should be sent
  - When there is advanced chronic osteomyelitis (with sequestrum, surrounding involucrum and cloacae draining pus to the surface), the surgical decision should be influenced by the extent of involucrum – Sequestrectomy may be deferred until there is structurally sound involucrum in selected cases. Recovery often exceeds expectation, and any consideration of amputation should be deferred in a paediatric population
- Management with antibiotics continues with monitoring of response clinically and with lab markers of infection (ESR, CRP and FBC). Conversion to oral antibiotics can be considered when sustained improvement is noted

Discitis

- The most common spinal infection in children
- May present with refusal to ambulate but normal lower extremity examination
- ESR and CRP usually elevated
- Bone scan is best investigation

Neonates

- Immune immaturity renders increased susceptibility. There are two groups
  - Neonatal unit infants with indwelling lines get staphylococcus or Gram-negative infections, with multiple sites in 40% and are systemically unwell
  - 'Normal' infants out of hospital get group B streptococcus infections at a single site
- Metaphyseo-epiphyseal infections occur and may be contiguous with a septic joint
- Poor immune response means laboratory indicators are of limited value – WCC may be normal; ESR is usually raised
- 50% have positive blood cultures
- Bone scan of value
- Initial broad antibiotic cover is required
- A septic hip requires emergency surgical treatment

Chronic recurrent multifocal osteomyelitis

- Inflammatory bone disease >6 months with exacerbations and remissions
- Lack demonstrable organism
- 70% females, 90% multifocal
- Lytic metaphyseal lesion, raised ESR/CRP but normal WCC, mimics osteomyelitis at onset
- Commonly tibia and femur
- Differential: Ewing's sarcoma, eosinophilic granuloma, leukaemia
- 90% show response to anti-inflammatory drugs

Brodie's abscess

This is a chronic localized bone abscess. The lesion is typically single and located near the metaphysis of the bone. Preferred sites are proximal femur, proximal and distal tibia.

Clinical features

Subacute cases present with fever, pain and periosteal elevation. Chronic cases often present without pyrexia with long-standing, dull pain. There may be a limp, often slight swelling, muscle wasting and localized tenderness. The patient has few signs or symptoms to suggest an infection. The white cell count is often normal but the ESR may be raised.

Pathology

Typically a well-defined cavity in cancellous bone containing seropurulent fluid (occasionally pus). The cavity is lined by granulation tissue containing a mixture of acute and chronic inflammatory cells. Typically no organisms are found but, if one is present, it is usually a *Staphylococcus aureus* (60%).

Radiology

Well circumscribed, round or oval cavity 1–2 cm in diameter, most often in the tibia or femoral metaphysis. Sometimes the cavity is surrounded by a halo of sclerosis (classic Brodie's abscess). Metaphyseal lesions do not cause a periosteal reaction, whereas diaphyseal lesions may be associated with cortical thickening and periosteal new bone formation. A bone scan reveals markedly increased activity.

Differential diagnosis

- Osteoid osteoma
- Ewing's sarcoma
- Langerhans cell histiocytosis
- Aneurysmal bone cyst
- Pigmented villonodular synovitis (PVNS)
- Giant cell tumour
- Non-ossifying fibroma

Management

Brodie's abscess is usually managed with biopsy and surgical debridement followed by intravenous antibiotics.

Examination corner

Paeds oral 1: Brodie's abscess

- Diagnosis
- Differential diagnosis
- Further tests
- Management
- Options
- Surgical management, including types of incision and techniques
- The examiner wanted to know what kind of material is seen at curettage

Paeds oral 2: Radiograph of classic Brodie's abscess in the distal metaphysis of the radius

EXAMINER: These are radiographs of a young boy who presents with a several-week history of localized pain and swelling in his wrist. What do you think of his x-rays?

CANDIDATE: There is a well-defined cavity in the distal metaphysis of the radius. There is no periosteal reaction but a halo of sclerosis surrounding the lesion. The radiograph is suspicious of a Brodie's abscess.

EXAMINER: These are his MRI scans, which did confirm the impression of a Brodie's abscess. How are you going to treat him?

CANDIDATE: I would treat him conservatively initially with IV antibiotics and see if it settles down. The condition may resolve in time[f]. If necessary, with recurrent flare-ups, the abscess should be curetted out.

EXAMINER: What you find is that the wall of the cavity becomes sclerotic and lined by a thick membrane and cannot be easily penetrated by antibiotics. The patient is then prone to recurrent flare-ups of pain. You need to go in and curette the cavity out.

Angular lower limb deformity

Natural history

Physiological genu varum (bowed legs) gradually improves as the child starts to stand and walk. By the age of 18–24 months the legs are straight. By 2–3 years of age, lower limbs have evolved naturally to genu valgum (knock-knees). There is a gradual transition to physiological valgus by 7 years of age, by which time the leg has assumed a normal adult value of 7–8° valgus.

Genu valgum

Aetiology

Pathological causes of genu valgum include:

- Skeletal dysplasias: Multiple epiphyseal dysplasia, Morquio's syndrome, Ollier's disease
- Primary tibia valga
- Previous trauma: Asymmetrical growth arrest following fracture
- Metabolic bone disease, particularly renal osteodystrophy
- Iliotibial band neuromuscular contracture: Polio
- Infection: Causing asymmetrical growth arrest
- Tumours: Osteochondromas
- Congenital: Congenital absence of the fibula

Unilateral genu valgum is strongly suggestive of pathological genu valgum.

Clinical examination

- Is it unilatersal or bilateral? If bilateral, is it symmetrical or asymmetrical?
- Measure standing and sitting height to rule out skeletal dysplasia
- Measure the distance between the medial malleoli with knees touching. The mechanical axis can be assessed by dropping a plumb line' from the centre of the femoral head to the centre of the ankle – In a teenager this line should bisect the knee
- Determine the site of valgus angulation, the degree of tibial torsion (tibia valga is associated with excessive lateral tibiofibular torsion) and carry out Ober's test to rule out iliotibial band contracture

If genu valgum is marked, the symptoms include:

- In-toeing to shift weight over the second metatarsal so the centre of gravity falls in the centre foot
- Lateral subluxation of the patella
- Fatigue

Management

- 95% resolve spontaneously
- Consider surgery if the intermalleolar distance (between medial malleoli when the child is standing with knees touching) is >10 cm or >15–20° valgus at age 10 years
- Hemiepiphysiodesis of the distal femur and/or proximal tibial growth plate by either stapling, '8' plates or fusing the medial-side physis
- If skeletally mature, carry out a tibial or femoral osteotomy

Valgus deformity in adults is usually caused by:

- Sequel to childhood deformity
- Secondary to osteoarthritis or rheumatoid arthritis
- Ligament injury
- Malunited fracture
- Paget's disease

Examination corner

Paeds oral 1: Clinical picture of a child with genu valgum within the physiological limit

- Discussion of whether this is normal or abnormal

[f] Incorrect answer. Some older textbooks suggest that the lesion in certain circumstances may be managed with oral antibiotics alone. Most orthopaedic surgeons would disagree with this and fail you if you mention it as primary management of the condition.

- How will you assess this child in the outpatient clinic?
- Management

Genu varum

Aetiology

Normal in children under 2 years old. Pathological causes include:

- Metabolic bone disease
- Vitamin D resistant rickets
- Vitamin D deficiency
- Hypophosphatasia
- Asymmetrical growth arrest or retardation
- Trauma
- Infection
- Blount's disease
- Skeletal dysplasia: Metaphyseal chondrodysplasia, achondroplasia, osteogenesis imperfecta
- Neuromuscular: Polio, spina bifida
- Congenital: Deficient tibia with relatively long fibula

Clinical examination

- Document height, weight and percentiles for age
- Examine pelvis, knees and feet
- Shortened limb relative to trunk may suggest dwarfing condition
- Document general appearance during standing and gait
- Assess deformity: Is there gradual bowing or abrupt angulation?
- Gait is characterized by painless varus thrust in stance phase
- Measure the intercondylar distance: The distance between the knees when the ankles are held together
- Internal tibial torsion: This is measured by the angular difference between the transmalleolar axis and the bicondylar axis of the knee
- Thigh-foot angle: This is measured with the child in the prone position and knee flexed 90°, by observing the angle of the foot and the thigh

Indications for radiographs

- Deformity outside the normal range
- Deformity – Unilateral or asymmetrical
- Child over 3 years
- Positive family history (bone dysplasia, syndromes or renal rickets)
- Short stature or disproportion (bone dysplasia or endocrine disturbance)

Radiographic evaluation

- **Tibiofemoral angle:** Measures varus severity
- **Drennan's metaphyseal–diaphyseal angle:** Formed by intersection of a line through the transverse plane of the proximal tibial epiphysis with a line through the transverse plane of the metaphysis. Normal is $<11°$

Tibia vara (Blount's disease)

Tibia vara is a growth disorder of the proximal tibial physis caused by repetitive trauma to the posteromedial proximal tibial physis from early walking on a knee with physiological varus alignment. The aetiology of late-onset tibia vara is unknown. The infantile form is commonly bilateral and is associated with internal tibial torsion. The late onset or adolescent form presents as a painful, unilateral, slowly progressive varus deformity of the knee:

- Unilateral or bilateral, asymmetrical
- Often a varus thrust
- The metaphyseal-diaphyseal angle (Drennan's) $>11°$
- Upper tibial metaphysis is fragmented
- Upper tibial epiphysis slopes medially
- Upper tibial physis is widened laterally

Langenskiöld's classification of tibia vara

Type I: Medial metaphyseal beaking
Type II: Cartilage-filled depression
Type III: Ossification at the inferomedial corner epiphysis
Type IV: Epiphyseal ossification filling the metaphyseal depression
Type V: Double epiphyseal plate
Type VI: Medial physeal closure

Management

- Bracing for Langenskiöld's stage I and II disease in patients <3 years old
- Surgery for failed orthotic management and Langenskiöld's stages III–V
- Initial operation is proximal tibial valgus osteotomy distal to the tibial tubercle to avoid damaging the tibial apophysis
- If growth arrest has occurred, a physeal procedure also needs to be performed, either stapling or epiphysiodesis of the lateral tibial physis (selective closure of half of the growth plate to allow the contralateral portion of the physis to correct with growth) or, rarely, a partial physeal bridge resection with interposition fat. For late-onset tibia vara, carry out a tibial osteotomy below the growth plate with correction of the tibiofemoral angle

Examination corner

Paeds oral 1

- Spot diagnosis – Unilateral Blount's disease
- Differential diagnosis
- Causes
- Management

Paeds oral 2: Clinical photograph of a young child with mild bilateral genu varum

CANDIDATE: This is a clinical photograph that demonstrates mild bilateral genu varum. The most common cause of this is a benign normal variant in which the knee will evolve into genu valgum and then a normal adult valgus angle will develop in time.

EXAMINER: This child comes to clinic with her mother. The mother is worried about the appearance of the knees. How will you reassure her?

CANDIDATE: I would say that it is a very common condition, which is seen often in the clinic. In the vast majority of cases, it is just a feature of normal growth and development of the leg, and corrects as the child grows. I would take a full history and perform an examination to reassure the mother. I would want to exclude any pathological cause for the genu varum.

EXAMINER: (*interrupts with*) What pathological causes are you thinking about?

CANDIDATE: Conditions such as Blount's disease.

EXAMINER: (*interrupting*) Come on, is the child black? How common is Blount's disease in a young white girl with normal build?

CANDIDATE: Not common. (*Regains composure.*) I would want to exclude rickets, a skeletal dysplasia or a syndrome. Other causes could include infection, trauma, tumours, but these are usually unilateral.

Next picture: clinical photograph of an obese girl, approximately 15 years old, with severe unilateral genu varum with gigantism of the limb.

CANDIDATE: (*big influx of breath as answer is prepared*) This is a clinical photograph of a young girl, which demonstrates a severe genu varum of the left leg and gigantism of this leg. The situation is grossly abnormal and I would be worried about a pathological cause for the condition.

EXAMINER: Can you name any causes that can give the limb this appearance?

CANDIDATE: The causes of gigantism are an arteriovenous (AV) malformation, nerve tumours, neurofibromatosis, lymphoedema of the leg, a neoplasm or idiopathic.

(Pass)

CANDIDATE: I did not sound very convincing to the examiners in how I would have reassured the mother in the first picture or, more accurately, the examiners seemed unimpressed with my answer. This was a failure to deliver the facts rather than any glaring omission. The examiner was making life just a little bit too uncomfortable. In the second picture I had never seen anything like it before. Luckily I managed to say something sensible in my answer. The examiner was stony-faced and gave no feedback on my answer. Even a simple topic like genu varum can be made difficult by an examiner's line of questioning.

I think you need to be aware of this classification for the exam but without necessarily knowing specifics. You would, however, be expected to spot the diagnosis on a clinical photograph or radiograph (an overweight child with severe genu varum). Recognition of the radiographic features of the disease is important.

Congenital pseudoarthrosis of the tibia

This is a rare condition, with an incidence of 1/250 000. It is almost always unilateral. This is one of the most challenging conditions to manage in orthopaedics. The condition may not be obvious at birth. It presents with a spectrum of disorders, ranging from anterolateral bowing to frank pseudoarthrosis or pathological fracture with an apex deformity.

Classifications

Boyd[29]

Boyd's is the best known and most complete classification of the disease and the most appropriate one for use in clinical practice. Cystic lesions tend to do better, whilst the dysplastic type is less favourable.

Type I: Born with anterior bowing and tibial defect

Type II: Born with anterior bowing and an **hourglass constriction. Spontaneous fractures occur before 2 years age.** Often associated with neurofibromatosis

Type III: Those developing **bone cysts** often at the junction of upper and lower thirds. Anterior bowing may proceed or follow a fracture

Type IV: Those originating in a sclerotic segment of tibia without narrowing or fracture. The medullary canal is obliterated

Type V: Those who also have **a dysplastic fibula** develop pseudoarthrosis later

Type VI: Those with **an interosseous neurofibroma** or schwannoma (very rare)

Management

Non-operative management includes prophylactic total contact bracing to try to prevent fractures or control developing ones. Surgical management options include:

- Intramedullary rodding and bone grafting
- Vascularized fibular graft
- Ilizarov frame
- Syme's amputation

ORIF includes excision of fibrous tissue at the pseudoarthrosis site, removal of sclerotic bone and correction of anterolateral angulation. Tibial pseudoarthrosis is a very challenging condition and optimal treatment is the subject of ongoing controversy.

Complications

- Re-fracture or non-union
- Stiffness of ankle and subtalar joints

- Limb shortening
- Progressive anterior angulation of tibia
- Infection
- Repeated operations
- Soft-tissue scarring

Fibular hemimelia

Definition

This condition consists of a spectrum of anomalies from mild fibular shortening to total absence of the fibula. It is the most common long bone congenital deficiency.

Classifications

Achterman and Kalamchi[30]

Type I: Hypoplastic fibula

Type Ia: Proximal fibular epiphysis is more distal than normal, and distal fibular epiphysis is more proximal than normal. There may be a ball and socket ankle joint

Type Ib: More severe deficiency with at least 30–50% of the fibula missing and no distal support to the ankle

Type II: Complete absence of the fibula. Angular deformities of the tibia are common and are associated with severe foot and ankle problems (tarsal coalition, lateral ray deficiencies)

Coventry and Johnson[31] (three types)

Based on the degree of fibular dysplasia and whether the deformity is unilateral or bilateral:

Type I: Shortened fibula – Partial absence of upper portion

Type Ia: Normal foot

Type Ib: Equinovalgus foot

Type II: Complete absence of the fibula and foot deformities, etc

Type III:Bilateral

Clinical features

- LLD always present
- Anteromedial bowing with a dimple over the apex of the tibia
- Absence of lateral rays of the foot
- Equinovarus of the foot
- Stiff hindfoot with tarsal coalition, particularly talus and calcaneus
- Ball and socket ankle joint
- Flexion contracture of the knee
- Ankle and knee instability
- Femoral shortening (if associated with PFFD)

Management

Management is difficult and complex. Generally, the following principles apply in deciding on reconstruction vs amputation:

- **Mild deformity:** Reconstruct
- **Severe:** Amputate
- **Intermediate:** Obtain a second opinion

Reconstruction options include:

- Posterolateral release to correct equinovalgus deformity of the foot
- Limb lengthening is indicated if the foot and ankle are relatively normal

Syme's amputation is ablation of the foot by ankle disarticulation, producing a sturdy end-bearing stump that can be walked on.

Tibial hemimelia

Definition

This condition represents a spectrum of deformities ranging from total absence of the tibia to mild hypoplasia. It is often associated with PFFD or a congenital short femur. This is the only skeletal deficiency with a Mendelian pattern of inheritance. Both autosomal dominant and recessive patterns are described. Thirty per cent of cases are bilateral.

Classifications

Kalamchi (three types)

Based on radiographs, clinical appearance and functioning of the quadriceps mechanism:

Type I: Complete absence

Type II: Absence of the distal half of the tibia

Type III:Hypoplastic

Jones classification

Classified into four types on the early radiographic appearance:

Type I: Absent tibia

Type II: Proximal tibia present

Type III: Distal tibia present

Type IV: Tibia shortened, proximal migration of the fibula and diastasis. Distal tibiofibular syndesmosis

Clinical features

- The involved leg is short with a varus or calcaneovarus foot
- There is often a skin dimple over the front of the leg
- Quadriceps muscle is often underdeveloped or absent; there are various degrees of fixed flexion at the knee

Management

Reconstruction options

These include:

- Distal fibulotalar arthrodesis or calcaneal-fibula fusion to stabilise the hindfoot
- Tibiofibular synostosis (fusion)

- Tibial lengthening with epiphysiodesis of the ipsilateral distal fibula and contralateral limb

Amputation

Through-knee amputation indicated in:

- Severe deformity
- If there is marked fixed flexion deformity of the knee
- Knee is unstable
- Tibia is completely absent

Avoid above- or below-diaphyseal amputations because of associated problems with overgrowth of the residual diaphysis.

Popliteal cyst

The common site is medial, originating in the gastrocnemius-semimembranosus bursa just below the popliteal crease. The cyst arises from the synovial sheaths of the surrounding tendons and contains clear viscous fluid. In contrast to those in adults, they do not communicate with the knee joint and are not associated with intra-articular pathology:

- Presents at 5–8 years of age as a painless, firm, rubbery swelling behind the knee
- Usually asymptomatic and of insidious onset; occasionally can cause vague mild local discomfort
- The mass is fluctuant and transilluminates. The rest of the knee examination is normal
- Ultrasound and CT scan demonstrate the lesion well, although this is not usually necessary unless the diagnosis is in doubt

Management

Reassure the child's parents that the lesion is benign; the vast majority will resolve in time and the lesion should be left alone. There are very few indications for surgery:

- When the diagnosis is in doubt
- Severe pain (check for other, more obvious, causes)
- Sinister cause

There can occasionally be great parental concern about this swelling. The desire for surgery from parents must be fiercely resisted because the majority (90%) resolve in time, surgery is not without its risks and the cyst can reoccur following excision.

Examination corner

Paeds oral: Clinical photograph of a child with obvious swelling at the back of the knee

Spot diagnosis with discussion afterwards of management, particularly how you deal with awkward parents demanding surgery for their child (second opinion!).

CANDIDATE: This is a clinical picture of a child, which shows an obvious swelling in the popliteal fossa. The swelling appears to be about 3 cm by 2 cm; the skin overlying the swelling appears normal. The picture is very suggestive of a popliteal cyst.

EXAMINER: How will you manage this condition?

CANDIDATE: Popliteal cysts are benign lesions, the vast majority resolve in time, surgery is not indicated and parents should be reassured about the condition.

EXAMINER: How long on average do they take to resolve?

CANDIDATE: Ninety per cent resolve over a 4-year period.

Table 25.6 Causes of in-toeing and out-toeing

	Causes of in-toeing	Causes of out-toeing
Most common causes	Persistent femoral anteversion Metatarsus adductus Internal (medial) tibial torsion	
Femur and hip	Persistent femoral anteversion Spasticity of internal rotators (CP)	Femoral retroversion Flaccid paralysis of IR
Leg and knee	Internal tibial torsion Genu valgum Blount's disease	External tibial torsion
Foot and ankle	Pronated feet Metatarsus varus Talipes equinovarus	Pes planovalgus Talipes calcaneovalgus

Assessment of rotational profile

Generally presents as either in-toeing or out-toeing (Table 25.6).

Foot progression angle (FPA)

Describes the direction in which the foot points during gait with respect to the line of progression, and can be altered by any abnormality at any level in the leg. Normal –5° to +20°. Average +10°.

Range of hip rotation (Staheli)

Place the child in the prone position with their knee flexed at 90° and their ankle held in neutral position. The leg acts as a protractor, indicating degrees of movement. Internal rotation (IR) is assessed by turning the legs away from the midline and external rotation (ER) by turning the legs one at a time towards the midline of the body. Normally IR is $<70°$. A value $>70°$ suggests anteversion of the femur. A normal value of ER is 20° and if it is $<20°$ this suggests femoral anteversion.

Ryder method

This calculates the degree of femoral anteversion. Place the child prone on the examination couch.

Flex the knees to 90° and internally rotate the leg while palpating the greater trochanter. Rotate the leg until the greater trochanter is most prominent laterally. The degree of IR at this point corresponds with the degree of femoral anteversion.

Anteversion is likely to be present if internal rotation exceeds 70° and external rotation is <20°.

Thigh–foot angle (TFA)

Tibial torsion is assessed by observing TFA. Flex the patient's knee to 90° and hold the ankle in neutral position by applying gentle downward pressure on the sole of the foot. Estimate the angle made by an imaginary straight line along the axis of the thigh and an imaginary line along the axis of the foot. Normally, TFA is 10–20° in external tibial torsion. If it is <10°, this indicates internal (medial) tibial torsion.

Transmalleolar–thigh angle

The transmalleolar axis is marked by palpating the medial and lateral malleoli joining the two points on the heel. A line perpendicular to this axis and the longitudinal axis of the thigh is assessed. The normal value is 0–45°. Less than this suggests internal tibial torsion.

Clinical approach

In-toeing accounts for the greatest number of paediatric orthopaedic referrals in the developed world.

The natural history of most cases of in-toeing (owing to wide normal variability in femoral anteversion) is of gradual spontaneous resolution over the first 7 or 8 years of life.

It is important to rule out an underlying pathology such as undiagnosed cerebral palsy or, in the case of out-toeing, a missed SUFE.

Idiopathic in-toeing does *not* respond to orthoses – Its natural history cannot be influenced save by derotation osteotomy which is certainly not indicated (unless persistent and disabling beyond 10 years of age).

There is *no evidence* to support the assertion that in-toeing causes spinal or hip problems, etc.

Examination corner

Paeds oral 1
Clinical measurement of anteversion of a femoral neck

Paeds oral 2: Clinical photo of in-toeing

How do you assess this child?

Obstetric brachial plexus palsy

This is said to occur in 1/2000 live births, with 75% of cases fully recovering, though these figures are highly subject to case ascertainment bias (i.e. it depends how hard you look for it).

The assessment of an older child with late presenting obstetric brachial plexus palsy has been seen in the exam. It is a challenging and highly specialized area of practice, but you must nevertheless have an appreciation of the condition and its consequences.

Aetiology

The cause is shoulder dystocia – In a cephalic delivery the shoulders are too wide for the pelvis and the upper roots are stretched to deliver the infant, whereas in breech delivery the lower roots are more vulnerable to being stretched.

Risk factors include large birth weight, instrumented delivery, C-section, breech.

Clinical picture

- Presentation is of a flail limb or one with much reduced movement – Classically held in the 'waiters tip position' for upper trunk (Erb's) palsies
- Assessment should pay attention to spontaneous shoulder elevation, elbow flexion and hand movements as indicators of root function
- Look for Horner's sign (poor prognostic factor)

Narakas classification

This is done at 2–4 weeks of age:

- Group 1 = C5/C6, biceps and deltoid paralysis
- Group 2 = C5/C6/C7, isolated long flexor function preserved
- Group 3 = Whole plexus, slight finger movement only
- Group 4 = Whole plexus ± Horner's

Differential diagnosis

- Pseudoparalysis owing to isolated clavicular fracture (which improves within days)
- Arthrogryposis
- Cerebral palsy

Natural history

- Function spontaneously recovers in most cases (neurapraxia), usually distally first
- Residual problems generally involve the shoulder
- Biceps recovery before 2 months of age is a good prognostic sign; conversely, failure of biceps to recover by 4 months is a poor prognosticator

Management

- Initially exercises (physiotherapy) to maintain range of movement (unopposed internal rotation of shoulder

eventually leads to posterior subluxation and remodelling of the shoulder joint)
- Referral of cases that fail to resolve fully by 2–3 months of age to supraregional obstetric brachial plexus palsy service is the norm in the UK
- Radiographs, MRI and neurophysiology may be required
- US assessment of the shoulder is gaining popularity and is likely to lead to increased intervention as more cases of posterior shoulder subluxation are identified
- The consequences of profound plexus injuries are dealt with by tendon transfer and bony realignment surgery about the shoulder (e.g. denotation osteotomy of the proximal humerus).

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Chapter 26

General principles, spine and pelvis

William Eardley and Paul Fearon

Introduction

Setting the scene

In order to pass the trauma viva you need to confer to the examiner that you have the required level of knowledge, practical exposure and experience that displays safe accepted practice for a newly appointed day 1 consultant in a District General Hospital.

The ability to demonstrate local and regional knowledge of accepted trauma pathway networks, in view of the establishment of Major Trauma Centres (MTCs) in England, should not be underestimated, especially with the establishment of bypass protocols and the management of complex and multisystem injuries.

The viva will cover simple fracture management/complications and postoperative care, and the questioning can be diverse and probing focusing on how you would manage postoperative complications, within the resources available to yourself.

Be prepared to be shown laminated photographs of implants, clinical illustrations, x-rays, CT and MRI scans, as these should all be part of your normal clinical exposure and patient management.

Anything from adult and paediatric fracture management is on the table, and the ability to approach the scenario in a logical stepwise fashion should not be made light of or put down.

Clinical case scenarios will be discussed and we try to construct answers that follow a logical pathway, such as:-

- Indications for surgery
- Operative options and "your own" preferred method and why?
- Consent – Process of informed consent and 'special cases' – E.g. Jehovah's Witnesses
- WHO checklist, special equipment required and briefing a theatre team
- Patient positioning and draping
- Surgical approaches
- Reduction and stabilisation methods
- How to avoid complications, any intraoperative tips?
- Rehabilitation and postoperative outcomes
- What does current literature show?

This at least creates a comprehensive informed management plan for the subject area that you are dealing with.

Subject areas

We will try and break the topics into general revision blocks as follows:

1. General trauma topics
2. Implants and principles of fixation – Biomechanics of trauma
3. Paediatric conditions
4. Spinal trauma
5. Pelvic trauma
6. Miscellaneous – Compartment syndrome, amputation

General trauma topics

Trauma networks – Creation of MTCs

Good trauma care involves getting the patient to the right place at the right time for the right care. It has been estimated that by improving the organization of trauma care, an additional 450–600 lives could be saved in NHS hospitals. Regional Trauma Networks went live across England in April 2012. These are based on agreed principles of care using local models and implementation in each geographical area. A map of the current English MTCs is available[1].

Regional models all follow similar pathways, looking at:

- Prevention
- Initial contact
- Prehospital assessment
- Acute trauma care
- Appropriate rehabilitation

Results from the Trauma Audit and Research Network (TARN) national audit show that 1 in 5 patients who would have died before these networks were established are now surviving severe injuries.

Patients are best treated in specialist centres that aren't always closest to their home. Similar to stroke and cardiac services, we know that whilst patients may spend longer in an ambulance the expert care provided at MTCs saves lives and improves outcomes for patients.

Professor Chris Moran, National Clinical Director for Trauma for NHS England said:

> 'Our patients now receive much more rapid care from specialist trauma teams who can identify life-threatening injuries much

quicker, access key tests such as CT scans faster and perform life-saving operations earlier'.

'Patients with complex fractures and soft-tissue injuries, which often cause permanent disability and require specialist surgery are also benefiting. Before, patients often waited 7–10 days before being transferred to specialist units, now over 90% of patients are transferred to the right hospital within 2 days.'

As well as improving survival, a key aim of the Major Trauma Networks is to improve the quality of life in the survivors. The networks have redesigned rehabilitation pathways to give the patients the best chance of recovery following surgery. It is predicted that for every additional survivor, three further patients will make an enhanced recovery, which hopefully will allow them to return to their families and to work[2]. This development was achieved by the coordinated efforts from the National Clinical Director for Trauma, improvements in inclusive trauma pathways, the British Orthopaedic Association Standards for Trauma (BOAST) and the National Institute Clinical Excellence (NICE) guidelines on good quality care Additional secondary levers included the Best Practice Tariff, CQUIN (Commissioning for Quality and Innovation) and Dashboards for Major Trauma.

Trauma Audit and Research Network (TARN)

Established in 1990 by The Universities of Manchester and Leicester. Their core work is supporting improvements in trauma care through audit and research. They ensure Quality Data coordination, with regular and informative feedback. By doing so then can offer responsive local reports and specific local improvement and publish National reports to inform the planning of trauma services. All of which creates improvements in Trauma Care.

Understanding the benefits and the risks associated with different types of treatment is important for all patients. However, it is not generally appreciated that there are variations in the success of treatments in different hospitals. It follows that there are probably opportunities to improve care.

TARN has enabled a system to benchmark practice through monitoring and publishing process measures, allowing on-going and continued appraisal and improvement for the system.

Those who are injured may have one or many injuries and the Injury Severity Score (ISS) is an anatomical score that measures the overall severity of injured patients.

All injuries are assigned an Abbreviated Injury Scale (AIS) code and score from an internationally recognised dictionary that describes over 2000 injuries and ranges from 1 (minor injury) to 6 (an injury that is thought to be 'incompatible with life'). Patients with multiple injuries are scored by adding together the squares of the three highest AIS scores in three predetermined regions of the body. This is the ISS which can range from 1 to 75. Scores of 7 and 15 are unattainable because these figures cannot be obtained from summing squares. The maximum score is 75 (25 + 25 + 25). By convention, a patient with an AIS 6 in one body region is given an ISS of 75. The ISS is non-linear and there is pronounced variation in the frequency of different scores. By stratifying patients this allows predictors of survival to be established when national results are compared.

Management of polytrauma patients

This is a common exam scenario and basic logical principles need to be addressed and applied. The questioning can cover the following subject areas:

- Multisystem patient involvement – Again this is an easily approached question, using an ATLS® approach, remembering the basics and approaching the patients in a logical management sequence
- Multiple casualty scenarios – Mass casualty incidents offer significant challenges for prehospital and emergency room workers. Fire, Police and emergency personnel must secure the scene, establish communications, define individuals' roles and responsibilities, allocate resources, triage patients, and assign transport priorities. Pre-alerts to appropriate emergency departments (EDs) in Trauma Networks allows transfer out of patients and reorganization of the available physical resources and availability and type of manpower. Mass-casualty incident trainings is pivotal, to ensure a well-coordinated response, such as communications, incident management system and triage. The use of triage skills in mass-casualty scenarios can only be improved with training and paper exercises and follow up discussions are simple tools for initial education[3], prior to simulated major incidents
- Multispecialties input and professional management of teams – This is closely related to communication skills, team management and having a coordinated approach in the management of such patients. You should be familiar with local major incident plans, and your potential role if called into action
- Prehospital management and treatment in the field – Treatment is often initiated in the field for life-critical injuries/massive haemorrhage. It is difficult to compartmentalize treatment stages, as resuscitation and treatment needs to be dynamic. An excellent review looking at an evidence-based, practical guide for the orthopaedic surgeon[4] covers these points in depth, but especially from the practical perspective
- Pelvic binders, there usage in prehospital setting, with correct application at the level of the greater trochanter is expected. Familiarity with the different commercial brands is required. The need for AP pelvic x-rays post binder release to ensure the absence of an open book pelvic injury that has been well reduced by the binder is also necessary. The application of external fixators in the ED has been greatly reduced by the usage of binders, and if a binder fails to stabilise haemodynamically a pelvic patient, it is unlikely that a fixator will have any additive effect

- Addressing haemorrhage in the multiply injured patient– Bleeding remains the leading cause of mortality in injured patients and a rational, contemporary approach to resuscitation is mandatory in candidates presenting for the examination

The ATLS® evaluation of the injured patient is seen as baseline. Detailing the recognition and classification of haemorrhage, similarly is a basic requirement. To score well on a question regarding haemorrhage, the candidate must be able to detail current concepts on both fluid resuscitation, laboratory evaluation of ongoing bleeding and pharmacological adjuncts to haemorrhage control.

All hospitals receiving seriously injured casualties are now mandated to have a major haemorrhage protocol. The candidate is advised to read the protocol in the hospital in which they work. Answering the question in this manner, with reference to their own hospital protocol or a case in which they were involved is much more likely to be a successful grounding from where to score points.

What triggers a major haemorrhage protocol to be initiated is decided on a local level; hence, why candidates are urged to be familiar with their own unit's response. In the generality, however, receipt of prehospital information of hypotension (systolic blood pressure <90 mmHg) significant tachycardia (heart rate >120 beats/min) and penetrating trauma in the casualty with a significant injury mechanism are useful baseline approaches.

Once the patient has arrived in the ED, additional information that informs the decision to activate the protocol is the presence of free fluid on ultrasound scan of the abdomen, a raised lactate and diminished haematocrit.

In addition to the ATLS® approach, the focus of the orthopaedic surgeon should be on ensuring that continuing extremity bleeding is addressed. Pre-hospital tourniquet efficacy can be limited and a low threshold for supplementing these with a padded pneumatic tourniquet should be maintained. A pelvic binder should be in situ already but if not, one should be applied. These measures help to arrest ongoing 'orthopaedic' sources of haemorrhage.

Candidates should be aware of what constitutes the blood product resuscitation response of their hospital in the major trauma patient and which personalities (haematologist/laboratory technician) are mobilized when a major haemorrhage scenario is activated. The simplicity of the situation is that in the bleeding patient, whilst ongoing sources of haemorrhage are being arrested, missing blood should be replaced with, not surprisingly, warmed blood.

The candidate may be asked to define massive transfusion. As with all aspects of trauma, multiple definitions exist and the unwary candidate may fall foul of attempting to commit too many definitions to memory. Keeping things simple and memorable, '*massive transfusion is transfusion of the total blood volume in a 24-hour period*'.

The candidate then may be asked to detail the fluid that they give and the ratio of blood products administered. Again there is always debate in the literature as to the exact ratio of blood products that should be given. The safest and efficacious approach to initial resuscitation in major haemorrhage is: *The early transfusion of warmed blood along with platelets and fresh frozen plasma in a 1 : 1 : 1 ratio*.

Further management and blood product replacement once the initial 'trauma pack' of these components has been administered begins to go beyond the remit of the examination. The candidate, however, should be able to produce an answer that safely addresses ongoing haemorrhage. An ATLS® type re-evaluation of potential bleeding sites, consideration of interventional radiology input and laboratory assessment of the blood parameters are all acceptable approaches. The latter involves mentioning the use of rotational thromboelastometry (ROTEM)[5]. Whilst previously considered out with the knowledge base of the orthopaedic trauma surgeon, ROTEM, a viscoelastic method for hemostasis testing in whole blood, is becoming increasingly commonplace in trauma resuscitation. In essence this is bespoke, targeted blood product resuscitation for ongoing blood therapy, based on the viscoelastic behaviour on in-vitro assessment of the patients' blood clot. This is predominantly dealt with by the critical care team but it is important that candidates are aware of this for the examination.

Alongside initial management, administration of a major haemorrhage protocol trauma pack, evaluating or arresting ongoing bleeding and assessing the individual clotting needs of the bleeding patient, administration of tranexamic acid needs to be detailed.

Tranexamic acid (TXA) is becoming familiar in both the trauma and elective setting of orthopaedics. TXA is an antifibrinolytic that competitively inhibits the activation of plasminogen to plasmin, thus, preventing the degradation of fibrin, the protein central to the framework of blood clots formed in trauma. Major studies in both the civilian and military environments (CRASH[6] and MATTERS[7] respectively) have demonstrated significant benefits of the administration of TXA in patients requiring blood product resuscitation, particularly those where major transfusion is required. What has been demonstrated is that timing is fundamental and TXA given within 3 hours of injury has a proven benefit compared to that given late.

By becoming familiar with the major haemorrhage protocol in their own hospital and structuring an answer among the lines suggested above, candidates should be able to practice safe trauma care and score well in the examination. Massive transfusion protocols with the early delivery of blood, fresh frozen plasma, platelets and tranexamix acid improves mortality. All MTCs will have their own protocols and currently there is now a move to utilizing the major haemorrhage packs in the prehospital setting with auditing of results.

- Trauma team make-up in the ED – This should be a simple question to answer, as long as a structured reply has been thought out. It leads onto
- Trauma team training – How do you ensure ongoing continuing education if not based in MTC – Knowledge of simulation models such as the European Trauma

Course/Advanced Trauma Life Support. How human factors play a pivotal role in the dynamics of a team and ultimately patient outcome

- Whole body CT scan. The appropriateness of early contrast enhanced CT with immediate reporting has greatly enhanced the management of trauma patients, with appropriate body zone surgery in a more timely fashion. (Be able to identify abnormal organ findings on a trauma scan)
- Damage control orthopaedics and resuscitation. This subject area is not an excuse to apply spanning external fixators and then leave the patient for colleagues to sort out later. Patients not physiologically robust to undergo definitive surgery even after resuscitation will need staged management and further supportive care in an ITU setting. Often temporary splintage/external fixators/pelvic packing, etc, is part of the resuscitation process. Knowledge of coagulopathy measurement, lactate, renal function and body temperature will be expected in the further management of these patients, and close working relations with trauma anaesthetists and intensivists is paramount
- Spanning external fixators – Be able to talk through spanning the knee, ankle, elbow. Open or close procedures? Appropriate pin sizes in relation to body zones and patient habitus. Difficulties encountered and how you deal with them are all acceptable questions
- Amputation – A full knowledge of the levels and the structures encountered will be needed. Be able to describe the creation of myodesis for stump stability. What is involved in the rehab setting? A knowledge of specialist limb fitting centres, psychological support and vocational training as necessary, will be expected
- Revascularisation (BOAST 6 guidelines)[8]. The basics still apply. Resuscitation and management of all life-threatening injuries must take priority. A full and comprehensive neurological examination must be undertaken and documented in the medical notes. The pulseless deformed limb should be realigned/reduced and appropriately splinted. A repeat examination should be undertaken and again documented before appropriate radiological imaging tests obtained. A devascularized limb requires urgent surgical exploration and should only be delayed to attend to life threatening injuries. All Trauma networks in conjunction with MTCs and trauma units must have an emergency referral protocol with appropriate specialty involvement (orthopaedics, plastics, vascular, etc). The limb must be revascularized as a surgical emergency. Warm ischaemic time (in surgery, the time a tissue, organ, or body part remains at body temperature after its blood supply has been reduced or cut off but before it is cooled or reconnected to a blood supply) varies depending upon level of amputation and is muscle dependant. On average a warm ischaemic time beyond 4–6 hours for a limb is associated with a higher rate of amputation. The sequence of surgical intervention can be critical. Temporary shunts followed by skeletal stabilisation, allow re-evaluation of limb viability prior to definitive vascular reconstruction. Any decisions to amputate should be made by two senior consultants experienced in trauma management. If reconstruction proceeds, peripheral nerves should be tagged for later repair or attended to at the same sitting. Post-reperfusion compartment syndrome is common and as such there should be a low threshold for fasciotomies
- Pelvic packing – Extra peritoneal pelvic packing. The aim is to directly compresses the life threatening retroperitoneal bleeders causing a direct pressure and tamponade effect that stops venous and arterial pelvic bleeding. This is a rapid method for controlling pelvic fracture-related haemorrhage that can supplant the need for emergency angiography. As part of the damage control resuscitation process, there is a significant reduction in blood product transfusion after extra peritoneal packing and this approach appears to reduce mortality in this select high-risk group of patients[9]
- Interventional radiology – Needs to be utilized as part of the MDT resusicitive process with coordinated senior clinical decision making. Unit protocols are modelled on North America experience, one such example is the Eastern Association for the Surgery of Trauma practice[10]. Broadly speaking after non-pelvic sources of blood loss have been ruled out, patients with pelvic fractures and hemodynamic instability or signs of ongoing bleeding should be considered for pelvic angiography. Additionally patients with CT-scans demonstrating arterial intravenous contrast extravasation in the pelvis may require pelvic angiography and embolisation regardless of hemodynamic status
- ITU resuscitation – Most patients with multiple injuries will have a raised lactate level. With resuscitation, splintage of limbs, fluid replacement and re-warming, lactate levels will begin to normalise. However, a period of supportive care may be necessary and the ITU is the most appropriate setting. With trends in lactate levels being more predictive regarding suitability for patient robustness under anaesthesia, there needs to be continued communication between both anaesthetic and surgical teams. With lactate levels >2.5 mmol/l continued resuscitation and damage control should be practiced and it is only when the trend of lactate is <2 mmol/l, should total care be instigated[11]

BOAST 4 guidelines[12]

The management of severe open lower limb fractures is frequently asked. Essentially best outcomes are achieved by timely specialist surgery, rather than emergency surgery by less experienced teams. Patients need to meet both certain fracture pattern and soft-tissue injury pattern criteria.

Fracture pattern

comminuted tibial fracture
Segmental fractures
Fractures with bone loss
Soft-tissue injury:
Skin loss, such that direct tension-free closure is not possible
Degloving

Muscle requiring excision
Associated arterial injury
Wound contaminated with marine, agricultural or sewage

Standards for practice and TARN audit are set out in the above reference, but essentially the BOAST 4 guideline requires:

1. Early identification of severe open fractures of the tibia
2. Joint care from orthopaedic and plastic surgeons
3. Surgical wound debridement and operative fracture stabilisation within 24 hours
4. Definitive soft-tissue cover within 72 hours of injury

BOAST 3 guidelines[13]

Major pelvic and acetabular fractures must be managed with an established trauma network system with defined acknowledged referral pathways. A mismanaged pelvic injury can lead to early death from haemorrhage or major disability while delayed or poor management of an acetabular fracture can lead to accelerated osteoarthritis and avoidable permanent hip dysfunction. Additionally urological, pain, and psychosocial dysfunction is common. As well as pelvic and acetabular evaluation as part of the resuscitative procedure, an index of suspicion for urological injures is required and early contrast studies and urgent senior urological input is necessary.

Concerning open pelvic fractures, with wounds to the groin, buttock, perineum, vagina or rectum, these require urgent bladder drainage by cystostomy tube and bowel diversion with an end-colostomy (with washout). These demand urgent senior general surgical and urologist input. Any colostomy should be sited in an upper abdominal quadrant remote from potential definitive pelvic surgical fixation approaches.

Hip dislocations must be reduced urgently and then an assessment of stability recorded. The neurovascular status before and after reduction must be documented. Skeletal traction should be applied. If the hip remains irreducible or unstable, then urgent advice should be sought from a specialist in acetabular reconstruction. Immediate transfer should be considered.

Post reduction, a CT scan must be done to exclude bony entrapment and to assess hip congruence and the extent of any fracture. These images should be referred to an expert in acetabular fracture reconstruction promptly. Displaced fractures requiring reduction and internal fixation should undergo surgery by an acetabular reconstruction expert as early as possible, ideally within 5 days but no later than 10 days from injury.

Rehabilitation pathways post-trauma

The rehabilitation prescription reflects the assessment of the physical, functional, vocational, educational, cognitive, psychological and social rehabilitation needs of a patient.

Patients with an ISS >8 undergo an initial rehabilitation prescription within 2–4 days of admission. The prescription states how these areas will be addressed. The prescription is an extension of a discharge/transfer summary and should include ongoing health and social care plans

The goals are:

To record important demographic and historic clinical data
To record the present situation
To specific the likely longer-term prognosis, acknowledging uncertainty but remaining realistic
To specify the rehabilitation needs, both specific and immediate and more general and longer-term
To specify the risks still extant at the time of transfer

Improving quality of emergency care and rehabilitation after trauma has become an NHS priority. The Rehabilitation Prescription was incorporated into the Best Practice Tariff (BPT) for Major Trauma to drive improvements in rehabilitation but was having little impact after hospital discharge. Currently Regional Trauma Networks are concentrating on the enhanced postoperative care to improve function, using acute and community rehabilitation providers.

Examination corner

Laminated radiograph of open book pelvis shown (Figure 26.1)

EXAMINER: This is a 24-year-old motorcyclist who has come off his bike at high speed. He has been brought to casualty with a blood pressure of 90/60 and a tachycardia of 130. How would you manage this patient?

COMMENT: The viva question is all about emergency resuscitation of the patient and not so much initially focused on the open book pelvis injury itself. Candidates have to recognise that this is a life threatening injury and manage appropriately. The examiners are testing whether you have practically encountered this situation and are you safe dealing with this injury. These are serious injuries. For the unwary if you don't answer in an appropriate fashion you will fail and you could end up quite easily with a

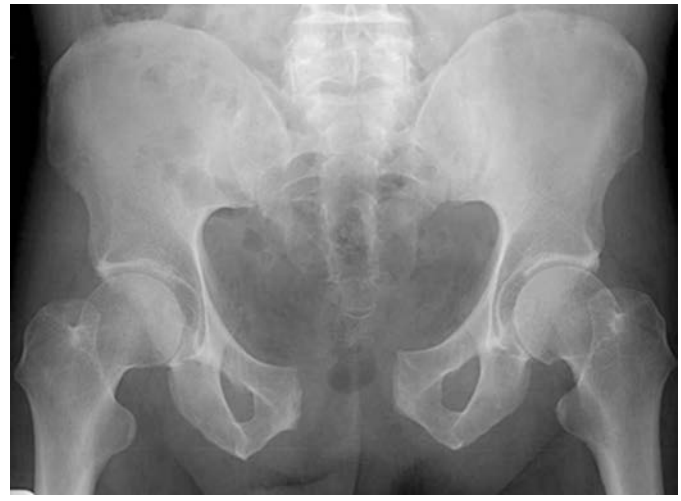

Figure 26.1 AP radiograph demonstrating open book pelvis injury

double 4 if you are unsafe which will significantly reduce your chances of getting through the exam.

CANDIDATE: I would manage him according to ALTS® principles, I would establish a patent airway with adequate ventilation and oxygenation. I would give supplementary oxygen via a face mask.

EXAMINER: His airway and breathing are fine.

CANDIDATE: I would control any obvious haemorrhage, obtain intravenous access with two large caliber cannulas. I would send blood away for cross-matching, FBC, U&Es, coagulations screen. I would give 1–2l of normal saline or lactated ringers.

EXAMINER: The patient already has been given 2 litres of normal saline by the ambulance crew.

CANDIDATE: I would give a further unit of normal saline until the blood arrives.

EXAMINER: What are the dangers in giving large amounts of crystalloid or normal saline in trauma patient. Is it safe practice?

CANDIDATE: (Pause and hesitancy) No.

COMMENT: The candidate didn't get the chance to fully answer the question.

EXAMINER: How long does cross-match blood take to arrive in casualty?

CANDIDATE: About 40 minutes.

EXAMINER: You would be lucky if that was the case in our hospital.

COMMENT: We think this is the examiner prompt that O-negative blood should be given instead of crystalloid.

EXAMINER: What about the pelvis?

CANDIDATE: I would apply a pelvic binder.

EXAMINER: How do you apply a pelvic binder?

CANDIDATE: The ambulance crew should have applied this at the scene of the accident.

COMMENT: We don't think this was a good answer to give in the exam and suggests the candidate doesn't know.

EXAMINER: The ambulance crew haven't done it. How do you put a pelvic binder on?

CANDIDATE: I would use the ones provided in casualty and wrap it round the pelvis.

EXAMINER: The binder should be centered over the greater trochanter and not placed over the iliac crest or abdomen, as this is ineffective. This provides the best mechanical stability of the pelvic ring structures. A misplaced binder may exacerbate a pelvic fracture if there is an injury through the iliac crest. When placed too high it will also obstruct access for laparotomy.

Do you log roll the patient first and check for spinal injury or do you just put on the pelvic binder?

CANDIDATE: I would log roll the patient and check for a thoracic and lumber vertebral injury as there is a high chance of coexisting injuries.

EXAMINER: How many personnel does it take to log roll a patient?

CANDIATE: Four.

EXAMINER: How many personnel does it take to put a pelvic binder on?

CANDIDATE: Three.

COMMENT: The correct answer is that you would put a pelvic binder on first as the pelvic injury be a severe life threatening injury this manoeuvre should help with haemorhage control. Two personnel gently lift the patient up slightly whilst maintaining a neutral spinal position whilst a third person slides the binder underneath the patient. Log rolling a patient with a possible pelvic fracture should be avoided as this may exacerbate any pelvic injury.

We think the examiner may have been probing the candidate as they sensed he/she hadn't actually put a pelvic binder on a patient and just read it from a book. Examiners seem to sniff out candidates who have just read facts from a book rather than having 'on the job' practical experience. They may then tailor their questions to confirm this initial impression. Ultimately candidates lacking practical experience may lose scoring opportunities

EXAMINER: How are you going to manage his patient's low blood pressure?

CANDIDATE: I would cross-match for 4 units of blood.

EXAMINER: When would you give this blood?

COMMENT: The patient needed O-negative blood immediately. Pelvic fractures are one of the true (few) life-threatening orthopaedic emergencies. The overall mortality rate is between 10% and 20%, and this jumps to 38% if the patient is hypotensive on admission, and to 50% if the patient has an open pelvic fracture. Underestimating the hemodynamic status of young patients can be fatal, particularly because the retroperitoneum can contain up to 4 l of blood.

CANDIDATE: (*No answer*)

EXAMINER: Are you going to wait for cross-matched blood to arrive?

CANDIDATE: I would give him O-negative blood.

COMMENT: The candidate wasn't convincing, an afterthought or guess that this was what the examiners wanted. We are not sure if the viva was recoverably at this stage but if it had suddenly clicked for the candidate they should have emphasized the need for immediate aggressive resuscitation and gone on to discuss massive transfusion policy instead of letting the examiner continue to be in charge of the viva situation.

EXAMINER: What about a massive transfusion policy?

CANDIDATE: Successful management of major haemorrhage requires a protocol-driven multidisciplinary team approach with involvement of medical, anaesthetic and surgical staff of sufficient seniority and experience, underpinned by clear lines of communication between clinicians and the transfusion laboratory.

I would give blood, platelets and fresh frozen plasma is a 1 : 1 : 1 ratio.

EXAMINER: What is in a local hospital shock pack?

CANDIDATE: Red blood cells and fresh frozen plasma.

EXAMINER: In what values?

CANDIDATE: Four units of red blood cells, 4 units of FFP, 1 bag of platelets and 2 bags of cryoprecipitate.

EXAMINER: It is variable depending on local hospital policy.

Debrief

This viva is all about if you adequately identify and resuscitate a patient in Grade 4 shock. Managing the unstable pelvic fracture is a four-step process: Identification, resuscitation, immobilization, and transportation (to a trauma centre).

It puts the candidate on the spot as to if they would immediately give O negative blood. There was a lot of probing of the practicalities of how you would apply a pelvic binder. This is trying to get away from book knowledge to test whether a candidate has actually performed the procedure

Other potential causes of shock in trauma such as tension pneumothorax, cardiac tamponade, cardiogenic shock, neurogenic/spinal shock were not discussed. The use of antifibrinolytics was not discussed. The candidate did not manage to progress the topic beyond the basics of massive transfusion

In 2011, the CRASH-2 trial identified that tranexamic acid significantly reduced all-cause mortality, in trauma patients with significant haemorrhage, if given within 3 hours of injury.

Implants and principles of fixation

Key viva themes – Plate design and use in orthopaedics

You may be handed a plate and asked to talk about the following points: What is it/application/mode of action/material/footprint/sizs of drill/taps/screws, etc. (Figure 26.2).

As an example, Figure 26.3 is a precontoured anatomical plate made from stainless steel used in the management of periarticular tibial plateau fractures. This plate is intended for use on the lateral tibial plateau and has combination holes to allow the insertion of both standard and locking screws, and so forth.

The new combination hole has two parts:

1. One part has the design of the standard Dynamic Compression Plate/Limited Contact Dynamic Compression Plate. (LC-DCP™) The dynamic compression unit (DCU) accepts a conventional screw allowing bidirectional axial compression or the placement of a lag screw through the plate
2. The other part is threaded and conical, to accept a locking-head screw

Combination plates may be useful in certain fracture patterns in which one aspect of the fracture would benefit from anatomic reduction and compression (i.e. intra-articular component), whereas another fracture component would benefit from bridging fixation (ie, comminuted metadiaphyseal portion)

As follows, stainless steel 316L. This type of stainless steel is particularly effective as a surgical implant when in cold-worked condition. What makes the type 316L ideal as an implant device is the lack of inclusion in this material. Material with inclusion will also contain sulfur and this is a key component to encouraging corrosion of metals. Stainless steel is a metal alloy metal. By adding the chromium (16%) element to stainless steel, this metal becomes corrosion resistant. The addition of carbon and nickel (7%) to stainless steel helps stabilise the austenite to stainless steel. With surgical implants, molybdenum is added to the stainless steel alloy that forms a protective layer sheltering the metal from exposure to an acidic environment. It has to be stressed that the ferrite element should not be incorporated into stainless steel as this gives the metal a magnetic property, which is never used for surgical implants as it could interfere with magnetic resonance imaging (MRI) equipment. These areas of questioning may also come up in the basic science viva, but be prepared to talk about equipment used in fracture management.

You may be handed a wrist plate and asked the above points but then asked what you know about the DRAFFT study? (Distal Radial Acute Fracture Fixation Trial)[14].

Questions are not only limited to implants, as you may be asked to explain how a vacuum-assisted closure wound system works. Under negative pressure, VAC® therapy with proprietary VAC® GranuFoam™ dressings applies both mechanical and biological forces to the wound to create an environment that promotes wound healing. These forces are known as macrostrain and microstrain.

Macrostrain is the visible alteration that occurs when negative pressure contracts the open-pore VAC® GranuFoam™ dressing.

- Draws the wound edges together
- Provides direct and complete wound bed contact

Figure 26.2 Multiple varied plates used in fracture management

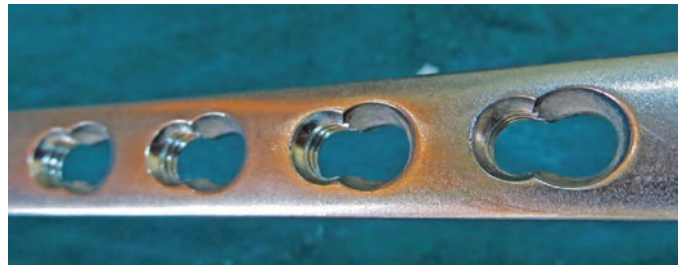

Figure 26.3 Combination plate. The combi holes allow placement of conventional cortex and cancellous bone screws on one side and threaded conical locking screws on the opposite side of each hole

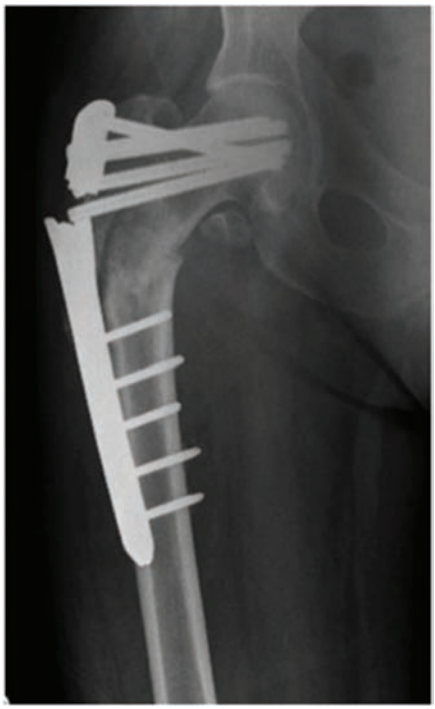

Figure 26.4 AP pelvic radiograph demonstrating broken proximal femoral locking plate

- Evenly distributes negative pressure
- Removes wound exudate and materials that may cause infection

Microstrain takes place at the cellular level, leading to cell stretching induced by the open-pore VAC® GranuFoam™ contact with the wound bed.

- Encourages moist wound healing
- Promotes granulation tissue formation
- Promotes perfusion through angiogenesis
- Increases cellular proliferation and migration
- Reduces oedema

There is considerable overlap between basic science questioning and trauma applications, and not having an understanding of the biomechanics will be identified in the trauma oral.

Examination corner

Trauma oral 1

Broken femoral plate shown (Figure 26.4).

EXAMINER: This 67-year-old male sustained a right intertrochanteric femoral fracture after a fall fixed with a locking plate 7 months ago. He presented to casualty last night with sudden onset of right hip pain and inability to weight bear.

CANDIDATE: This is an AP radiograph of the right hip. It shows a comminuted extracapsular fractured neck of femur, which has been treated with a locking plate. The locking plate has broken. There is implant failure. This looks like a periarticular limited contact large fragment locking femoral plate, which has locking and non-locking screw options. It should resist angular (varus/valgus) collapse. The plate is anatomically precontoured to the metaphyseal zone of the proximal femur.

COMMENT: The candidate took the harder option in describing plate features. The other route to take is to say the fracture hasn't healed and at 7 months it looks like an established non-union has occurred. One of the first points the candidate should have mentioned is that infection must be excluded. The candidate should try and steer the viva down to the factors that influence fracture healing and then move onto treatment options. This then becomes a less challenging viva.

EXAMINER: Why has the plate failed?

CANDIDATE: The most likely cause is non-union at the fracture site with plate failure occurring secondary to fatigue failure. Even with stable fixation with sturdy implants, plate breakage is a risk in the absence of bony healing.

Proximal femoral fractures are usually managed with either a dynamic hip screw or intramedullary nail so the use of a proximal femoral plate would suggest a more complicated proximal femoral fracture such as multifragmentary pertrochanteric or reversed intertrochanteric.

The stability of a locked plate construct is related to the length of the plate, cross-sectional area, the material properties, the screw density and diameter about the fracture site and the use of unicortical vs bicortical screws.

It is important to achieve anatomic reduction, as the PF-LCP does not allow controlled collapsed of the fracture fragments. Fractures that are inadequately reduced result in high varus strains at the fracture–implant interface. This leads to progressive loosening of the locking screws, varus collapse of the fracture and eventual plate failure.

Multiple locking screws increase bony purchase of the femoral neck and are especially advantageous in fractures with significant bone loss. It is important to plan well for the surgery, ensure good fracture reduction, template for the plate size, ensure the correct sequence of screw placement is adhered to. I am familiar with use of the plate but would make sure the company rep was available if the theatre team were unsure or inexperienced with the kit.

Pelvic and acetabular fractures

High-energy pelvis fractures

Key viva themes – Principles of resuscitation and polytrauma, treatment of major haemorrhage

There are now BOA guidelines for the management of pelvic and acetabular fractures. These are discussed below.

Initial assessment – ATLS® principles and resuscitation

- Major haemorrhage may occur after pelvic fracture; these injuries are frequently associated with other major life threatening injuries of the thorax and abdomen. A protocol for managing haemodynamic instability in pelvic fractures is shown in Figure 26.5

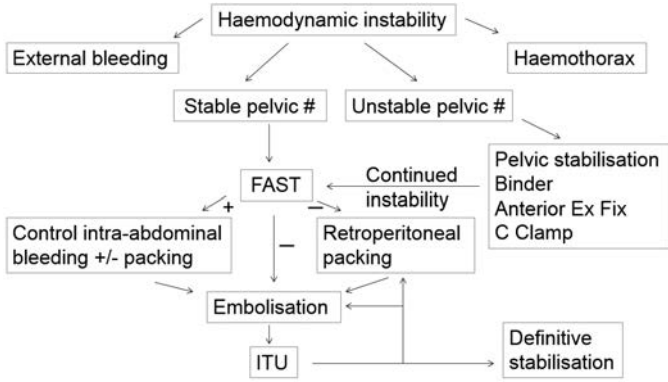

Figure 26.5 Managing major haemorrhage

- The early application of a pelvic binder will aid resuscitation, control haemorrhage and facilitate a laparotomy if required. Pelvic binders control bleeding by compressing and stabilising fractures, not by significantly reducing pelvic volume. Binders may be used in all fracture patterns and not just open-book injuries. When there is ongoing haemorrhage with haemodynamic instability, blood products should be given and the bleeding stopped either through angiographic embolisation (where possible), or pelvic packing/tamponade. A CT scan should be performed once the patient is haemodynamically stable
- Severe displacement of the pelvis and marked posterior disruption are poor prognostic signs. The presence of severe neurological and vascular injury suggests significant instability
- Around 5–10% of pelvic fractures are associated with major urological injury and this must be actively excluded as their presence suggests greater energy and instability. Contrast studies and specialist input is required urgently as intraperitoneal injuries require open repair. Open pelvic fractures (with groin, rectal or vaginal wounds) require early senior specialist input with diversion. Ensure the end-colostomy is sited in an upper quadrant away from any potential pelvic surgical approaches
- Specialist input should occur within 24 hours from a specialist pelvic unit for treatment advice and to facilitate transfer if required. Definitive surgical treatment should be carried out within 5 days

Investigations – AP pelvis with pelvic inlet and outlet views. CT scan is part of the pelvic ring evaluation process and will give better detail of posterior ring injuries, define comminution and fragment rotation

A plain radiograph may show displacement of the posterior sacro-iliac complex or avulsion fracture of the transverse process of the lower lumbar vertebrae. Anterior lesions may include pubic rami fractures and diastasis of the pubic symphysis

Young and Burgess' classification[15]

This is based on the mechanism of injury and severity of pelvic fracture. Type A (lateral compression), type B (AP compression) and type C (vertical shear). Within the AP compression (APC) and lateral compression (LC) categories there are three further subtypes

- AP compression
  - APC I – Symphysis widening <2.5 cm
  - APC II – Symphysis widening >2.5 cm. Posterior ligaments intact
  - APC III – Disruption of anterior and posterior SI ligaments. High risk of vascular injury
- Lateral compression
  - LC I – Rami fracture with ipsilateral anterior sacral ala compression fracture
  - LC II – Rami fracture with ipsilateral posterior ilium fracture
  - LC III – LC II injury with contralateral posterior injury
- Vertical shear
  Vertically displaced injury with anterior and posterior disruption
- Combined mechanical injuries
  Combination of other injury patterns: Lateral compression/vertical shear or lateral compression/AP compression

Tile classification[16]

This is based on the integrity of the posterior SI complex

Type A – Stable ring. The SI complex is intact
Type B – Rotationally unstable. Partial disruption of the posterior SI complex. Often requires operative fixation
Type C – Rotationally and vertically unstable. Complete disruption of the posterior SI complex

Definitive management of pelvic fracture

Non-operative – Undisplaced fractures, patient too ill or significant co-morbidities.

Operative management

Pubic diastasis – External fixation or plate fixation of pubic diastasis via a Pflannesteil approach. If associated rami fracture, use ilioinginal or stoppa approach and plate fixation. In cases of anterior comminution or soft-tissue injury, an anterior internal fixator may be used (INFIX), using supra-acetabular pedicle screws connected by a subcutaneous connecting rod[17].

Sacroiliac disruption – Open reduction and anterior plate fixation or posterior trans-iliac rods or closed reduction and percutaneous sacroiliac screw fixation.

Complications – Chronic pain, mental health issues, pelvic obliquity, leg length or rotational discrepancy, gait abnormalities, urological and sexual disabilities are common after pelvic

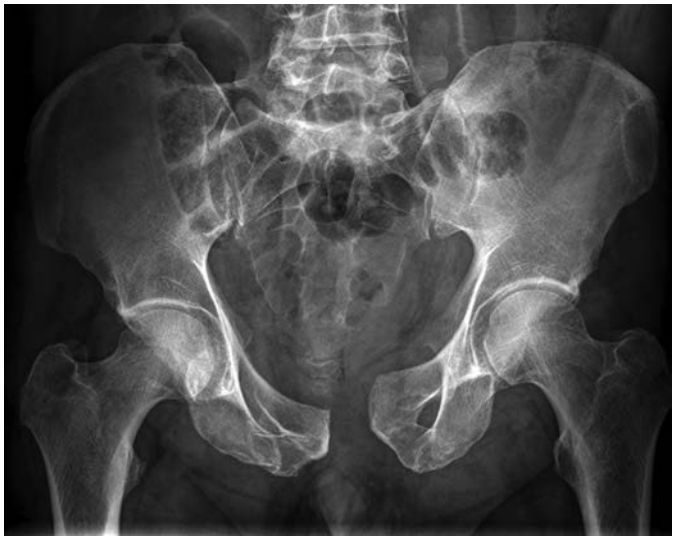

Figure 26.6 AP pelvis showing open book pelvis injury

fractures. Nerve injuries are common after vertical shear fractures. Vascular injuries may be associated with the initial injury, and care must be taken during the ilioinguinal approach not to injury the corona mortis vessel. Non-union, mal-union and DVT. Morel–Lavalle lesions (closed degloving injuries that lead to significant soft-tissue injury).

Examination corner

Trauma oral 1

Laminated radiograph shown of open book pelvis (Figure 26.6)
This is a 41-year-old male paedestrian hit by a car.

CANDIDATE: AP radiographs demonstrate an open book pelvis injury. There is significant diastasis of the pubic symphysis and widening of the right sacro-iliac joint. This is a type B (APC II) injury. There is >2.5 cm symphyseal diastasis, widening of the sacroiliac joints caused by anterior sacroiliac ligament disruption. Disruption of the sacrotuberous, sacrospinous and symphyseal ligaments with intact posterior sacroiliac ligaments results in the open book pelvis. The intact posterior SI joint acts as a hinge allowing the right hemipelvis to externally rotate but vertical stability is maintained.

COMMENT: Two other pelvic injuries that would be reasonable to viva on would be a type APC III injury and a vertical shear (VC) injury. With an APC III injury there is complete disruption of the anterior and posterior SI ligaments. This is a completely unstable injury with the highest rate of associated vascular injuries and blood loss.

A vertical shear injury is associated with complete disruption of the symphysis, sacrotuberous, sacrospinous and sacroiliac ligaments resulting in extreme instability most commonly in a cephaloposterior direction because of the inclination of the pelvis. There is a high rate of significant neurovascular injury and haemorrhage.

EXAMINER: How would you manage this injury?

CANDIDATE: I would manage this injury according to ATLS® principles. Appropriate attention must be paid to airway management, spinal immobilization, adequacy of ventilation and provision of supplementary oxygenation.

There is a high likelihood of associated injuries. I would put out a trauma call so the patient can be managed with a multidisciplinary team involving general surgeons and anesthetists.

Significant shortening or external rotation of one of the patient's lower extremities on inspection suggests a VS or an open book APC type pelvic injury.

The patient needs aggressive resuscitation, two large bore cannulae in the antecubital fossa, bloods for crossmatching, start IV fluids.

EXAMINER: What IV fluids would you give?

CANDIDATE: I would give crystalloid or normal saline.

EXAMINER: Which one?

CANDIDATE: I would initially give normal saline as a bolus as this provides transient intravascular expansion and further stabilisers the vascular volume by replacing accompanying fluid losses into the interstitial and intracellular spaces.

EXAMINER: Are there any dangers in giving normal saline?

CANDIDATE: There is some concern about loss of first clot.

EXAMINER: Go on. What else?

CANDIDATE: I would follow ATLS® protocol.

COMMENT: Balanced resuscitation – Do not blow the clot. The candidate has hinted at this but should have expanded on their answer instead of letting the examiner control the viva.

EXAMINER: Studies have shown a detrimental effect of large volume of fluids in diluting clotting factors, reducing blood viscosity, clot disruption from increased blood flow, increased risk of ARDS along with GI and cardiac complications.

COMMENT: There is now a trend in giving blood early, thus, avoiding the rapid administration of IV fluids (filler fluid) that has been traditionally promoted by the ATLS® guidelines. This involved giving 2 litres of crystalloids and continuing with packed red blood cells (PRBCs) and fresh frozen plasma (FFP) if there was transient or no response, with the aim of rapid restoration of intravascular volume and vital signs towards normal and achieving normotension.

Duke et al.[18] provided a retrospective analysis of 307 patients admitted to a level 1 trauma centre with penetrating torso injury and a systolic blood pressure below 90 mmHg. Patients were divided into two groups – One receiving standard fluid resuscitation (SFR) and one receiving restricted fluid resuscitation (RFR). The SFR group received more preoperative fluid then the RFR group (2275 ml vs 129 ml) and had a higher intraoperative mortality rate (32% vs 9%) and overall mortality rate (37% vs 21%). This was attributed to the effect of a large volume of fluids in diluting clotting factors and reducing blood viscosity and the increase of blood pressure. RFR was beneficial as it allowed permissive hypotension (systolic blood pressure of 90) until damage control surgery was achieved[19].

The candidate didn't know this info and was trying to second guess what the examiner wanted. This is quite an important new concept which challenges traditional ATLS® teaching. In the setting of uncontrolled hemorrhage, aggressive fluid resuscitation may be harmful, resulting in increased hemorrhagic volume and subsequently greater mortality.

EXAMINER: You would want to give blood early.

COMMENT: **Massive transfusion protocol (1 : 1 : 1).** Candidates should also mention giving FFP and platelets to support clot formation and prevent disseminated intravascular coagulation. The use of activated Factor VII as an adjuvant to massive transfusion is recommended in some situations.

EXAMINER: What do we mean by massive blood loss?

CANDIDATE: Class IV haemorrhage >40% blood volume loss.

COMMENT: This wasn't the answer the examiner was looking for. Major haemorrhage is variously defined as

- Loss of more than 1 blood volume within 24 hours (around 70 ml/kg, >5 litres in a 70-kg adult)
- 50% of total blood volume lost in <3 hours
- Bleeding in excess of 150 ml/min

EXAMINER: So how are you going to manage this patient?

CANDIDATE: A major risk factor for mortality in patients with pelvic ring fractures is hypotension not responsive to fluid resuscitation. The pelvis should be stabilised with a pelvic binder. A chest radiograph (to rule out hemothorax) and a FAST scan (to rule out hemoperitoneum and need for exploratory laparotomy) should be undertaken. If available a detailed trauma CT should ideally be obtained, which may obviate the need for a chest and lateral C-spine radiograph. These can be difficult to obtain out of hours in a District General Hospital but should be available on a 24-hour basis without any difficult in a Level 1 trauma centre.

Once I have eliminated other sources of hemorrhage, the most likely source of bleeding is from the pelvic venous and arterial system or from the cancellous bone at the fracture site. If the patient continues to be unstable despite aggressive fluid resuscitation, they should be taken for pelvic angiography and embolisation. If a patient is hemodynamically stabilised, full imaging (including inlet, outlet, Judet, and CT scan) can be performed. If the pelvic fracture type is unstable (Tile B or C; Young and Burgess APC II, APC III, LC II, LC III, VS), the patient will require operative fixation and can be treated with more definitive stabilisation, such as an external fixator in the interim until transfer to a dedicated Level 1 trauma centre can be arranged.

EXAMINER: Would you apply the external fixation in casualty?

CANDIDATE: I would prefer to apply the external fixator in a more controlled environment such as theatre under II control.

EXAMINER: Why not just leave the pelvic binder in place and not delay transfer to the nearest Level 1 trauma centre?

CANDIDATE: If the Level 1 trauma centre is nearby and there wasn't the available expertise to apply an external fixator at the local hospital there would be some merit in arranging immediate transfer provided the patient had no other sources of bleeding that required urgent laparotomy. However, if the Level 1 trauma centre was 40 miles away, the pelvic binder could become loose in the ambulance journey so it would be advisable to apply an external fixator to the pelvis before transfer. I would personally have a low threshold in applying an EF but also take advice from the on call pelvic surgeons, as I would not want the fixator to compromise any definitive pelvic surgery.

COMMENT: This type of question is not in the standard orthopaedic textbooks and is testing a candidate's practical knowledge and reasoning of real-life dilemmas as an NHS consultant[a].

EXAMINER: How do you rule out an open fracture?

CANDIDATE: It is important to recognise open fractures early they are dangerous injuries with a mortality rate reported in some studies as high as 50%. A rectal examination should be performed during the initial evaluation. Blood in the rectum should raise the level of suspicion for an open injury. Palpable bony spicules within the rectum or vagina may be present indicating an open injury.

Wounds must be adequately debrided and irrigated. Management includes bladder drainage by a cystostomy tube and bowel diversion with a colostomy. The colostomy should be sited away from potential pelvic surgical fixation skin wounds.

EXAMINER: What about bladder or urethral injury?

CANDIDATE: The overall incidence of genitourinary injury associated with a fracture of the pelvis has been variably reported from 4.6% to 13.5%. Men and women are equally likely to sustain an injury to the bladder but damage to the male urethra is more common than the female urethra.

A high-riding prostate may also be detected on rectal examination, indicating the presence of a periurethral or periprosthetic hematoma occurring secondary to genitourinary injury.

Widening of the symphysis pubis and sacroiliac joint may predict bladder injury while fractures of the inferior and superior pubic rami are more commonly associated with urethral injuries.

Where there is suspicion of a urethral or bladder injury a cystourethrogram should be performed.

Traumatic urethral injuries may result in strictures, incontinence, recurrent infection and erectile dysfunction.

EXAMINER: How would you definitely manage this fracture?

CANDIDATE: The patient would be positioned supine on a fluorescent table. I would use a Pfannenstiel incision. I would stabilise the anterior symphyseal distraction with a symphyseal plate and the posterior sacroiliac diastases with sacro-iliac screws. A plate is fixed to the superior surface of the symphysis. The screws are inserted on the anterosuperior surface of the pubis on either side of the symphysis and directed to the full depth of pubis in a posteroinferior direction. Reduction is obtained using large pointed reduction clamps applied to the obturator foramen and closed.

[a] The exam board is attempting to develop questions that test candidates on the practical difficulties of working in the UK NHS system.

Low-energy pelvic fractures

These are common injuries affecting the elderly, frequently after a simple fall:

Pubic rami fractures
Acetabular fractures

Acetabular fractures

Key viva themes – Interpretation of Judet views, description of surgical approaches and anatomy

There are BOA guidelines for the management of acetabular fractures. These injuries are less likely than pelvic fractures to be associated with major haemorrhage or haemodynamic instability. Acetabular fractures may be associated with hip dislocation.

Mechanism – High energy trauma (posterior wall fracture with dashboard injury). Low-energy trauma in elderly.

Initial management – ATLS® principles and resuscitation as necessary. Hip dislocations must be reduced urgently and their subsequent stability assessed and documented. Neurovascular status before and after reduction must be documented. Skeletal traction should be applied. If the hip is not reducible, urgent specialist advice should be sought. A CT scan should be performed within 24 hours of reduction to assess articular congruence and to exclude bone fragments within the joint. Knee joint stability must be assessed as dashboard injuries can be associated with PCL injuries.

Investigation – AP radiographs and Judet views (Iliac and obturator obilques). Assess the following lines to diagnose the type of acetabular fracture: Iliopectineal line, ilioischial line, anterior and posterior walls, acetabular weight-bearing surface and tear drop. Obturator oblique demonstrates posterior wall and anterior column; iliac oblique demonstrates the anterior wall and posterior column. Inlet, outlet and Judet views can be reconstructed from CT scans. However, the argument for obtaining radiographic Judet views preoperatively is to compare against Judet views obtained during acetabular reconstruction to access fracture reduction.

CT provides information on size and position of column fractures, impacted fractures of the acetabular wall, degree of comminution and sacroiliac disruption. Retroperitoneal haematoma and soft-tissue injury may also be evident on CT scan. Look for any femoral head injury or loose bodies

Classification – Letournel and Judet classification describes five elementary (simple) and five associated (complex) types. An associated fracture includes at least two of the elementary forms (Table 26.1).

Definitive treatment – Guidelines recommend that patients requiring surgery have this performed by a specialist surgeon ideally within 5 days from injury but no later than 10 days (as per BOAST 3).

Non-operative management – Limited indications, possible role if severe medical co-mobidities, infection, poor premorbid activity level, severe osteoporosis or presence of severe associated injuries.

Undisplaced fractures, secondary congruence in associated both column fracture and very low transverse or anterior column fractures when most of the weight-bearing surface is intact.

Operative management – Displaced fractures, incongruent hip joint, fractures in the weight-bearing area acetabulum, intra-articular loose bodies, hip instability, unstable fractures

The approach (es) to the fracture depends upon the fracture configuration. Pelvic anatomy is complex and visualisation within the pelvis is limited. The fracture is reduced to ensure articular congruence and the columns are stabilised, often using reconstruction plates that are contoured to fit the shape of the pelvis.

Anterior column and/or anterior wall – Ilioinguinal approach (be aware of Corona mortis – Anastomosis between external iliac and obturator systems around the superior pubic ramus), or Stoppa approach.

Posterior column and/or posterior wall, transverse fracture – Kocher–Langenbeck approach.

Anterior column with posterior hemitransverse, T fractures, associated both columns – Ilioinguinal ± Kocher–Langenbeck.

Instrumentation includes straight and curved 3.5 locking and non locking. Reconstruction plates that are contoured to fit the shape of the bone are used to stabilise the fractures. Spring plates used for bone fragments too small for screw placement.

Plate bending instruments, specialised retractors (blunt, spoon, pelvic, sciatic nerve), large pelvic reduction forceps, pointed reduction forceps, collinear reduction clamp etc

It is unlikely candidates will be asked specific details about fracture fixation but it is useful to have an idea of the equipment needed for surgery. Default back if pressed AO 4 basic principles (1) Anatomical reduction, (2) stable fixation, (3) early active mobilisation and (4) preservation of blood supply which is just as applicable to pelvic fractures as to any fracture.

Complications – Nerve injury (depends upon surgical approach), vessel injury during surgery, heterotrophic ossification (rate depends on approach), thromboembolism

Table 26.1 Letournel and Judet classification

Elementary (simple)	Associated (complex)
Posterior wall	(Posterior column and posterior wall
Posterior column	Transverse with posterior wall
Anterior wall	Anterior column and posterior hemi-transverse
Anterior column	T-shaped fracture
Transverse	Associated both column

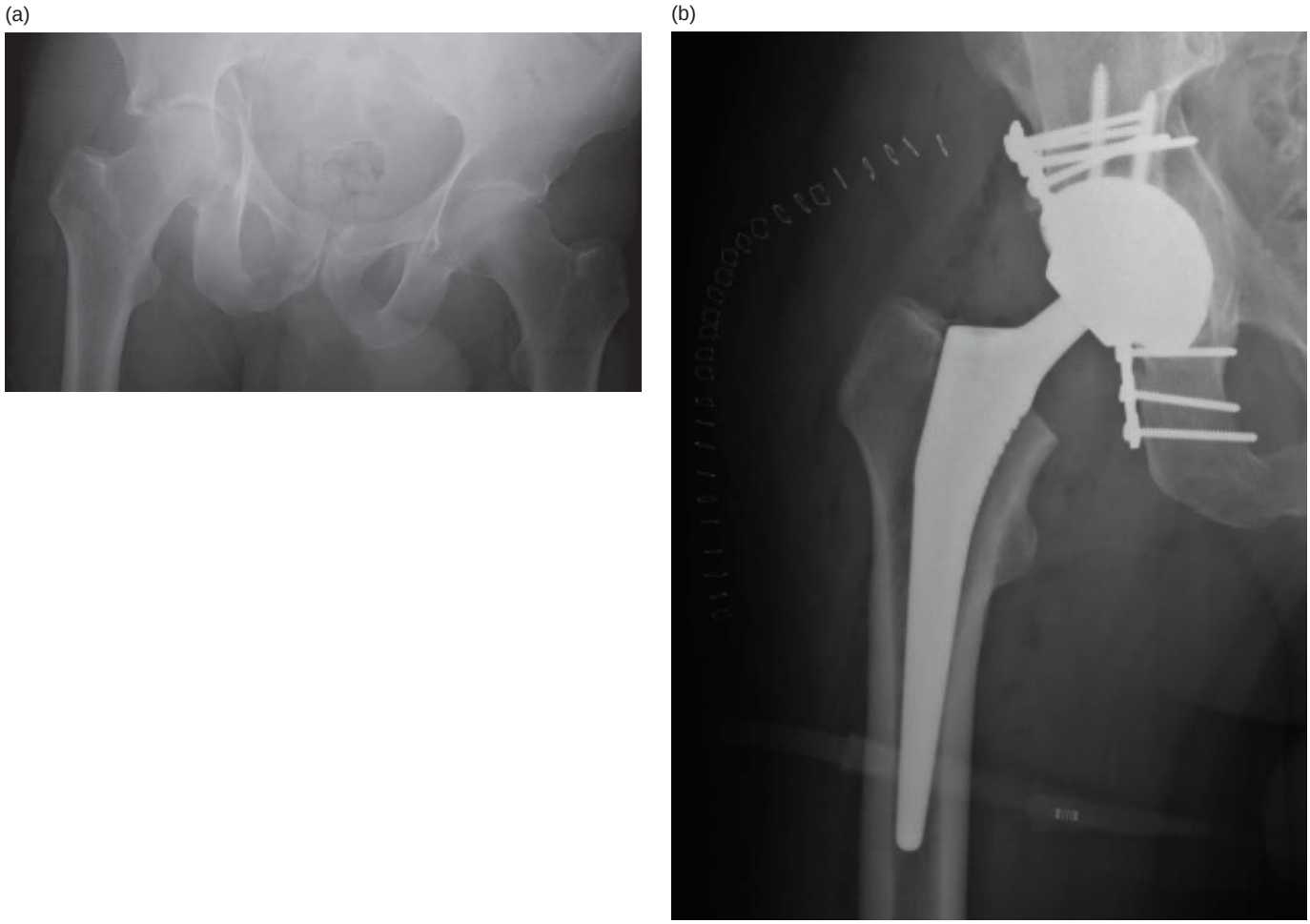

Figure 26.7 (a) Acetabular fracture in an elderly patient. (b) Fracture in Figure 26.7a treated with fixation and THA

(consider IVC filter in high risk cases), wound infection (5%), non-union, mal-union, avascular necrosis (5%), secondary osteoarthritis (20%).

Displaced acetabular fractures in the elderly

Issues – Bone is soft, often associated with fracture fragment impaction and comminution

Options – Non-operative management is a reasonable choice for undisplaced fractures and secondarily congruency associated with both column fractures. Consider THA when fracture has healed at 6 months. Operative management by fracture fixation and total hip arthroplasty in same sitting (Figure 26.7 a and b)

Combined pelvic and acetabular fractures

The initial resuscitation is the same including ATLS® principles and pelvic binder. When physiologically stable, surgery should anatomically reduce and stabilise the acetabular fracture first, followed by stabilisation of the pelvic fracture. If the pelvis is fixed first, any incongruity would lead to incongruity of the acetabular fracture, which is associated with a poor outcome.

Examination corner

EXAMINER: Can you identify the lines on this drawing? (Figure 26.8a)

COMMENT: This is a 30-second first question in a viva topic. You must know this info in order to proceed smoothly in the viva to the next level.(Figure 26.8b).

Spinal trauma

With the FRCS (Tr & Orth) viva component in recent years there has been a conscious move towards including a compulsory spinal elective question for the adult and pathology section. Less definite but still possible is a spine trauma topic in the trauma oral section

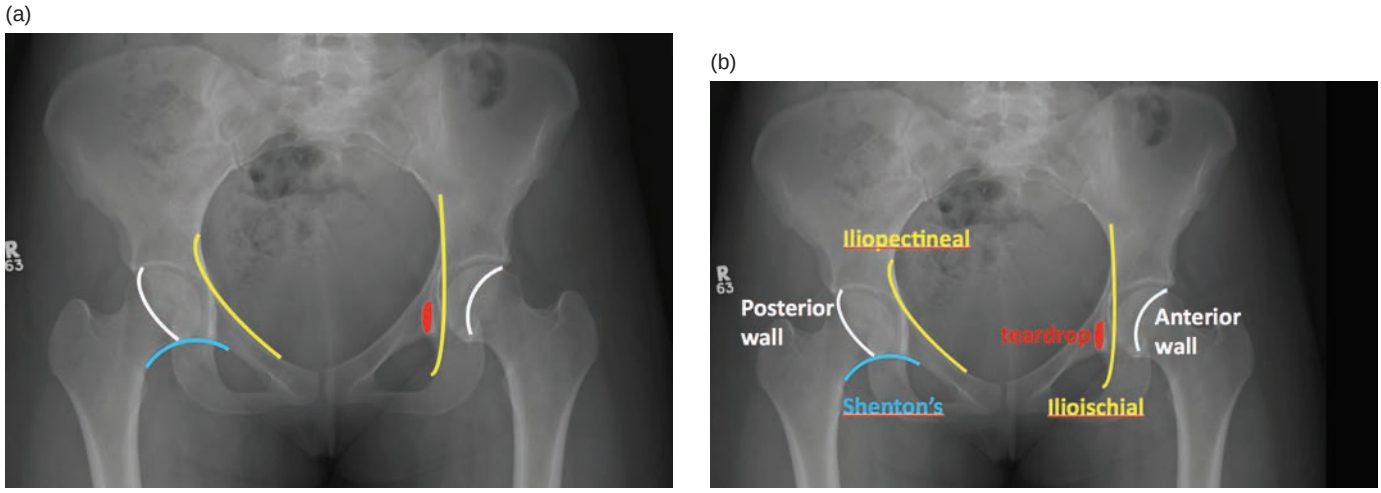

Figure 26.8 (a) AP pelvis radiograph. (b) Radiographic lines on AP pelvis

Table 26.2 Subaxial cervical spine injury classification system

		Points
Morphology		
	No abnormality	0
	Compression	1
	Burst	+1=2
	Distraction	3
	Rotation/translation	4
Disco-ligamentous complex (DLC)		
	Intact	0
	Indeterminate	1
	Disrupted	2
Neurological status		
	Intact	0
	Root injury	1
	Complete cord injury	2
	Incomplete cord injury	3
	Continuous cord compression in setting of neurological deficit	+1

Very occasionally an osteoporotic vertebral compression fracture will be shown in the adult and pathology viva that will then lead on to a general discussion of osteoporosis[b].

This contrasts sharply with the EBOT exam where one 30-minute viva station is dedicated exclusively to spinal topics. In this viva station around 7–8 questions are asked covering elective, trauma and paediatric spinal topics.

[b] In the basic science viva laminated results from a DEXA scan is the usual prop shown to lead into discussing osteoporosis.

Lower cervical spine injuries

The five major classification systems for acute subaxial cervical trauma are: (1) Holdsworth classification[20]; (2) Allen and Ferguson classification[21]; (3) Harris classification[22]; (4) Subaxial Cervical Spine Injury Classification system (SLIC)[23]; and (5) Cervical Spine Injury Severity Score (CSISS)[24].

The older classifications systems have focused on the mechanisms of injury while the newer systems have discarded this in favour of radiological findings and, in the case of Vaccaro et al.[23], neurological status. Comparisons of the classification systems show that there are clear advantages to the system presented by Vaccaro et al.[23] (SLIC scale) because it may be used to guide treatment; however, it has lower reliability and validity compared to the older systems.

Allen and Ferguson classification[21]

This is based on the mechanism of injury, and is divided into six categories: (1) compression–flexion; (2) vertical compression; (3) distraction–flexion; (4) compression–extension; (5) - distraction–extension; and (6) lateral flexion.

Subaxial cervical spine injury classification system (SLIC)[23]

The SLIC system consist of three main components: Injury morphology, disco-ligamentous complex (DLC) and neurological status.

The DLC consists of the anterior and posterior longitudinal ligaments, the intervertebral disc, the facet capsules, interspinous and supraspinous ligaments and the ligamentum flavum. Neurological status is 'an important indicator of the severity of spinal column injury' and 'significant neurological injury' infers a significant force of impact and potential instability to the cervical spine.

If the score is between 1 and 3, the patient does not receive surgery, while for a score $\geq$5 surgery is recommended. Score 4 is equivocal for surgery.

Cervical spine – Uni/bifacetal dislocation

Mechanism of injury – Motor vehicle accident, fall from height and diving accidents. Facet dislocations are classified as distraction–flexion injuries and account for approximately 10% of all subaxial cervical spine fractures

Level – Most common levels for dislocations are C4–C5, C5–C6 and C6–C7. The lower cervical spine is particularly at risk because of its increased mobility (compared to upper thoracic spine), more horizontally oriented and smaller superior facets

Types – One or both inferior facets displace anteriorly over the superior facet or facets of the vertebra below. Perched facets is a subtype of dislocation where there is complete loss of apposition of the articular surfaces, but the tip of the inferior articular process only abuts, without extending past, the superior articular process

Clinical – Unilateral may present with nerve root injury or incomplete neurological deficit. High incidence of cord damage with bilateral facet dislocation

Investigations – Lateral radiographs reveal the amount of anterior vertebral displacement with unifacet dislocation leading to ~25% and bilateral ~50% subluxation. Loss of disc height may indicate retropulsed disc in canal. AP radiograph reveal altered spinous process alignment

MRI is used to detect associated disc herniation and any compression haematoma

Management – Non-operative management of unilateral dislocations more often results in treatment failure, neurological deterioration and persistent pain when compared to surgery. Halo vest immobilization is used until fusion occurs. However, around 50% of patients still demonstrate persistent instability on flexion/extension views requiring surgical fusion

Reduction can be attempted as long as the patient is awake, alert and frequent neurological examinations are possible. This is achieved with Gardner–Wells skull tongs adding sequential weights to the traction cord. The patient is supine and either image intensier views or lateral cervical spine radiographs are taken after each additional load is added. An initial 10 lb is added, weights are increased by 5 lb increments every 20 minutes. After each addition of weight check for any change in neurological status. Various reduction manoeuvres described. For unilateral facet dislocation (UFD) rotate head 30–40° past midline in the direction of the dislocation.

There is controversy as to whether an MRI is needed before attempting closed reduction with many experts believing there is no absolute need for this. The safe answer for the exam is to say you would prefer to have an MRI before reduction if it can be done immediately without delay and you would also discuss the case with the MTC spinal unit for advice.

If there is associated intervertebral disc rupture, facetal fracture or the dislocation does not reduce by traction then, surgical intervention is indicated. If MRI scan shows disc rupture, then, anterior approach is performed first to remove the disc, followed by posterior approach to reduce the dislocation and posterior stabilisation and anterior intervertebral fusion.

If MRI scan shows disc is intact, posterior approach to reduce the dislocation and posterior stabilisation is performed followed by anterior fusion.

Examination corner

This usually involves being shown a radiograph of either a unilateral or bilateral facet subluxation. Bilateral should be fairly obvious so to make the viva difficult a unilateral or perched facet(s) may be shown instead. Management is controversial and complicated the issues being:

- If unilateral does the injury need reducing
- Should a closed reduction be attempted without an MRI scan
- The indications for surgical intervention
- Surgical approach (es) used

Try and keep the discussion simple, straightforward and non-controversial. We have seen the viva given where examiners wanted to discuss controversial areas of management and the relevant evidence-based literature. The carefully prepared candidates avoided this trap by saying that standard practice would be to reduce a unifacet dislocation, obtaining an MRI scan pre-reduction because if there was an associated large disc herniation open surgery would be safer.

Examination

Cervical spine evaluation is part of ATLS® protocol. If a cervical spine injury is suspected or confirmed, repeated neurological examination is essential to assess any progression of neurological compromise.

Local

Bruising, local tenderness, gaps or asymmetric gap between spinous processes.

Neurological

Glasgow Coma Scale (to give an idea of how valid the peripheral neurological examination is), cranial nerves, nerve roots.

Sensory

Touch (crude and fine), vibration, temperature, pain. C5 – Over deltoid, C6 – Lateral aspect of forearm, C7 – Tip of middle finger, C8 – Little finger, T1 – Medial aspect of forearm T2 – Medial aspect of arm, T4 – Nipple area, T10 – Umbilicus, L1 – Groin, L2 – Upper and mid thigh, L3 – Lower thigh and anterior knee, L4 – Medial aspect of lower leg, L5 – First dorsal space, S1 – Over the tendoachilles, S2 – Posterior thigh, S3, 4, 5 – Saddle and perineal area.

Motor

Tone, power (MRC grade). C5 – Deltoid, C6 – Extensor carpi radialis longus and brevis, C7 – Flexor carpi radialis, C8 – Long flexors of fingers, T1 – Intrinsics. L2 – Psoas major (hip

flexion), L3 – Quadriceps, L4 – Tibialis anterior, L5 – Extensor hallucis longus, S1 – Flexor hallucis longus.

Reflex

Tendon reflexes (biceps, triceps, supinator, knee and ankle) and superficial reflexes (abdominal, bulbocavernal and plantar).

Frankel's grade of function

A – Complete motor and sensory loss below level of injury

B – Preservation of sensory but not motor function below level of injury

C – Some motor function preserved below level of injury but mostly < MRC grade 3

D – Motor function preserved below level of injury with most at MRC grade 3 or more

Examine both sides. Level of spinal cord lesion is the lowest level of normal sensory or motor function

E – Normal motor and sensory function

Complete spinal cord injury – No motor and sensory function in the anal and perineal region representing the lowest sacral cord (S4–S5).

Incomplete spinal cord injury – Some sensory or motor function caudal to the injury level and in the S4 and S5 segments.

American Spinal Injury Association (ASIA) classification

ASIA grading is an important predictor of recovery of ambulation after spinal cord injury.

A = Complete (paralysis): No sensory or motor function in sacral segments S4–S5

B = Incomplete (sacral sensory sparing): Sensory, but not motor function below the neurological level and extends through sacral segments S4–S5

C = Incomplete: Motor function is preserved below the neurological level, with most key muscles below the neurological level having muscle grade <3

D = Incomplete: Motor function is preserved below the neurological level, with most key muscles below the neurological level having muscle grade ≥3

E = Normal: Sensory and motor functions are normal

Spinal shock

In the immediate aftermath of a spinal cord injury, there is complete absence of motor, sensory and autonomic function distal to the level of injury. This relates to a physiological, rather than structural disruption. There is loss of muscle tone, and with no autonomic function, hypotension and bradycardia occur. This can last from 24 hours to 72 hours. The return of the bulbocavernosus reflex, marks the end of spinal shock. Later, there is gradual recovery of nerve functions that have not been injured which leads to hyperreflexia and clonus of the affected muscles. The persistence of a complete absence of neurological function below the injury level after return of the bulbocavernosus reflex is a poor prognostic sign for neurological recovery

Neurogenic shock

This is a loss of circulatory blood volume due to loss of sympathetic tone to the peripheral vasculature leading to hypotension and bradycardia. It most commonly occurs following cervical and upper thoracic trauma. Patients may also experience flaccid paralysis and absence of sensation and reflexes below the injuried level

Blood supply to spinal cord

Two posterior spinal arteries and one anterior spinal artery (all branches of vertebral arteries). Radicular arteries are segmental supply from ascending cervical, intercostal, lumbar and sacral arteries. In the thoracic and lumbar regions radicular arteries are on the left side. Artery of Adamkiewicz segmental supply between T8 and L2 on the left side.

Spinal cord anatomy

White matter

- Dorsal column – Cuneate and Gracilis tracts – Gross touch, vibration and pressure
- Lateral column – Ventral and lateral spinothalamic tracts – Pain and temperature
- Anterior column – Corticospinal tracts – Axons of the motor neurons

Grey matter

- Dorsal horn – Sensory neurons
- Intermediate horn – Preganglionic sympathetic or parasympathetic neurons
- Ventral horn – Motor neurons

Incomplete spinal cord syndromes

- Central cord syndrome – Usually hyperextension injury, motor loss more in upper limbs (flaccid paralysis) than lower limbs (spastic paralysis), bladder dysfunction, sacral sparing occurs, good prognosis. Often no bony fracture seen but underlying cervical spondylosis
- Anterior cord syndrome – Flexion injury, motor and pain sensation loss, gross touch and vibration preserved, guarded prognosis
- Posterior cord syndrome-Absent proprioception and vibration sense, with preservation of all motor and other sensory modalities
- Brown–Sequard syndrome – Hemiresection of cord most commonly following penetrating trauma. Motor, gross touch, vibration and pressure loss below the level of injury on same side, pain and temperature loss from one or two levels below on the opposite side. Pain and temperature fibres cross over to the opposite side one or two levels above the level they enter the spinal cord
- Conus medullaris syndrome – All the lumbar and sacral segments are very close to each other behind T12 and L1 vertebrae and any injury at this level can cause a combination of upper and lower motor neuron deficits

with bladder and bowel dysfunction due to injury at conus medullaris
- Cauda equina syndrome – Damage to lumbar and or sacral roots leading to bladder and or bowel dysfunction, perianal sensory loss, loss of reflexes and often asymmetric

Initial cord or nerve root injury due to compression, traction or laceration can be compounded by ischaemia and oedema. Primary care of spinal cord injured patients is to avoid secondary injury due to hypoxia, ischaemia and oedema by maintaining blood pressure, oxygenation, preventing raised intracranial pressure and hypovolaemia.

Management of bony/soft-tissue injury

Initial management – Triple immobilization of cervical spine, endotracheal intubation (if required)

Investigations – Lateral view of cervical spine is part of ATLS® protocol. However, absence of any injuries identified does not rule out cervical spine injury, especially in a multiply injured patient, associated head injury and substance misuse

CT and MRI scans provide information about the bony and soft-tissue injury respectively

Other cervical spine injuries

Atlanto-occipital dissociation

It is often fatal secondary to respiratory arrest caused by injury to lower brain stem. Mechanism of injury is extreme hyperextension with distraction. More common in children as flat articulation between occiput and atlas, increased ligamentous laxity. If patient survives, skull traction is contraindicated. Temporary reduction and stabilisation using halo vest followed by occipitocervical fusion using plates and screw. Survivors usually have severe neurological deficit.

C1 fracture (Jefferson's fracture)

The atlas is a ring of bone that surrounds the spinal cord and the odontoid process. The lateral masses joined by the anterior and posterior arches articulate with the occipital condyles by the superior facets and with C2 by the inferior facets. A complex series of ligaments provides stability.

Burst fracture of the ring of C1. Normally caused by axial loading. There are usually no neurological deficits as the space available for spinal cord is large. Treatment is by Halo vest till fracture heals.

Levine described five variants of C1 fractures (Table 26.3). Fifty per cent of these fractures are associated with other cervical spine fractures, especially odontoid fractures and spondylolithesis of the axis.

Table 26.3 Levine classification of C1 fractures

Classification	Mechanism of injury	Stability
I – Extra-articular fracture of transverse process	Avulsion	Stable may involve vertebral foramen/ artery
II – Isolated posterior arch fracture (28%)	Hyperextension	Stable
III – Isolated anterior arch fracture	Hyperextension, the Dens is forced anteriorly through the arch	Unstable if displaced
IV – Comminuted lateral mass fracture	Axial compression and lateral bending	Unstable
V – Burst fracture, three or more fragments (33%)	Axial compression	Depends on displacement/ integrity of transverse atlantal ligament

Odontoid fracture

Anderson and D'Alonzo classification

Type I – Oblique apical avulsion #, Type II – # base of odontoid, Type III – # extending through the cancellous bone of C2 body.

Type I and III – Non-operative management with Halo vest. Type II – Due to involvement of cortical bone risk of non-union is higher. Hence, internal fixation is preferred for fresh fractures and fusion for delayed presentation.

Classic reference

Anderson LD, D'Alonzo RT. Fractures of the odontoid process of the axis. *J Bone Joint Surg Am*. 1974;56:1663–74.

Fractures of the cervical spine may lead to pain and disability from non-union or may result in devastating neurological complications or even death if they displace. The authors present their classification of *Odontoid Peg* (Dens) fractures based on a review of 49 patients. Fractures are classified into three types based on the location of the fracture. This classic article identifies a high rate of non-union in type 2 fractures (junction odontoid process with body C2) that are managed conservatively. The classification into three types predicts prognosis and guides treatment. Despite being published over 50 years ago it remains the definitive classification system used for this fracture.

Spondylolisthesis of C2 (Hangman's fracture)

Fracture of the pars articularis of C2 and disruption of the C2–C3 junction. Mechanism of injury includes motor vehicle accidents, diving and falls. Hanging lesion is hyperextension and distraction but most injuries are caused by a combination of flexion, extension and axial loading.

The incidence of spinal cord injury is low with types I and II and high with type III.

Levine and Edwards classification

- Type I: Minimally displaced with no angulation; translation<3 mm; stable
- Type II: Significant angulation at C2–C3; translation >3 mm; unstable; C2–C3 disc disrupted. Subclassified into flexion, extension, and listhetic types
- Type IIA: Avulsion of entire C2–C3 intervertebral disc in flexion, leaving the anterior longitudinal ligament intact. Results in severe angulation. No translation; unstable due to flexion–distraction injury
- Type III: Rare; results from initial anterior facet dislocation of C2 on C3 followed by extension injury fracturing the neural arch. Results in severe angulation and translation with unilateral or bilateral facet dislocation of C2–C3; unstable

Type II, IIA and III are treated surgically by open reduction and posterior C2–C3 fusion.

Thoracic/lumbar spine

Fracture classification

Historical aspect

Watson–Jones first introduced the concept of instability and believed that the integrity of the posterior ligamentous complex was essential for spinal stability. Nicoll further attempted to define the concept of instability using an anatomical classification.

In 1963, Holdsworth reported that although Watson–Jones had previously published good results using extension bracing for thoracolumbar fractures he could not replicate these results[25]. He went on to expand the previous classification system proposed by Nicoll[26]. Neither reduction nor maintenance of position could be achieved by extension casting. He described a two-column theory emphasizing the importance of the posterior ligamentous complex for spinal stability. He observed that fractures involving both anterior and posterior columns of the spine were less stable. In 1983 Denis reported that this two-column theory was not compatible with biomechanical reports, the experience of scoliosis surgeons or experiments in the anatomy dissecting room. Denis suggested that unstable injuries (both neurological and mechanical) could occur with an intact posterior ligamentous complex and, furthermore, that an injured posterior ligamentous complex didn't necessarily lead to instability. Denis defined a middle column and proposed his three-column theory. The anterior column is made up of the ALL and anterior two-thirds of the vertebral body and disc, the middle column compromises the PLL and posterior one-third of the vertebral body and disc. The posterior column includes the facet joints, pedicles, and supraspinous ligaments. All fractures can be described as one, two or three column. Fractures involving only one column are stable whilst those affecting two or three columns are unstable.

Minor

- Fractures of the transverse process, pars interarticularis or spinous process

Major

1. Compression fractures are classified according to whether they are caused by anterior or lateral flexion. These injuries cause anterior column compression and sometimes associated posterior column distraction
2. Burst fractures are classified according to whether an axial load alone caused the fracture, or whether flexion, rotation, or lateral flexion was also involved. These injuries cause anterior and middle column compression, and may sometimes also produce associated posterior column distraction
3. Flexion with distraction (chance fracture or seat belt fracture). These fractures are defined by their distinctive flexion–disruption mechanism of injury. The anterior column may be intact or compressed, but the middle and posterior columns are distracted
4. Fracture–dislocations are classified according to whether the dislocation involves rotation, shear, or flexion–distraction. These injuries can produce any pattern of column involvement

The Magerl (AO) system[27] classifies fractures on a grid system using the standard AO letter and number system. The system was constructed from an analysis of CT scans and plain radiographs of over 1400 injuries. Three main groups are type A (compression injuries), type B (posterior or anterior distraction injuries) and type C (rotational/shear type injuries). Each are then assigned two numbers to indicate the severity and overall severity increases as the classification moves from A1.1 (most simple) to C3.3 (most severe). This results in a total of 53 patterns.

Vaccaro and the Spinal Trauma Study Group proposed the Thoraco-Lumbar Injury Classification and Severity Score (TLICS)[28]. The TLICS scoring system aids surgical decision-making and takes into account three parameters: (1) injury morphology; (2) neurological status; and the (3) integrity of the posterior ligamentous complex. Each of these features is given a score and surgery recommended if the final score is >4 (Table 26.4).

Table 26.4 Thoraco-Lumbar Injury Classification and Severity Score (TLICS)

Injury morphology	Posterior ligamentous complex	Neurological status
Compression 1	Intact 0	Intact 0
Compression burst 2	Indeterminate 2	Nerve root 2
Translation/ rotation 3	Injured 3	Spinal cord injury (complete) 2
Distraction 4		Spinal cord injury (incomplete) 3 Cauda equina 3

Spinal instability

White and Panjabi defined instability of the thoracolumbar fracture as inability to maintain structural integrity under physiological loads to prevent progression of neurological deficit or pain[29].

The thoracolumbar junction is most commonly involved as it is a transitional zone between the mobile lumbar vertebrae and immobile thoracic vertebrae (due to rib cage).

Mechanisms of injury

Although there are often complex forces occurring at the time of injury, most thoracic and lumbar fractures can be described as one of the following: (1) axial compression; (2) flexion; (3) lateral compression; (4) flexion/rotation; (5) shear; (6) flexion/distraction; and (7) extension.

Compression fractures

Axial loading on a flexion spine. The anterior column fails in compression, middle column remains intact. The posterior column may remain intact or fail in tension, depending on the energy level of the injury. It is easy to miss posterior column injury on radiographs and incorrectly assume a stable injury. If any doubt organize a CT scan

Radiographic features include:

- Anterior wedging of vertebra
- Loss anterior body height and NO loss of posterior body height
- Measure and compare with vertebrae above and below
- Posterior cortical disruption or widening of the pedicles suggests a burst fracture

If the injury involves <50% of anterior column and there is no involvement of posterior column then, non-operative management in an extension brace is sufficient.

Burst fractures

There is involvement of the middle column and often some degree of canal compromise. The extent of collapse and kyphosis as well as the integrity of the posterior column are key determinants of stability. Management continues to be controversial. Two decisions need to be made: (1) Should the patient be treated with or without surgery? (2) If surgery, which approach?

Non-operative care may involve the use of a thoracolumbar sacral orthosis (TLSO), body cast, hyperextension brace or no orthosis. Surgery may involve an anterior approach, posterior approach or combined.

Non-operative management is recommended for burst fractures as long as there is no neurological involvement, significant posterior element disruption or significant deformity (kyphosis over 25–30°). Surgery would be indicated in higher degrees of kyphosis and posterior ligament injuries. In the presence of neurological injury these injuries are generally stabilised. Longer segment posterior fixation reduces the risk of instrumentation failure. Anterior surgery is performed for vertebral body resection, decompression of the anterior spinal canal and anterior fixation with plates.

Flexion distraction injuries

Chance fracture or seat belt fracture. This can be either bony or purely ligamentous. Unstable injury with high risks of neurological deficit and associated abdominal injuries. This is an unstable injury with a risk of developing post traumatic kyphosis, neurological deficit and a poor outcome.

Fracture–dislocations

These injuries have the highest incidence of neurological deficit. Patients who are neurologically intact should be surgically stabilised to prevent neurological injury and allow early mobilization. Patients with complete neurological injuries are also surgically stabilised to improve nursing care and rehab.

Imaging

CT scans best demonstrate the extent of bony injury, comminution, spread of fragments and involvement of the posterior elements.

An MRI scan is the best modality for assessing soft-tissue injury including spinal ligaments, neural elements and intervertebral discs.

Indications for surgical management

Progressive neurological deficit, unstable fracture with progressive deformity, part of multiple injuries, paraplegia (enables early sitting out, nursing care).

Surgical options

Posterior decompression and instrumentation alone is sufficient for flexion distraction type injury. However, an anterior approach provides excellent visualisation for anterior decompression and instrumentation. Anterior instrumentation has better biomechanical strength. In thoracic fractures with retropulsed fragments and spinal cord involvement – Access to retropulsed fragments is not possible via a posterior approach without risk to the spinal cord. The anterior approach provides excellent exposure to the spinal cord and facilitates removal of incarcerated bony fragments.

Classic reference

Denis, F. The three column spine and its significance in the classification of acute thoracolumbar spinal injuries. *Spine*. 1983;8:817–31.

Aided by the relatively recent introduction of CT scanning, Denis presents a novel biomechanical model applicable to thoracolumbar fractures. The model allows the classification of spinal fractures into minor and major types with four different types of major fracture.

The results of previous biomechanical studies (undertaken by other authors) are commented on, the three-column theory proposed, and 412 acute thoracolumbar injuries are retrospectively reviewed and classified

In this paper Denis presents his attempt to classify acute spinal fractures in order to detect those fractures that are at high risk of neurological complications and those in which intervention might prevent secondary deterioration. The paper seeks to integrate, the basic science (biomechanics), the mechanism of injury, the radiological appearance and outcome. It highlights burst fractures are at risk of subsequent neurological deterioration.

Examination corner

Trauma viva 1: Burst fracture

In the trauma viva a lumbar burst fracture is a common topic that regularly crops up. Try and keep your answer 'neutral, down the middle' as the treatment of burst fractures continues to be one of the most controversial areas in spinal trauma despite the high incidence of this injury and extensively published research.

EXAMINER: This is a radiograph of a 17-year-old male after a fall 10 feet from a height (Figure 26.9a). He presents to your local hospital complaining of severe lumbar pain.

CANDIDATE: This is an AP radiograph of the lumbar spine.

There is a loss of height of the vertebral body of L1 with a widening of the interpedicle distance.

EXAMINER: This is the lateral radiograph (Figure 26.9b).

What is your diagnosis?

CANDIDATE: I will be honest I don't know.

(Recovering composure) I think he has a fracture of the lumbar spine.

The posterior wall of the vertebral body has been pushed backwards.

EXAMINER: Do you see any fragments in the canal?

CANDIDATE: Yes.

EXAMINER: These are retropulsed fragments. So this is a what fracture.

CANDIDATE: Burst.

EXAMINER: Burst???

CANDIDATE: Burst.

EXAMINER: What other types of vertebral fracture do you know?

CANDIDATE: There is the AO classification and there is the Denis classification.

COMMENT: The examiner didn't ask for a classification system for lumbar spine fractures but let this pass as the candidate was struggling.

EXAMINER: Tell me about the Dennis classification?

CANDIDATE: The fracture can affect one or two columns. The mechanism of injury is flexion and distraction.

EXAMINER: Flexion–distraction injuries are called Chance fractures and result from compression failure of the anterior column and tension failure of the posterior and middle columns.

CANDIDATE: The mechanism of injury is fracture–dislocation.

EXAMINER: Fracture dislocations are severe injuries with disruption of all three columns of the spine. These are distinguished from the simple Chance fracture by the presence of significant translation.

So you have a patient with a burst fracture how do you go about treating him?

COMMENT: The candidate has lost the thread of the viva question and the examiner has recognised this and brings him back into the topic.

CANDIDATE: I think the fracture needs an operate and you have to decompress the fracture.

EXAMINER: Decompression from posterior or anterior?

CANDIDATE: Anterior.

COMMENT: Surgery is generally recommended if there is an associated neurological deficit. Patients with no neurological deficit can be managed conservatively but patients with a mechanically unstable burst fracture, defined by a disrupted posterior ligamentous complex (PLC), are at high risk for neurological decline without surgical stabilisation. An anterior approach is favoured for surgical decompression of the cord, posterior approach for stabilisation only.

COMMENT: The whole viva question wasn't answered well.

Trauma viva 2

EXAMINER: This is a radiograph of a 17-year-old male after a fall 10 feet from a height (Figure 26.9a). He presents to your local hospital complaining of severe lumbar pain.

CANDIDATE: This is an AP radiograph of the lumbar spine.

There is a loss of height of the vertebral body of L1 with a widening of the interpedicle distance compared to the level above and below. I cannot definitely visualize a fracture so I would want to see a lateral radiograph.

EXAMINER: This is the lateral radiograph (Figure 26.9b).

CANDIDATE: This confirms a burst fracture. There is anterior wedging and posterior body height loss. There is disruption of the posterior vertebral body line and retropulsion of fracture fragments. I cannot see any posterior element fractures or other evidence of a posterior distraction injury.

I would manage this fracture as unstable as the PLC has been disrupted. The patient should be immobilized with full spinal protection. Initial assessment should be with airway, breathing and circulation as per ATLS® guidelines.

A semi-rigid cervical collar should be applied and a long backboard. There must be a search for associated injuries such as abdominal or chest injuries. There may be a neurological deficit, so I would want to perform baseline neurological status and serial assessments. I would want to exclude other spinal trauma and prevent secondary spinal injury.

EXAMINER: Any other investigations you would perform?

CANDIDATE: I would obtain a CT scan. Kyphotic and translation injuries can be visualized on sagittal and coronal reconstructions.

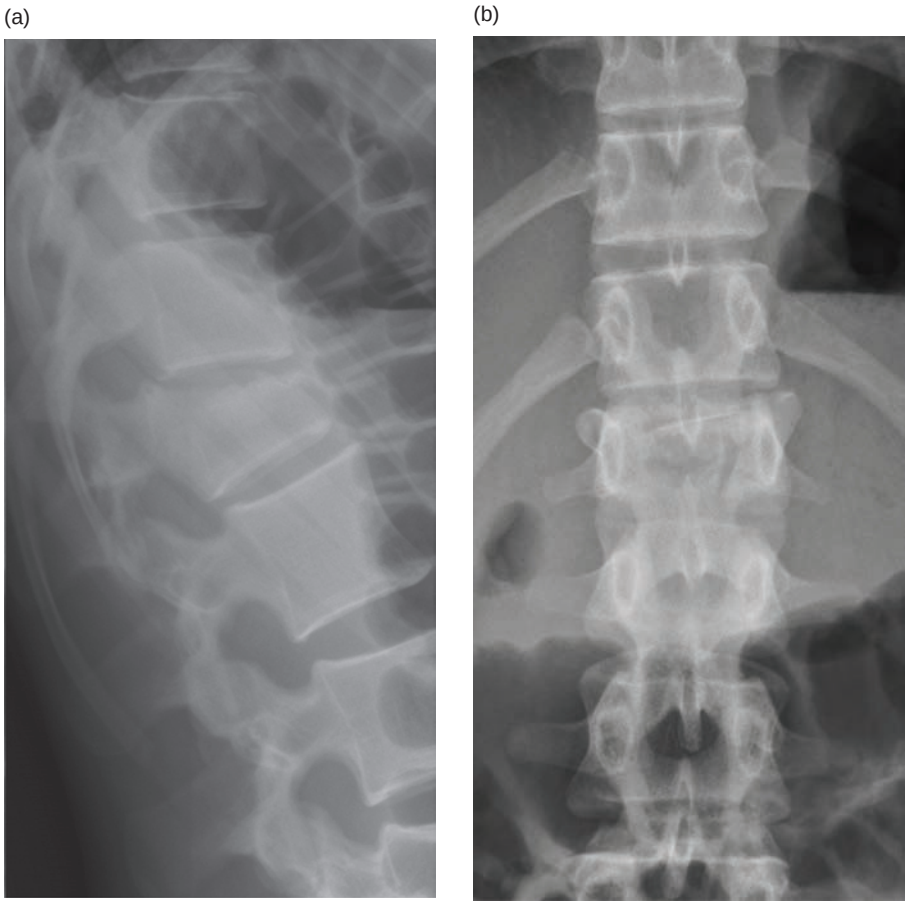

Figure 26.9 AP (a) radiograph and lateral (b) of burst fracture

Vertebral body height, disc spaces, interpedicular distances and interspinous process intervals can be compared between injured and the non-injured levels. The amount of osseous retropulsion can be measured in terms of AP diameter as well as the relative percentage of central canal involvement.

I would also obtain an MRI scan as CT scans have a limited role in visualizing soft-tissue injuries. Disc herniations, epidural hematomas (indirect sign of ligament injury) and spinal cord parenchymal injury are best assessed on MRI. The PLC is frequently torn secondary to osseous retropulsion and on MRI, the ligament may be either discontinous or lifted off the posterior vertebral body with interposed fluid.

EXAMINER: What is the mechanism of such an injury?

CANDIDATE: Burst fractures are the result of axial loading and compression forces to the anterior and middle columns. They typically occur in patients who have either been involved in a motor vehicle accident or sustained a fall from significant height. Burst fractures are more common at the thoracolumbar junction with T12, L1 and L2 the most commonly affected. Neurological deficit is present in up to 40% of patients.

EXAMINER: How are you going to manage this patient?

CANDIDATE: If the patient has no neurological compromise and intact posterior ligament complex then I would treat the fracture conservatively. The anterior vertebral body height should be >50% of the posterior height and kyphotic angulation <25°. More than 50% spinal canal compromise, initially considered a surgical indication, has been debated in patients with intact posterior elements.

Mumford et al.[30] showed that approximately 65% of intraspinal fragments are resorbed and most are completely remodelled within 1 year after the injury. De Klerk et al.[31] also showed reduction of canal compromise by 50% within the first year after non-operative treatment, even in patients with neurological injury.

The indications for operative treatment for a thoracolumbar burst fracture remains controversial, especially in neurologically intact patients. Progressive neurological deterioration is generally accepted as an absolute indication for early surgical intervention. Other strong surgical indications include incomplete neurological injury, >50% spinal canal compromise, >50% anterior vertebral body height loss, more than 25°–35° angle of kyphotic deformity, and multiple noncontiguous spinal injuries. Relative indications include associated nonspinal injuries and patients with nursing or comorbidities such as obesity that make nonoperative treatment extremely difficult.

The main goal of surgery is to decompress the spinal canal and nerve roots, realign the spine, correct and/or prevent the development of post-traumatic kyphotic deformity, and provide long-term stability of the injured spinal segments[32].

The type of surgical procedure is based on the fracture pattern, the severity of neurological injury, and the surgeon's experience.

Accepted methods for operative decompression and stabilisation of thoracolumbar burst fractures include:

- Posterior reduction and instrumented fusion without decompression
- Posterolateral decompression and posterior instrumented fusion
- Anterior decompression and instrumented fusion. Especially indicated for radiographically demonstrated neural compression by bone or disk fragments
- Combined anterior and posterior approach. Used if the PLC is injured in association with a significant anterior column injury (loss of anterior height >50%)

Trauma viva 3

Burst fracture again but most of the viva was spent discussing the classification systems for spinal injuries. There was particular emphasis on discussing the TLICS system[33] and the Magerl (AO) classification[34]. The candidate was also asked about the American Spinal Injury Association (ASIA) classification system.

Sacral fractures

Sacral fractures often occur during high-energy trauma as part of pelvic or spinal injuries. The Denis classification is:

- Zone 1 fractures are lateral to the neural foramina,
- Zone 2 fractures pass through the foramina, and
- Zone 3 fractures are medial to the foramen and involve the spinal canal.

Displaced fractures require specialist input and fixation. Bilateral sacral fractures may lead to a spino-pelvic dissociation. The mechanism of injury is axial loading often as a result of fall from a height. Types of bilateral sacral fractures include 'U' or 'H' type fractures. These fractures often require closed or open (posterior approach) reduction. Stabilisation options include percutaneous iliosacral screws, posterior sacral tension band fixation, lumbopelvic fixation and triangular osteosynthesis. Decompression is indicated in fractures with neurological deficit.

Paediatric conditions

Expect to be questioned in a logical sequence along the following lines.

You will be given laminated radiographs (Figure 26.10a and b). You would be expected to answer the following.

Describe the x-rays – Use accurate clear language – Be prepared to explain deformity in relation to muscle forces/attachments. Explain the importance of maintaining alignment in order to preserve forearm rotation.

What is the initial management and assessment? Analgesia, history, examination, splintage, etc.

Review the neurovascular anatomy – You need a full detailed understanding of clinical anatomy. Show me how you test for the nerves at risk? What muscles are affected?

Be able to discuss the safe administration of analgesia and how you would monitor for compartment syndrome.

Explain consent process

https://www.gov.uk/government/uploads/system/uploads/attachment_data/file/138296/dh_103653__1_.pdf

This is an excellent and comprehensive review of the consent process as published by the Department of Health. You may be simply asked who is able to seek consent.

'The clinician providing the treatment or investigation is responsible for ensuring that the person has given valid consent before treatment begins, although the consultant responsible for the person's care will remain ultimately responsible for the quality of medical care provided. The GMC guidance states that the task of seeking consent may be delegated to another person, as long as they are suitably trained and qualified. In particular, they must have sufficient knowledge of the proposed investigation or treatment, and understand the risks involved, in order to be able to provide any information the patient may require.

Review of this document is mandatory for all parts of your elective and trauma practice. The legal position concerning consent and refusal of treatment by those under the age of 18 is different from the position for adults. For the purposes of this guidance 'children' refers to people aged below 16 and 'young people' refers to people aged 16–17. The Children Act 1989 sets out persons who may have parental responsibility. These include:

- The child's mother
- The child's father, if he was married to the mother at the time of birth
- Unmarried fathers, who can acquire parental responsibility in several different ways, depending on when their children were born but please review the above documentation

This area of the law needs constant review and is particularly important in 'blended families' where multiple fathers may have no legal responsibility.

You would be very unlucky to be questioned in such depth and if unsure, you would seek appropriate senior informed advice.

You may as be questioned in relation to radiation protection measures (ALARA principle: As Low As Reasonably Achievable). In order to minimize the potential risks of biological effects associated with radiation, dose limits and guidelines have been established. The ALARA programme and associated work practices further reduces risks by keeping doses well below the limits. Persons operating x-ray equipment should practice dose-reduction methods whenever using the equipment. Basic safety measures include keeping exposure time short, keeping distance from the source large, and using appropriate shielding, such as lead aprons. All users of x-ray equipment must have specific training to maintain safety at work.

Eventually you may now get to answer the question of the bent forearm. Structure your answer in a stepwise progressive manner. Your thought process should be logical and safe with alteration of your surgical plan as necessary.

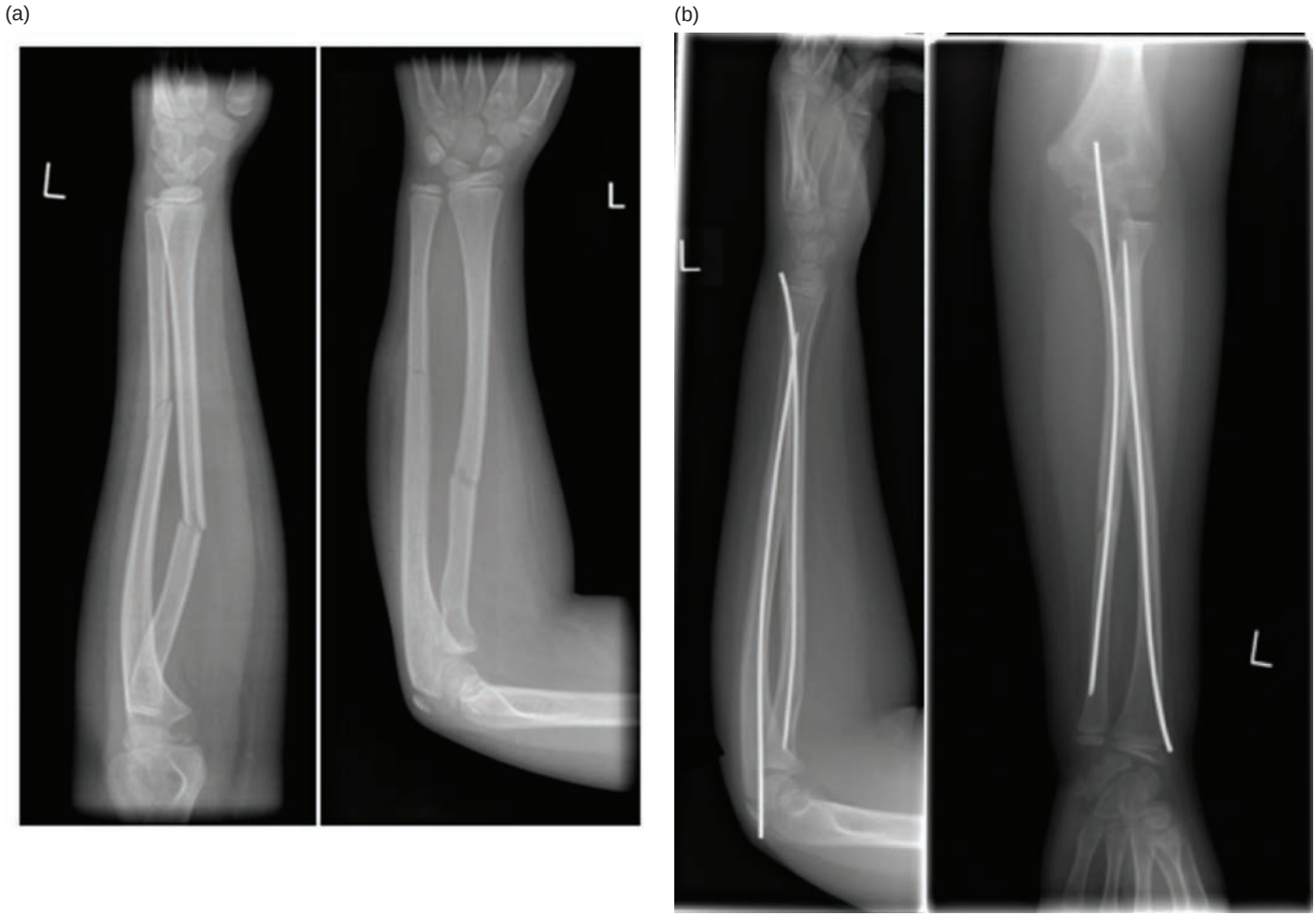

Figure 26.10 AP (a) and lateral (b) radiograph left forearm. Postoperative radiographs TEMS left forearm

Patient positioning – Supine on radiolucent arm board/table with tourniquet applied, should closed reduction and casting fail to maintain a stable reduced fracture configuration.

Theatre layout and image intensifier location – Position yourself so that not only can you have surgical access to efficiently operate but radiology have access to undertake imaging and that you can easily see these images on the radiology VDU. Be prepared to draw an outline diagram of theatre setup with positioning of equipment and personnel.

- Steps of reduction
- Assessment of stability
- Casting material – Differences/pros-cons
- Cast index – Explain as predictor of loss of reduction – Mention fracture/surgeon factors

Be prepared to sensibly explain your

- Follow up
- If there is secondary loss of position – What now?
- Open reduction vs flexible nails
  - Pros/cons of each procedure
  - Approaches
  - Technique – Talk me through how you would do it?
  - What size of flexible nails would you use?
  - Postop management – Cast, short arm vs above elbow?
  - Removal of metal work

There are multiple paediatric fracture cases that can be discussed, but an extensive knowledge of supracondylar elbow fractures, with and without neurovascular compromise and the subsequent timing of surgery is a frequent question. The cases you will be examined upon are those that you will deal with frequently and those cases which are likely to have complications or adverse effects, e.g. physeal injuries with growth plate arrest. Can you work out remaining growth from growth charts? Do you need to investigate using CT scans for a physeal bar? And how might you treat the resulting growth deformity? This area of paediatric fracture management is just as important as adult trauma, but trainees' exposure can be limited if there are not sufficient opportunities for such placements in a training programme. Although not trauma, the presentation of a limping child may be discussed. What is a limping child? An abnormal gait pattern usually caused by pain, muscular weakness or bony deformity. There is usually a shortened stance

phase – Antalgic pattern, whereby the patient will hurry off the painful side

How would you assess?

- History
  - Consider age of patient
  - Duration of symptoms
  - Presence of pain
  - Associated features – Unwell, off food, smelly urine
  - Referred pain?
- Exam – Hip/knee/back/groin/abdomen
  - Look, feel move approach
  - Interaction with carers
- Investigations – Bloods/temp/cultures as necessary/ urine/radiology

Differential diagnosis by age

- **0–3 years** Septic arthritis/osteomyelitis, DDH, fracture, NAI
- **3–10 years** Irritable hip, septic arthritis/osteomyelitis, Perthes', fracture
- **10–15 years** SUFE, septic arthritis/osteomyelitis, Perthes', fracture, blood diseases, neoplasia, discitis, inflammatory disease

Kocker's criteria is a useful diagnostic tool when dealing with conditions that you many not deal with frequently

- Fever >38.5°C
- Cannot weight bear
- ESR>40 mm
- WBC $>12 \times 10^9$/l

Probability of septic arthritis

- No factors: <0.2%
- 1 factor: 3%
- 2 factors: 40%
- 3 factors: 93.1%
- 4 factors: 99.6%

You would then be expected to formulate a plan. An ultrasound scan may well tell you that there is an effusion but doesn't add to the diagnostic process, unless facilities exist for aspiration at the same time. It may not be possible to obtain an MRI scan in your facility but you need to be able to discuss the relationship of septic arthritis secondary to osteomyelitis. The examiner may push you in giving antibiotics before a sample of hip fluid can be obtained, but what antibiotics should be administered? Causative organisms of septic arthritis can be related to the age of the patient and possible underlying medical conditions. The predominant causative pathogens in septic arthritis are *Staphylococcus aureus* and *Streptococcus*, accounting for up to 91% of cases. In the elderly, the immunocompromised and in those patients who have had intravascular devices or urinary catheters inserted, infection with a Gram-negative enteric bacillus is more common. A retrospective cohort study by Li et al.[35] looked at the serum WCC, erythrocyte sedimentation rate (ESR) and the synovial fluid WCC in 156 adult and paediatric patients who had undergone arthrocentesis. Of those 10% had septic arthritis confirmed microbiologically, and the remaining 90% had a variety of other inflammatory conditions or no diagnosis confirmed. The authors concluded that of the three tests, the synovial fluid WCC was the most informative. The diagnostic utility of the test was optimal using a threshold of 17 500/mm^3 above which the diagnosis of sepsis could be made with a sensitivity of 83% and a specificity of 67%. The positive likelihood ratio at this level was 2.5 with a negative likelihood ratio of 0.25.

You need to be able to discuss the surgical management of hip drainage/aspiration. Do you know the surgical approach and the steps involved? (Smith–Peterson) Do you use a vertical or bikini skin incision? And do you leave a drain in situ? Have you performed this procedure or at the very least would be able to undertake as necessary, even though most such procedures are undertaken by surgeons with paediatric orthopaedic experience. Septic arthritis in children affects the hip in a third of cases, the knee in a third, and other joints in the remaining third. Septic arthritis can occur at any age in childhood but is most common among infants, toddlers, and children of preschool age. Transient synovitis is a common idiopathic inflammatory condition which presents in a similar manner to the 'do not miss' diagnosis of septic arthritis, but must remain a diagnosis of exclusion. We advise you to obtain paediatric trauma experience, as often these are the more challenging cases that you will be required to deal with.

The following paediatric topics are not an inclusive list but are frequently asked subjects:

- Non-accidental injury
- Simple buckle fractures – Discuss virtual clinic establishment
- What is an acceptable deformity – Discuss remodelling potential in relation to zones of bone
- Open paediatric fracture management
- Supracondylar elbow fracture management
- Assessment/timing/dysvascular/method of management
- Off-ended distal radius – Management – Technique
- Both bone forearm fracture management – Open/ percutaneous/compartment syndrome/single bone fixation, removal of metal ware and when
- Pulled elbow
- Femoral fractures in varying age groups – Flexible/ORIF/ adolescents
- Tibial fractures – Pop/flexiblenails/x-fix/percutaneous plating
- Triplane fractures – Diagnosis/management/classification
- SUFE – Diagnosis, associations, evaluation, contralateral fixation, management – Pinning in situ/osteotomy, use of traction prior to surgery, risk factors for poor results
- Damage control orthopaedics – Polytrauma paediatric cases
- Spinal/head injuries/vascular/abdominal injuries/respiratory / CV/fluids IV access/splintage/head and neck trauma

Miscellaneous conditions

Compartment syndrome

Definition (What is it?)

A condition characterized by raised intracompartmental pressure within a closed fascio-osseous compartment, that if left untreated will result in muscle necrosis and cell death or permanent damage to nerve in the compartment.

Causes

It can result from tight dressings or casts, long bone fractures (closed or open), burns, haemorrhage into compartment, soft-tissue injuries, electrocution, infection, arterial injury, snake bite and clotting disorders. In orthopaedic trauma the majority of cases will be in young males who have suffered a closed tibial fracture.

Pathophysiology

Normal tissue pressure ranges from 0 mmHg to 4 mmHg. The gradient between tissue pressure and diastolic pressure allows tissue perfusion to occur. When the tissue pressure rises to near diastolic pressure, tissue perfusion will be impaired or cease. Ischaemia of <4 hours is usually reversible, but after 8 hours the outlook is guarded.

Tight compartments → increased interstitial pressure → reduced venous outflow → further increasing interstitial pressure (vicious cycle) → critical interstitial pressure is reached → cellular level hypoxia due to reduced inflow and outflow.

Diagnosis

Relying on pain, pallor, paralysis, pulselessness and paraesthesia is negligent, as these are associated with established compartment syndrome and effective treatment is often too late. The earliest clinical sign is pain out of proportion to the injury and pain on passive stretch of the affected compartment. However, in unconscious patients objective measures are required and continuous compartment pressure monitoring should be utilized when available. This requires the use of a slit catheter placed within 5 cm of the fracture site. The difference between the compartment pressure and the diastolic pressure (ΔP) is the most important determinant and values of 30 mmHg or less are diagnostic.

What is your unit practice and why? Be able to discuss setting up arterial line slit catheter in an unconscious patient.

Management. When associated with fractures, do you fix the fracture first then perform fasciotomy or not and why?

Remember to remove all constricting bandages or casts while waiting to get the patient to theatre.

- Do you use tourniquet or not?
- Explain you set up and exact surgical procedure
- Structures at risk list
- How do you assess muscle viability?
  - Colour/consistency/contractibility/capacity to bleed when cut
- When have you performed fasciotomies?

Forearm – three compartments (superficial flexor, deep flexor, extensor)

Typically an anterior Henry approach is utilized to release the superficial compartment, which is easily identified, before fascia over the deep flexors is released, care is need to release all muscles in the deep compartment. A separate posterior Thompson approach is the utilized to attend to the extensor compartment. The medial nerve and ulnar nerve should be released and covered by skin post release.

Tibia – 4 compartments (anterior, lateral/peroneal, superficial posterior, deep posterior)

Standard 2-incision fasciotomy – The anterior and lateral compartments are the easier to release with a laterally based incision, and either two incisions on the lateral fascia of both compartments, or release of the anterior compartment and then release of the lateral compartment via the anterior compartment, care being taken with the superficial peroneal nerve in the distal one-third of the tibia.

The posterior compartments are released by an incision approximately 2 cm posterior to the medial tibial border. This gives access to the fascia over gastrocnemius, which is released, before the deep compartment is identified distally, and the fascial attachment of soleus to the tibia are released. This then allows access to carefully release the fascia over flexor digitorum and the remaining deep compartment muscles.

Be able to discuss treatment of compartment syndrome in the thigh. Again, cross-sectional anatomical diagrams are to be expected.

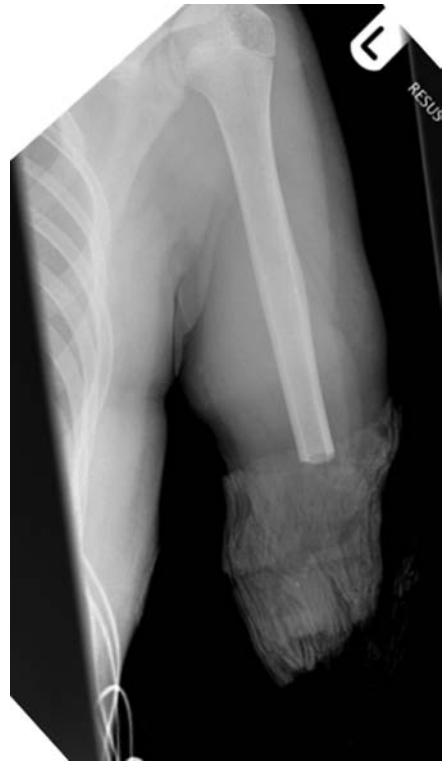

Figure 26.11 AP x-ray of transhumeral amputation

Is there any evidence to suggest a delay to healing post tibial fractures associated with compartment syndrome?

Differentiate between compartment syndrome and a crush syndrome

Crush syndrome. Reported in World War II blitz secondary to entrapment under rubble. Essentially crush syndrome is a reperfusion injury secondary to traumatic rhabdomyalysis causing acute renal failure. Seen in earthquakes, mine cave ins, terrorist acts, e.g. 9/11.

Treatment is supportive prior to removal of the crushing substances. Toxins released from direct cell death, ischaemia and vascular compromise results in electro cardiac events, renal failure and systemic/multiple organ failure. Haemodialysis, and inotophic support is often required.

Traumatic amputations

Although rare (Figure 26.11), a structured management plan needs to be delivered to the examiners. Remember to fall back onto first principles and a saving life approach (ATLS®).

History

- Timing of injury
- Type and location of amputation
- Number of digits involved
- Preservation of amputated tissue
- Associated injury
- Past medial history

Examination

Stump examined for: Zone of injury/tissue viability/supporting tissue structures/contamination

Amputated portion inspected: Segmental injury/bone and soft-tissue envelop/contamination

Indications for reimplantation

Primary: Thumb/multiple digits/wrist level or proximal to wrist/almost all parts in children

Relative: Individual digits distal to the insertion of flexor digitorum superficialis (FDS) (Zone I)

Contraindications

Primary: Single digit proximal to FDS insertion (Zone II)/mangled limb or crush injury/segmental amputation/prolonged ischemia time

Relative: Medically unstable patient/disabling psychiatric illness/tissue contamination

Transport of amputated tissue

Any salvageable tissue should be transported with the patient to hospital. Keep amputated tissue wrapped in moist gauze in isotonic solution. Place in sealed plastic bag and place in ice water (avoid direct ice or dry ice). Wrap, cover and compress stump with moistened gauze

Replantation times

Proximal to carpus	Distal to carpus (digit)
Warm ischemia time <6 hours	Warm ischaemia time <12 hours
Cold ischemia time <12 hours	Cold ischemia time <24 hours

Operative sequence of replantation – BEFAVNS: Bone/extensor tendons/flexor tendon(s)/arteries/veins/nerve/skin)

In reality this topic is out with most people's clinical experience, but something which is encountered in the exam.

We hope that this chapter has given you a better understanding of the trauma oral which can cover all aspects of adult/paediatric trauma management both clinical and organisational as well as related basic science topics. The variety and exposure of trauma management that requires constant patient re-evaluation from initial care to postoperative management and rehabilitation, is what makes this our main specialist clinical area of interest.

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Chapter 27

Upper limb trauma oral core topics

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Sternoclavicular joint dislocation

Mechanism

Usually high-energy trauma (road traffic accident (RTA), contact sports), direct/indirect impact

Assessment

Examination

Localized swelling, bruising and deformity (position of medial clavicle in relation to sternum according to type of dislocation).

Imaging

'Serendipity' radiograph views or CT scan (better) to confirm diagnosis.

Classification

- **Anterior**: Most common, usually asymptomatic despite being unstable
- **Posterior**: 30%, may compress posterior structures causing dysphagia, dyspnoea and sensation of fullness in the throat
- **Pseudodislocation**: Rare fracture mistaken for dislocation. Salter–Harris type I or II in patients <25 years who have an open medial clavicular epiphysis

Management

Non-operative

Sprains, subluxations and the majority of anterior and posterior dislocations. Only treatment for symptoms (pain, swelling) is warranted.

Operative

Acute dislocation: Anterior and posterior closed reductions are performed under general anaesthesia in theatre with a thoracic surgeon on standby. The patient is supine with arm at edge of table. Anterior dislocation is reduced using longitudinal traction of the arm with the shoulder abducted whilst applying direct posterior pressure over medial clavicle. These are usually unstable reductions, but function returns eventually despite this, so either accept the deformity or excise the medial clavicle. Posterior dislocation requires a sandbag between the scapulae, longitudinal traction of the arm with the shoulder extended and abducted (usually snaps back and stable). Should this be unsuccessful, bone forceps can be used to grasp the medial clavicle and pull anteriorly, or failing that cautious open reduction. Once reduced, these are usually stable. A figure-of-8 bandage to brace back shoulders is worn for 3 weeks

Chronic reducible dislocation: Reduction with reconstruction of costoclavicular (CC) ligaments with thoracic surgeon on standby

Chronic irreducible dislocation: Resection of medial clavicle with preservation (<15 mm resection) or reconstruction (if torn) of CC ligaments. Thoracic surgeon on standby

Complications: Infection, blood vessel injury (thoracic surgeon) and cosmetic deformity

Clavicle shaft fracture

Mechanism

Direct trauma or indirect injury from fall onto an outstretched hand.

Assessment

Examination

Localized swelling, bruising and deformity. Assess skin integrity for tenting and blanching. Open or comminuted fractures are associated with high rates of pulmonary and head injuries. Other complications include neurovascular injury. Assess for scapula tenderness (floating shoulder).

Imaging

Anteroposterior (AP) and 10–15° cephalad radiograph views of the clavicle, including the acromioclavicular (AC) joint and sternoclavicular (SC) joint.

Classification

Anatomical according to thirds (Allman):

- Middle: 80%
- Lateral: 15%
- Medial: 5%

Management

Non-operative

The majority of middle third fractures treated in a sling for 7–10 days, with early range of motion exercises. A 'figure-of-8' provides no advantage in outcomes. Serial radiographs to monitor union. Although difficult to predict which will go into non-union, the risk it may be higher in fractures that are displaced, shortened >2 cm, comminuted, in females and the elderly.

Complications: Non-union (open reduction with internal fixation (ORIF) with compression plate and bone graft – autologous cancellous or tricortical for shortening/comminution, with good results), symptomatic mal-union (osteotomy in young active patients) and reduced shoulder strength and endurance (15% of middle third fractures, if shortened and comminuted).

Operative

Indications

- **Absolute:** Skin under tension, skin viability threatened
- **Relative:** Displacement, severe shortening, open fracture, neurovascular deficit, polytrauma and ipsilateral glenoid neck fracture
- **ORIF:** Anatomical dynamic compression or locking plate. Superior plating has better biomechanical strength but more prominent and may require removal of metalwork, compared to anterior
- **IM fixation:** Percutaneous insertion (e.g. Rockwood pin, Hagie pin and Knowles pin), but higher rates of metalwork irritation and complications
- **Kirschner-wires (K-wires) and Steinman pins:** Can migrate and so must be avoided
- **Outcomes:** ORIF results in a faster time to union (16 vs 28 weeks) and less non-unions (2% vs 7%) compared to non-operative treatment. Patient satisfaction and outcomes are better with ORIF at 1 year postoperatively, although there may be hardware problems requiring removal[1]. However, a recent Cochrane review[2] concluded that there is limited randomised evidence on whether to manage acute middle-third clavicle fractures operatively or non-operatively. They recommended an individualized treatment approach based on risks, benefits and patient preference
- **Complications:** Infection, subclavian vein injury, brachial plexus injury, supraclavicular cutaneous nerve injury (numbness distal to scar), pneumothorax, non-union, hardware intolerance (30% removal rate with superior plates)

Lateral clavicle fracture

Mechanism

Usually a direct injury to the shoulder (e.g. contact sports).

Assessment

Examination

Localized swelling, bruising and deformity. Assess overlying skin integrity (tenting and blanching), neurovascular status and scapula for tenderness (floating shoulder).

Imaging

AP and 10–15° cephalad radiograph views.

Classification (Neer)

- Type I – Fracture lateral to CC ligaments (trapezoid, conoid) or interligamentous with minimal displacement (CC, AC). Stable
- Type IIA – Fracture medial to CC ligaments (conoid/ trapezoid ligaments attached to distal fragment). Medial clavicle unstable
- Type IIB – Fracture either inbetween (conoid torn and intact trapezoid attached to distal fragment) or lateral (both torn) to CC ligaments. Medial clavicle unstable
- Type III – Intra-articular fracture Involving AC joint. Intact CC ligaments and stable
- Type IV – Periosteal sleeve avulsion/ physeal fracture in skeletally immature patients. Intact CC ligaments attached to periosteum and stable
- Type V – Comminuted fracture with intact CC ligaments attached to comminuted fragment. Medial clavicle unstable

Management

Non-operative

In a sling supporting the elbow, if minimally displaced, extra-articular and stable (type I/II).

Type III non-operatively initially with delayed AC joint excision should post-traumatic AC joint osteoarthritis occur.

Paediatric distal clavicle fractures (type IV).

Operative

Indications

Absolute: Skin under tension, skin viability threatened

Relative: Displaced (non-union rate of up to 56% of type IIA and 30–45% of type IIB fractures), extension into AC joint and unstable fractures. However, many non-unions are asymptomatic and require no intervention

ORIF: Anatomical lateral clavicle locking plate – Requires a large enough lateral fragment to gain purchase with screws (minimum of 2–3 bicortical). Hook plate – If the lateral fragment is too small and invariably requires removal of metalwork due to subacromial impingement. Tightrope technique (e.g. 'Surgilig') using an open or arthroscopic technique

Non-union: If symptomatic, options are ORIF with bone grafting or excision of the lateral fragment (Mumford

procedure) which may also require stabilisation of the medial clavicle if unstable (like AC joint stabilisation)
Outcomes: Hook plate had significantly lower complications and hardware problems, as well as better return to work and functional activity compared to tension-band wiring[3]
Complications: Non-union (11.5% of fractures treated non-operatively[4]), infection, metalwork failure/cut out, need for removal and stiffness

Acriomioclavicular joint dislocation

Mechanism

Direct injury to the point of the shoulder (e.g. rugby players and motorcyclists), or an indirect injury (e.g. fall onto an outstretched hand).

Assessment

Examination

Localized swelling, bruising and deformity. Assess integrity of the skin and scapula (floating shoulder).

Imaging

AP and 10–15° cephalad (Zanca) radiograph views. Bilateral AC joint stress radiographs (holding a weight) if diagnosis in doubt.

Classification (Rockwood)

Type I – Sprain of AC joint ligaments (AC and CC ligaments intact)
Type II – Rupture of AC ligaments and sprain of CC ligaments
Type III – Rupture of AC and CC ligaments with <100% displacement
Type IV – Posterior displacement into or through trapezius (rupture of AC and CC ligaments)
Type V – Detachment of trapezius and deltoid (rupture of AC and CC ligaments, separating clavicle and acromion) with >100% displacement
Type VI – Inferior displacement to coracoid process and posterior to the conjoined tendon (rupture of AC and CC ligaments)

Management

Non-operative

Sling and 7–10 days of immobilization/rest (type I/II). Type III also treated non-operatively in the majority of cases, unless occupation requires significant overhead activity (or sports throwers).

Operative

Indications

Absolute: Skin under tension, skin viability threatened
Relative: Reduction and repair of AC joint in some type III, and all type IV–VI injuries.

Acute: Hook plate (rests in subacromial space and limits abduction to 90°, with inevitable need for removal), Bosworth procedure or open/arthroscopic CC ligament reconstruction (e.g. Endobutton, Surgilig).
Chronic: Weaver–Dunn procedure, open CC ligament reconstruction (Surgilig, Endobutton).
Complications: Infection, bleeding, nerve damage, pneumothorax, AC joint osteoarthritis, shoulder stiffness, fractures coracoid process and clavicle, osteolysis lateral clavicle and persistent AC joint instability.
Outcomes: A meta-analysis comparing operative and non-operative treatment of type III injuries showed better cosmetic outcomes but greater sick leave with operative treatment without any difference in strength, pain, throwing ability and AC joint osteoarthritis, although there are a lack of well-designed studies to identify the optimum treatment[5].

Scapula fracture

Mechanism

Usually a significant mechanism of direct injury (e.g. RTA), although may be indirect (e.g. fall onto an outstretched hand).

Assessment

Examination

Associated commonly with head injury, pulmonary injury, rib fracture, proximal humerus/ clavicle fracture (floating shoulder), brachial plexus injury, vascular (axillary artery) injury, spine fracture and pelvic injury.

Imaging

Radiographs: AP and lateral shoulder views. Chest radiographs to exclude a pneumothorax.
CT scan: For intra-articular glenoid fractures.

Classification

Scapula fractures (Zdravkovic and Damholt)

Type I – Body
Type II – Coracoid and acromion
Type III – Neck and glenoid

Glenoid fractures (Ideberg)

Type I – Anterior avulsion fracture
Type II – Transverse/oblique fracture, inferior glenoid free
Type III – Upper third of glenoid and coracoid
Type IV – Horizontal glenoid through body
Type V – Combination of types II–IV

Management

Non-operative

Preferred option for most fractures

Scapula body: Sling to rest/immobilize for 7–10 days, then early range of motion exercises

Glenoid: Undisplaced and <25% of articular surface involvement, small fragments and no humeral head instability – Sling to rest/immobilize for 7–10 days, then early range of motion exercises. Glenoid neck predominantly all managed non-operatively

Operative

Scapula: Large, displaced and unstable fragments (e.g. glenoid with clavicle fracture) with ORIF using precontoured locking plates. The posterior (Judet) approach is most common using an internervous plane between infraspinatus (suprascapular nerve) and teres minor (axillary nerve)

Glenoid: Intra-articular fractures displaced >2 mm or significantly displaced extra-articular fractures with ORIF. An anterior approach may also be used depending on the type of fracture and site of fragments

Glenoid neck: Anterior translation or medial displacement of glenoid neck and humeral head, with ORIF. Displaced glenoid neck fracture with ipsilateral clavicle ('floating shoulder) usually requires ORIF, but the stability of the superior shoulder suspensory complex (SSSC) should be considered

Complications: Suprascapular nerve and artery (when retracting infraspinatus) in posterior approach

Scapulothoracic dissociation

Mechanism

Significant mechanism of injury to the arm, shoulder and chest wall.

Assessment

Examination

Life-threatening injury with a 10% mortality rate due to associated chest wall (heart, lung) injury. Specifically, there may be avulsion of brachial plexus, injury to subclavian or axillary artery or AC joint/sternoclavicular dislocation and clavicle fractures. High suspicion of scapulothoracic dissociation when neurological and/or vascular compromise.

Imaging

Radiographs: AP and lateral shoulder views. Chest radiograph with >1 cm lateral displacement of scapula

Angiography: In haemodynamically stable patients

Management

Non-operative

Haemodynamically stable patients with vascular injury but good collateral blood supply around shoulder, may not require any musculoskeletal surgery.

Operative

Haemodynamically unstable patients may need high lateral thoracotomy or midline sternotomy to control bleeding. Severe cases require forequarter amputation.

Shoulder glenohumeral joint dislocation ± fracture

Mechanism

Anterior: Fall onto abducted, externally rotated arm

Posterior: Violent muscle contraction (e.g. tonic clonic seizure, electric shock), or direct injury

Inferior: RTA or sports injury

Assessment

Examination

Traumatic have unilateral, deformed (loss of shoulder contour, asymmetrical anterior axillary fold), painfully reduced range of motion in shoulder held with arm in external rotation (anterior), internal rotation (posterior) or abduction 100–160° (inferior). Assess neurovascular status (damage to axillary nerve, brachial plexus and axillary artery) and rest of limb for ipsilateral injuries. Of note, multidirectional are bilateral, not painful, subluxation anteriorly and posteriorly with ligamentous laxity

Imaging

Radiographs: AP, Y view and axillary (most useful) shoulder radiographs to assess direction of dislocation. If there is too much pain for an axillary view, a *Velpeau* view is useful. Anterior dislocations have an abducted humerus and may have a greater tuberosity fracture. Posterior dislocations can be missed ('light bulb' sign, humerus adducted) and often have associated lesser tuberosity (revere Hill–Sachs) fracture

CT scan: Confirm position and identify associated fractures (e.g. glenoid rim, proximal humerus)

Classification

Anatomical

Anterior, posterior and inferior (luxata erecta).

Management

Closed reduction

Anterior

Multiple closed reduction techniques available under analgesia and sedation:

1. **Kocher**: Elbow at 90° with adduction, traction, external rotation 70–8 until resistance) and medial (internal) rotation (after lifting arm in sagittal plane and bringing hand to contralateral shoulder) (ATEM)
2. **External rotation**: Modification of Kocher's, using first stage of adduction and progressive external rotation
3. **Spaso**: Arm lifted vertically in supine position by the wrist with gentle traction and external shoulder rotation
4. **Matsen's**: Traction–counter-traction with a folded sheet in the axilla and traction in abduction. External and internal rotation to disengage humeral head

General anaesthesia when the above fails (e.g. muscular patients), fracture–dislocations (e.g. greater tuberosity, to avoid fracture propagation), posterior and inferior (50% success) dislocations.

Anterior dislocation may require a sling (in internal rotation) until pain settles down for up to 2 weeks in elderly patients and up to 4 weeks in young patients, although there is no evidence for prolonged use. A suggested approach is to repeat radiographs in 1 week to exclude a recurrent dislocation and start gentle mobilization with physiotherapy for range of motion and rotator cuff strengthening exercises. Formal delayed assessment of stability and cuff (e.g. with ultrasound scanning) is useful.

Posterior

Posterior dislocations should have an attempted closed reduction if the dislocation is acute (<3–6 weeks old). Reduction technique involves 90° flexion and adduction with axial traction on the arm, and direct posterior pressure on the humeral head. For humeral heads locked on the glenoid rim, internal rotation to stretch the posterior capsule and rotator cuff and/or lateral traction of the proximal humerus may help. Once unlocked, external rotate the humerus to reduce. Shoulder stability is then assessed.

Once reduced posterior dislocation can be managed in a similar way to anterior dislocation with a sling for up to 3 weeks if stable in internal rotation. Radiographs should be repeated in 1 week before starting gentle mobilization. In those cases that are unstable in internal rotation but stable in external rotation, immobilization for 3–6 weeks in an external rotation brace (20°), followed by rotator cuff strengthening is required.

Chronic missed posterior shoulder dislocations can be very difficult to reduce beyond 6 months. Despite the cosmetic deformity and loss of rotation, many activities of daily living can be performed as there is little pain and some forward flexion persists. 'Supervised neglect' can, therefore, be used in the elderly with limited functional demands, a functional range of motion and normal contralateral shoulder. In more demanding patients open reduction, with reconstruction of the defect is required (see below).

Complications: Recurrent dislocation, fracture propagation, surgical neck of humerus fracture (especially Kocher method if performed incorrectly), axillary nerve injury, rotator cuff tear (older patients) and recurrence (<25 years of age, high-energy injury, large bone defects and non-compliance).

Open reduction

Indications

Failure of closed reduction (e.g. button holing) or where humeral head is locked onto glenoid rim (acute or chronic).

Approach

For anterior dislocation, a deltopectoral approach (± coracoids osteotomy and re-fixation) is suggested. For posterior dislocation, a deltopectoral approach (preferable), posterior shoulder approach or a combination of the two is suggested.

Additional operative

Anterior

There is controversy on the management of first-time dislocation in a young active patient (initial primary stabilisation vs a 'wait and see' approach) and a Cochrane review found the limited evidence favoured primary surgery for young, male, highly active patients with their first acute traumatic shoulder dislocation, and non-operative treatment for all other patient types[6].

Soft tissue – Anterior labral repair, ± ramplissage of Hill–Sachs defects, in young patients. Open or arthroscopic repair of rotator cuff if significant tear, commonly in those >45 years.

Bony – For those with large glenoid or humeral head bony defects the options are a Latarjet procedure, iliac crest bone graft or shoulder arthroplasty.

Posterior

If the shoulder is unstable with a <25% reverse Hill–Sachs defect then the upper third of the subscapularis tendon is transferred into the defect using non-absorbable transosseus sutures. The patient is immobilized in external rotation for 3–4 weeks. With a 25–50% humeral head defect, open reduction with lesser tuberosity transfer (with attached subscapularis) to the defect (modified McLaughlin's procedure). Other options for defects of this size are rotational osteotomies of the proximal humerus or allo/autograft reconstruction to restore humeral head sphericity. With a >50% defect, hemiarthroplasty or total shoulder arthroplasty if significant glenoid wear should be considered.

Inferior

Unstable inferior dislocations may require capsular reconstruction.

Recurrent shoulder dislocations/instability

Mechanism

Direct or indirect injury. May be atraumatic depending on the type below.

Assessment

Examination

As for shoulder dislocation above but with *Beighton* score (hypermobility), apprehension test, sulcus sign, scapula dyskinesia and drawer test.

Imaging

Radiographs: AP, Y view and axillary (most useful) shoulder views. West Pint view for glenoid rim fracture, Stryker notch view for Hill–Sachs lesion

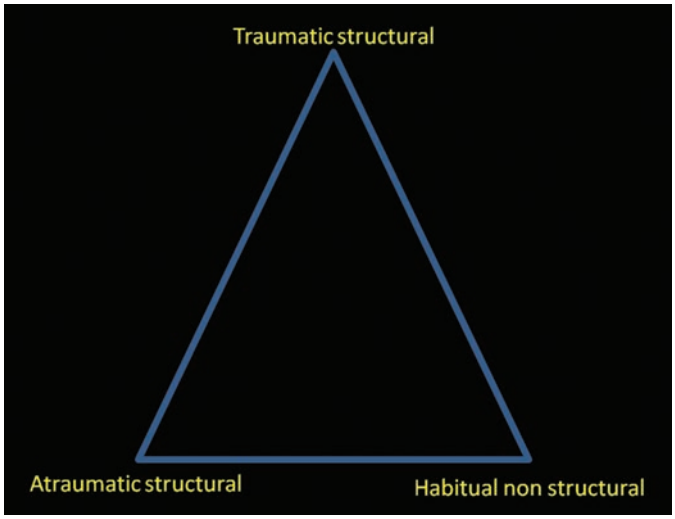

Figure 27.1 Stanmore classification of shoulder instability

MRI: For capsulolabral and superior labrum from anterior to posterior (SLAP) lesions
CT scan: For bony pathology (Hill–Sachs, glenoid, glenoid version and bone stock)

Classification

Matsen

Traumatic (TUBS) – **T**raumatic, **U**nidirectional, **B**ankart lesion treated with **S**urgery. Often a rotator cuff tear in those >45 years
Multidirectional (AMBRI) – **A**traumatic, **M**ultidirectional, **B**ilateral, treated with **R**ehabilitation and **I**nferior capsular shift

Stanmore

Overlap between traumatic structural, atraumatic structural and habitual non-structural (muscle patterning) dislocations, which change with time (Figure 27.1).

Management

Non-operative

Predominant management for multidirectional instability.

Operative

Anterior: Arthroscopic anterior capsulolabral repair (Bankart), unless there is a significant glenoid defect (>25%), which may require coracoid transfer (Bristow–Laterjet), or iliac crest bone block. Inferior capsular shift for multidirectional instability only if prolonged non-operative management unsuccessful
Posterior: Open or arthroscopic repair of posterior defect with posterior capsular shift if no bone defects for chronic recurrent instability and pain on loading of arm in forward flexed position. Bone defects may require bone grafting, modified McLaughlin's procedure (<50% defect, >6 months old) or hemi/total shoulder arthroplasty (>50% defect, >6 months old) depending on bone stock and pattern of osteoarthritis

Proximal humerus fracture

Mechanism

Direct fall onto shoulder or indirect onto outstretched hand.

Assessment

Examination

Painfully reduced range of motion of shoulder, with swelling and bruising which can be significant down the arm. Assess axillary nerve and rest of neurovascular status of arm.

Imaging

Radiographs: AP, Y view, and axillary views of the shoulder to look at fragment number, displacement, angulation and dislocation. Double shadow signifies a head-splitting fracture
CT scan: Useful to assess articular surface and tuberosities, and surgical planning

Classification (Neer)

Parts are humeral head, greater tuberosity, lesser tuberosity and shaft, which are counted when there is displacement of >1 cm or angulation >45°:

- One part – Undisplaced or minimally displaced fracture of the surgical neck, anatomical neck, greater tuberosity or lesser tuberosity
- Two part – Displaced or angulated fracture of the surgical neck, anatomical neck, greater tuberosity or lesser tuberosity
- Three part – Displaced or angulated fracture of greater or lesser tuberosity and articular surface
- Four part – Displaced or angulated fracture of greater tuberosity, lesser tuberosity and articular surface. The articular surface may be split ('head splitting') and require arthroplasty

Management

Non-operative

Sling for comfort and early mobilization for one-part fractures and two-part surgical neck of humerus fractures. Some three- (possibly four-) part fractures with a relatively good position of articular and greater tuberosity fragments may be treated with initial rest in a sling, then early pendular exercises and progressive rehabilitation with regular radiographic evaluation, particularly in those with multiple co-morbidities.

Operative

Indications: Displacement, open fracture, fracture–dislocation and neurovascular deficit

Simple: Suture, tension-band or screw fixation for two-part tuberosity fractures or anchor fixation

ORIF: With a periarticular anatomical locking plate (e.g. Philos) for significantly displaced two-part surgical/anatomical neck fractures, or three- and four-part fractures with tuberosity/rotator cuff repair especially in younger patients

Hemiarthroplasty (e.g. Neer): In three- and four-part fractures with tuberosity/rotator cuff repair in elderly patients, or head-splitting fracture types. Hemiarthroplasty can restore comfort and function in four-part fractures, compared to non-operative management, which often leads to pain, stiffness and shoulder dysfunction. However, they are not good overall with regards to function and motion and should be reserved for low demand/frail patients. In those with a pre-existing massive rotator cuff deficiency, or in elderly, a reverse total shoulder arthroplasty (RSA) is an option. One systematic review showed that RSA had improved forward flexion and functional outcomes over hemiarthroplasty with similar complication rates, although studies with longer follow-up are needed[2]

Postoperative rehabilitation: Range of motion exercises as tolerated

Complications: Avascular necrosis – Patients with the following radiographic Hertel criteria[7] are more likely to develop this

- <8 mm of metaphyseal extension remaining attached to the head
- >2 mm disruption of the medial hinge (shaft displacement)
- basic fracture type (anatomical neck)

A positive predictive value of 97% for avascular necrosis was obtained when the above criteria were combined (short calcar, disrupted hinge and anatomical neck).

Other complications: Infection, neurovascular injury – Axillary nerve, musculocutaneous nerve and cephalic vein, stiffness, metalwork failure – Screw cut out most common after locking plate fixation, non-union – Most common after two-part surgical neck of humerus fracture and mal-union

Outcomes: There has traditionally been insufficient evidence to determine whether operative management produces better outcomes than non-operative management. A recent meta-analysis comparing both options for complex 3- and 4-part fractures found no difference in functional outcomes, although there was a higher rate of additional surgery up to 24 months with operative management[8]

The ProFHER (**Pro**ximal **F**racture of the **H**umerus: **E**valuation by **R**andomization) multicentre RCT examined the cost and clinical effectiveness of surgical vs non-surgical management of acute displaced proximal humeral fractures involving the surgical neck and found no differences in outcomes, complications or secondary surgery. Surgery was, therefore, found not to be cost-effective[9]

Humeral shaft fracture

Mechanism

High-energy direct or bending forces or low-energy rotational forces (e.g. arm wrestling).

Assessment

Examination

Painfully reduced range of motion of shoulder and elbow, with possible deformity, pain and crepitus mid arm. Assess soft-tissue condition, radial nerve and rest of neurovascular status, including compartment syndrome.

Imaging

AP and lateral radiographs of the humerus, including the shoulder and elbow joints.

Classification

No formal classification but based on fracture type.

Holstein–Lewis fracture is a distal third humeral shaft spiral fracture commonly associated with a neurapraxia of the radial nerve (22% incidence). The nerve is in a fixed in position when winding round the lateral intermuscular septum and, therefore, at high risk of injury.

Management

Non-operative

Indications: <20–30°AP angulation, <30° varus/ valgus angulation and <3 cm of shortening as the shoulder compensates for any mal-union

'U-slab': Applied after manipulation acutely for comfort and allowing swelling to settle, although they are difficult to apply well and often lead to further fracture displacement. The radial nerve must be assessed pre- and post change

Functional humeral (Sarmiento) brace: With careful assessment of rotation is applied as soon as possible, with frequent adjustments (avoid fracture distraction) and serial clinical assessment/radiographs (assess fracture position and healing). Of note, standard 'off-the-shelf' braces may not be able to reproduce the excellent results shown by Sarmiento

Operative

Absolute indications: New radial nerve palsy after manipulation

Relative indications: Open fractures, brachial plexus injury, pathological fracture, segmental fractures, polytrauma, floating elbow, neurovascular injury, fracture displacement/distraction (Figure 27.2) and initial radial nerve palsy, obese females

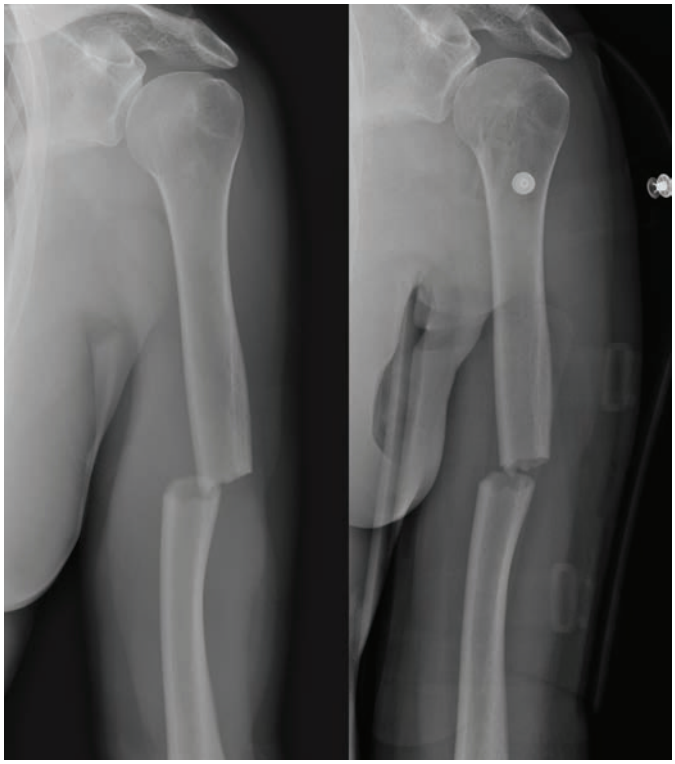

Figure 27.2 Humerus fracture initially treated with brace that ended with distraction at the fracture site

ORIF: Gold standard option which allows radial nerve exploration. The approach varies according to the level of the fracture:

- **Proximal shaft**: Anterolateral approach, which is the deltopectoral approach extended distally (Henry's). The radial nerve can be identified between the brachialis and brachioradialis distally. ORIF using a long proximal periarticular locking plate (e.g. Philos)
- **Middle shaft**: The commonly used posterior approach where the triceps can be split or elevated with a lateral tricipital exposure. It allows radial nerve exploration as it is located medial to the long and lateral heads and 2 cm proximal to the deep head, and as it exits the posterior compartment 10 cm proximal to the radiocapitellar joint by piercing the lateral intermuscular septum. ORIF using lag screws and compression plate with simple fractures, and bridge plate with comminuted fractures
- **Distal shaft**: Posterior approach. ORIF using single posterolateral periarticular anatomical plate, or double medial and lateral plates

ORIF has shown to have a lower rate of delayed union, with similar rates of non-union, infection and radial nerve palsy to intramedullary nailing[10].

Intramedullary nailing: Antegrade for midshaft/proximal, segmental and pathological fractures with distal locking screws. Retrograde for distal fractures. Higher complication rate than for ORIF (see below) and contraindicated in radial nerve palsy

Multiple flexible nails (e.g. Rush pins, Enders nails): Inserted distal to proximal

External fixation (e.g. monolateral or circular frame): For open or infected fractures, as a temporary or definitive treatment

Complications

Radial nerve palsy (5–10%)

Occurring at time of injury: Observe when treating non-operatively as it is most often a neurapraxia with 92% recovering in 3–4 months (firstly with brachioradialis, then ECRL and lastly EPL, then EIP). The mean time to onset of first recovery was at 7.3 weeks and a limited period of waiting had no effect on final recovery[11]

Occurring following closed manipulation: Explore radial nerve. There is some evidence to suggest that even radial nerve palsies occurring after manipulation is not an absolute indication for exploration. Shao et al.[5] reported that there was no significant difference in the recovery rate between primary (occurring at the time of injury) and secondary (occurring after the injury, or as a result of a closed reduction) nerve palsies. The recovery rate in primary nerve palsies was 88.6% and 93.1% in secondary nerve palsies. Be aware of this controversy, but for the exam nerve exploration should be the answer, and only discuss controversy if specifically asked about other options of management

Occurring following open reduction internal fixation (plating): If the nerve was identified and protected during surgery, and the surgeon is confident that it was not under plate, observe. If nerve was not identified during surgery but still sure not under plate, still observe. If any doubt that nerve may have been trapped in fixation then explore

Occurring following IM nailing: Explore

Radial nerve palsy and open fracture: Explore due to higher likelihood of transection

Tardy palsy (e.g. due to callus formation): Explore

Radial nerve palsy treated by observation, that does not recover: Requires baseline nerve conduction studies at 4–6 weeks to confirm type of injury and early referral to a peripheral nerve injuries unit if recovery does not occur by 3 months (nerve fibres regenerate by this time). The timing of nerve exploration and neurolysis/nerve grafting is controversial and suggested to be at 4–6 months if there is no resolution. Splint and maintain passive joint movements to avoid contractures with consideration of early tendon transfers whilst awaiting recovery

Other complications: Infection, nerve injury from distal-locking screws: Radial (lateral to medial), musculocutaneous (anterior to posterior); non-union: Higher risk for transverse fractures and intramedullary nailing with distraction (treat with ORIF with compression plate and bone graft); shoulder

pain: Secondary to rotator cuff damage following intramedullary nailing and iatrogenic distal humerus fracture/elbow motion restriction from retrograde intramedullary nailing

Outcomes: A Cochrane review[12] examining surgical vs non-surgical management of humeral shaft fractures in adults found there is no evidence available from randomised trials stating that one is better or worse than the other. When comparing the outcomes of intramedullary nailing and plate fixation, a Cochrane review found there was insufficient evidence to determine any difference in functional outcome. It found similar union rates but nailing was associated with an increased risk of shoulder problems (pain, impingement, stiffness) and need for metalwork removal[13]. Hence, along with other meta-analyses[10], ORIF may be considered the gold standard for treating humeral shaft fractures

Holstein–Lewis fracture

These are fractures of the distal humerus shaft with associated radial nerve palsy. As the radial nerve winds round the radial groove and enters the anterior compartment of the arm via the lateral intermuscular septum, it is fixed in position. Any fracture of the humerus shaft around this area (Figure 27.3) that is displaced tugs on the radial nerve, which is 'fixed' at the lateral intermuscular septum and may cause a radial nerve palsy. Holstein and Lewis recommended surgical exploration of the nerve.

Several authors believe, however, that the special relationship between this fracture pattern and radial nerve palsy is not as strong as Holstein and Lewis suggested. As such, a spiral fracture pattern of the distal humerus with associated nerve palsy is not an absolute indication for radial nerve exploration. Be aware of this controversy, but for the exam nerve exploration and fracture fixation is recommended.

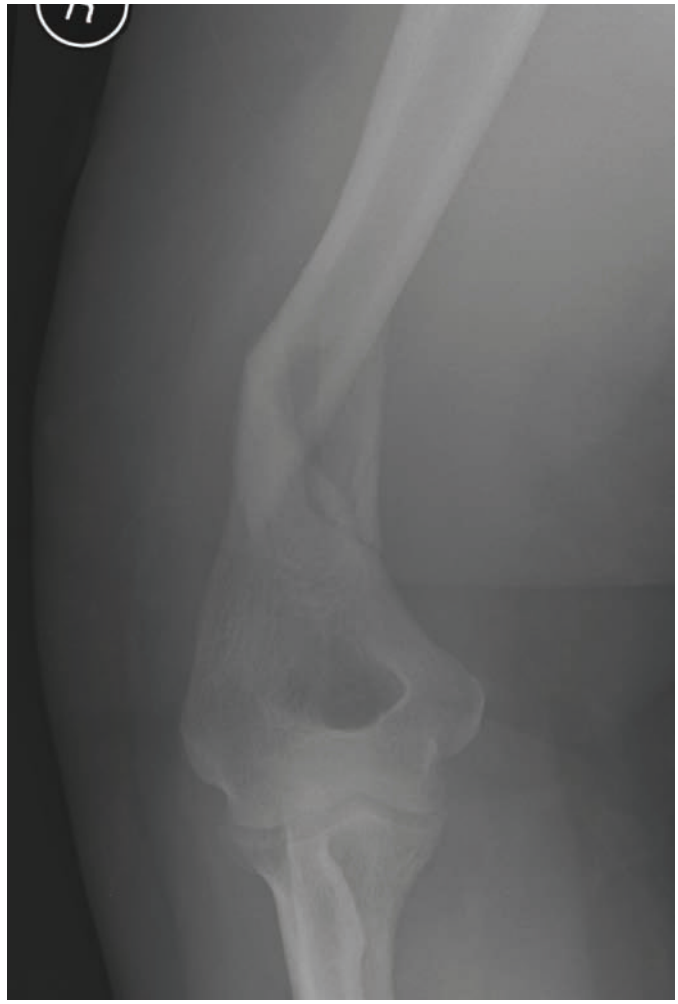

Figure 27.3 Distal humerus fracture in a patient with associated radial nerve palsy

Distal humeral fracture

Mechanism

Indirect fall onto outstretched hand or direct fall onto the elbow.

Assessment

Examination

Swollen, bruised and limited range of motion elbow. Assess soft-tissue and distal neurovascular status.

Imaging

Radiographs: AP and lateral radiographs of the elbow

CT scan: In displaced intra-articular fractures to fragments and plan surgery

Classification

Supracondylar (AO/OTA):

- Type A: Extra-articular
- Type B: Intra-articular, single column
- Type C: Intra-articular, both column, with joint and shaft dissociation

Single column (condyle) – Milch:

- Types I and II lateral condyle fractures (more common) – Type I lateral trochlear ridge intact and type II it is not
- Types I and II medial condyle fractures

Both column (Jupiter):

- High T – Transverse component proximal or at level of olecranon fossa
- Low T (common) – Transverse component just proximal to the trochlea
- Y – Oblique fracture through both columns with a vertical distal fracture

- H – Trochlear fragment is free and at risk of AVN
- Medial lambda – Proximal fracture exits medially
- Lateral lambda – Proximal fracture exits laterally
- Multiplanar – T type with coronal plane fractures

Capitellum (Bryan–Morrey with McKee modification):

- Type I (Hans–Steinthal): Complete fracture of capitellum
- Type II (Kocher–Lorenz): Shear osteochondral fracture
- Type III: Comminuted
- Type IV (McKee modification): Coronal shear fracture of capitellum and part of trochlea

Management

Supracondylar

Non-operative

Cast for up to 6 weeks with serial radiographs in undisplaced or minimally displaced supracondylar fractures.

Operative

ORIF for displaced supracondylar fractures. A posterior triceps-sparing approach can be used for extra-articular and simple intra-articular fractures with full thickness medial and lateral flaps, and identification of ulnar and radial nerves by elevating the deep fascia. Can be converted to an olecranon osteotomy if needed.

Complications: Infection, neurovascular damage, non-union (1–11% with ORIF), mal-union and stiffness (contracture, bony block, fibrosis).

Single column

Non-operative

Cast for Milch type 1 undisplaced condyle fractures in supination (lateral condyle) or pronation (medial condyle).

Operative

Closed reduction and percutaneous K-wires, screws or plating (depending on the size of the fragment) for displaced Milch type I fractures.

ORIF for Milch type II fractures. A lateral approach can be used with elevation of ERCB and part of ECRL off supracondylar ridge, anterior to LCL, allowing visualisation of the articular surface.

Complications: Cubitus valgus (lateral), cubitus varus (medial), ulnar nerve injury and joint degeneration

Both columns

Non-operative

Cast and **'bag of bones'** treatment with early mobilization for unreconstructable intra-articular fractures in cognitively impaired patients with poor bone quality and multiple co-morbidities. Even if fracture does not heal, the non-union site may be painless and good motion may be present.

Operative

ORIF using a posterior approach with an olecranon osteotomy (for complex intra-articular fractures) which gives the best articular surface exposure. The same exposure as the triceps sparing approach (see above), with a distal pointing chevron apex osteotomy, fixed with a 6.5 mm screw, plate or tension-band wiring. Anatomical periarticular locking pates are placed in parallel to reconstruct the medial and lateral columns. Alternatively, plates may be placed at 90° to each other with equivalent biomechanical strength, although recent evidence favours the parallel technique. For intra-articular fractures, provisional K-wire fixation is followed by countersunk screws to fix articular fragments and the condyles first. The articular segment is then fixed to the shaft. Early mobilization with <3 weeks in a cast is the aim.

Total elbow arthroplasty: Patients >65 years with poor bone quality or rheumatoid arthritis with unreconstructable intra-articular fractures. Alternatively, it can be used in the healthy elderly patient with displaced transcondylar fractures. Two-year functional outcomes in the elderly with distal humeral intra-articular fractures were better with arthroplasty compared to ORIF, with lower re-operation rates[14].

Complications: Stiffness (most common, initial treat with static progressive splinting, although 50% of low T types require revision with good results), loss of elbow muscle strength of 25%, ulnar nerve injury (anterior transposition), AVN of trochlea fragment in H-type, osteotomy non-union and heterotopic ossification (4% with ORIF).

Capitellum

Non-operative

Cast for undisplaced (<2 mm) types I, II and III capitellum fractures for up to 3 weeks, before mobilization.

Operative

ORIF for displaced type I fractures (>2 mm) and all type IV fractures. A lateral approach is predominantly used, unless there are other elbow injuries warranting a posterior approach. Fixation using headless compression screws if anterior to posterior, or minifragment screws if posterior to anterior. Blood supply to the capitellum is posterior; thus, the posterior soft-tissue attachments must be maintained.

Excision of comminuted fragments in displaced types II and III fractures (>2 mm) which cannot be reduced and fixed[15].

Total elbow arthroplasty in elderly patients with unreconstructable fractures and medial column instability in particular.

Complications: As above, including avascular necrosis of capitellum.

Radial head fracture

Mechanism

Fall onto outstretched hand

Figure 27.4 Charalambous classification of radial head fractures[16]

Assessment

Examination

Lateral elbow pain, with tender radial head and limited elbow range of motion. Assess for coronoid process fracture and elbow instability, as well as distal radius fracture and Essex–Lopresti injury.

Imaging

AP and lateral radiographs of elbow and wrist (based on clinical examination).

Classifications

Mason (with Hotchkiss modification)

- Type I – Undisplaced or minimally displaced (<2 mm) head or neck fracture. No mechanical block to motion
- Type II – Articular or neck displacement/ angulation (>2 mm). Possible mechanical block to motion
- Type III – Comminuted and displaced head and neck fracture. Mechanical block to motion
- Type IV (Hotchkiss modification) – Radial head fracture with concomitant fractures or ligamentous injuries/ elbow dislocation

Charalambous

Based on the number and location of fragments and whether the fracture is un-displaced (U) or displaced (D) as shown in Figure 27.4[16]:

- Two-part partial articular (U or D)
- Comminuted partial articular (U or D)
- Comminuted complete articular (U or D)
- Isolated radial neck fractures (U or D)

Management

Non-operative

Indication: Undisplaced fractures.

Sling for pain relief then early range of motion exercises, which may be facilitated by aspiration of elbow joint and local anaesthetic injection. Similar for displaced fractures associated with a stable elbow, cause no block to motion, and displacement acceptable (<3 mm).

Operative

Indication: Substantial displacement, block to motion, elbow instability

ORIF: With headless compression screws (two-part partial articular or comminuted partial articular fractures), or periarticular plates (comminuted complete articular, radial neck fractures). The approach can be posterolateral (Kocher) using the interval between ECU and anconeus; lateral (Kaplan) using the interval between EDC and ERCB; or Wrightington with elevation of anconeus off the ulna and osteotomy of the supinator crest which is re-attached following fracture fixation (does not disrupt the substance of LCL). Forearm pronation helps to protect the PIN. The safe zone for plate placement is a 110° arc from radial styloid to Lister's tubercle laterally (~25%)

Radial head excision: In comminuted partial or complete articular fractures, isolated radial neck fractures, patients with low functional demands. Avoid in elbow instability or Essex–Lopresti injury

Radial head replacement: In comminuted partial or complete articular, isolated radial neck fractures, high demand patients or those with elbow instability or Essex–Lopresti injury. Avoid overstuffing the radiocapitellar joint

Outcomes: Patients with comminuted fractures undergoing ORIF had better range of motion, strength and function than those patients undergoing radial head excision[17].

Complications: Stiffness, posterior interosseous nerve injury (pronate to avoid injury), proximal migration of radius (Essex–Lopresti)

Elbow dislocation

Mechanism (for posterolateral dislocation)

Fall onto outstretched hand with a combined mechanism of axial loading, supination of the forearm and a posterolateral valgus force.

Elbow dislocation results from complete or near complete disruption of circle of capsuloligamentous stabilisers and bone (Horii circle). This occurs sequentially from lateral to medial in thre stages. Stage 1 (posterolateral rotatory instability) from partial or complete LCL failure (usually epicondyle avulsion and less commonly a midsubstance tear); stage 2 (perched ulna) with additional anterior and posterior capsular structure injury; and stage 3 (dislocation) with MCL failure depending on the degree of injury (often intact).

Assessment

Examination

Swelling and deformity of elbow. High energy (anterior and divergent types) associated with open and neurovascular injuries. Exclude compartment syndrome.

Imaging

Radiographs: AP and lateral views of the elbow to assess joint congruency, direction of dislocation and associated fractures, with an optional oblique view for periarticular

bony involvement. Wrist views to exclude Essex–Lopresti injury
CT scan: To assess type and displacement of fractures
MRI: To assess both ligaments and fractures.

Classification

Anatomical

- Posterolateral (80%)
- Posterior
- Anterior (high energy)
- Medial
- Lateral
- Divergent (high energy)

Severity

- **Simple**: No associated fracture, often first-time dislocation
- **Complex**: Associated fracture including medial/lateral epicondyle avulsion, osteochondral injury, radial head/neck fractures and coronoid process fractures

Coronoid process fractures (Regan and Morrey)

- Type I – Tip of coronoid process
- Type II – <50% of coronoid process
- Type III – >50% of coronoid process

'Terrible triad'

Elbow dislocation with lateral collateral ligament injury (avulsion from humeral origin), radial head fracture and coronoid process fracture.

Management

Initial

Emergent closed reduction using analgesia and sedation, or failing that general anaesthesia, which allows assessment of stability and appropriate reconstruction. Neurovascular status must be assessed pre- and post manipulation. Radiographs are assessed for congruency of reduction.

Non-operative

Simple dislocation: Stability in a functional range of motion with a congruent reduction can be placed in a sling with early range of motion exercises. Instability at extreme range of motion with a congruent reduction requires 1-week cast immobilization, followed by range of motion exercises
Complex dislocations: Undisplaced fractures post reduction with a congruent joint can be treated in a cast for up to 3 weeks with serial radiographs followed by range of motion exercises
Coronoid process: Type I fractures without symptoms of instability, and types II/III which are stable on examination

Operative

Indications: Inability to reduce, incarceration, incongruent reduction, gross instability
Simple dislocations: Gross instability with congruent reduction requires a cast and MRI scan to plan reconstruction
Complex dislocations: Reconstruction required for unstable injuries (e.g. 'terrible triad') and displaced fractures (CT scan). A lateral approach is used, with a medial approach if needed. A posterior 'utility' approach can be used to access medial and lateral elbow. A stepwise approach I used starting with radial head ORIF (reconstructable) or replacement (comminuted and unreconstructable) with a lateral collateral ligament repair. Only if still unstable, coronoid process ORIF (same lateral approach if radial head excised for replacement or separate medial approach). If instability persists, medial collateral ligament repair via a medial approach
Coronoid process: ORIF with cerclage wire, intraosseous sutures or plate if fragment large enough. Type I fractures with persistent instability – These are a marker of anterior capsular injury, and surgical fixation aims to reattach the stripped off anterior capsular structures rather than fixing bone. Types II and III
Postoperative rehabilitation: Range of motion exercises as tolerated
Complications: Infection, recurrence of instability in high-energy injuries, stiffness/ flexion contracture (if immobilized >3 weeks), heterotopic ossification of collateral ligaments, joint degeneration, brachial artery injury, median or ulnar nerve injury and capitellar erosion ('overstuffing' of radial head replacement)

Olecranon fracture

Mechanism

Direct fall onto elbow or indirect violent contracture of triceps.

Assessment

Examination

Swollen, bruised elbow, often with skin contusions. Assessment of elbow extension, radial head tenderness and neurovascular status.

Imaging

AP and lateral radiographs of the elbow to assess fracture for fragment size, type (see classification below), displacement, extension distally (making elbow unstable) and dislocation.

Classification (Colton)

- Type I – Avulsion
- Type II (A–D) – Oblique fracture, with increasing complexity

- Type III – Fracture–dislocation
- Type IV – Atypical, high-energy comminuted fracture

Management

Non-operative

Indications: Undisplaced (<1–2 mm) fractures and displaced fractures in low-demand elderly patients.

Immobilize in a cast at 60–90° for 1–2 weeks (maximum 3 weeks), followed by gentle range of motion exercises.

Operative:

Indications: Displaced >1–2 mm

Tension-band wiring: With stainless steel wire, with two dorsal loops to allow tension forces to be converted into compression forces at the fracture site. K-wires buried into the anterior ulnar cortex increase stability, but may reduce pronosupination if protrude beyond. Migration of these wires and metalwork irritation occurs in 70% with a high chance of requiring removal

ORIF: More stable than tension-band wiring. Contoured third tubular or periarticular-locking plate applied to the dorsal (tension) side for comminuted fractures, oblique fractures and those extending distal to the coronoid process

Intramedullary screw: Partially threaded 6.5 or 7.3 mm cancellous crew with a washer, ideally with tension-band wiring.

Excision with triceps advancement: Highly comminuted fractures in patient with poor bone stock and low demands. Excise <50% of olecranon and attach triceps close to articular surface suture anchors or intraosseous sutures to attach triceps

Postoperative rehabilitation: Early range of motion exercises

Complications: Infection, stiffness, metalwork failure or prominence (requiring removal), joint degeneration, non-union (usually a fibrous non-union which is stable enough to avoid revision), ulnar nerve injury and instability

Monteggia fracture–dislocation

Radial head dislocation with proximal ulna fracture.

Mechanism

Fall onto outstretched hand.

Assessment

Examination

Swollen, deformed and reduced elbow range of motion. Assess for tenderness at the wrist for possible TFCC/interosseous membrane injury and distal neurovascular status.

Imaging

Radiographs: AP and lateral views of elbow, forearm and wrist. Interosseous membrane injury when >3 mm instability when radius pulled proximal, and both interosseous membrane and TFCC when >6 mm.

USS or MRI: To confirm interosseous membrane injury.

Classification (Bado)

Based on direction of radial head dislocation:

- Type I (80%) – Anterior radial head dislocation and apex anterior proximal ulna fracture
- Type II (15%) – Posterior radial head dislocation and apex posterior proximal ulna fracture
- Type III – Lateral radial head dislocation and proximal ulna metaphyseal fracture
- Type IV – Anterior radial head dislocation and proximal ulna and radial shaft fractures

'**Monteggia equivalent/ variant**': Radial head fracture instead of dislocation

Management

Initial

Emergent closed reduction and above elbow plaster 'backslab' application under sedation and analgesia.

Operative

Indications: All adult injuries as unstable requiring operative management. Non-operative management leads to late radial head dislocation

ORIF using plate fixation of the ulna using a subcutaneous border approach usually allows reduction of the radial head, which remains stable (Figure 27.5). Ensure the fixation is anatomical, and assess stability of the radioulnar joint. If there is comminution of the ulna shaft fracture, the length must be restored to achieve stability. If the radial head cannot be reduced despite this (e.g. delayed presentations, annular ligament interposition in radiohumeral joint), then an open reduction is required via a posterolateral (Kocher) approach with reconstruction of the annular ligament

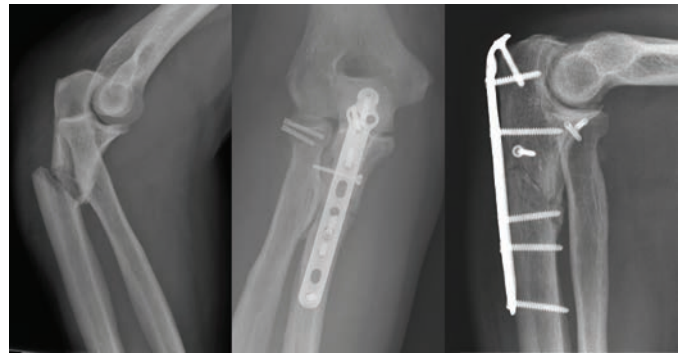

Figure 27.5 Monteggia fracture of the ulna and radial head stabilised with plate fixation for ulna and screws for the radial head

Monteggia equivalent/variant: Injuries may require a posterolateral or lateral approach to the elbow (See Radial head fracture section) to address the radial head fracture

Postoperative rehabilitation: Early range of motion

Complications (higher for type II and Monteggia-equivalent): Infection, PIN palsy (e.g. annular ligament reconstruction) – Observe for 3 months, as resolves spontaneously usually; stiffness, late radial head subluxation/dislocation (non-operatively treated injuries) and radioulnar synostosis

Isolated ulnar shaft fracture ('Nightstick' fracture)

Mechanism

Direct trauma to the ulnar border of the forearm.

Assessment

Examination

Swelling and crepitus over ulnar fracture. Assess proximal radioulnar joint (?Monteggia fracture dislocation) and distal neurovascular status.

Imaging

AP and lateral radiographs of the forearm, including the wrist and elbow.

Classification (anatomical)

Location and type of fracture, whether undisplaced or displaced.

Greater than 25–50% displaced or greater than 10–15° angulated are considered unstable.

Management

Non-operative

Below elbow moulded (interosseous) cast in distal two-third shaft and stable fractures. Consider 3 weeks initially an above elbow cast, although studies have demonstrated no difference in outcomes between a below elbow and above elbow cast[18].

Operative

Indication: Unstable injuries, proximal third or very distal fractures

ORIF using compression plating via a subcutaneous border approach. Also used for those fractures initially treated non-operatively with displacement, delayed- or non-union (with bone grafting)

Complications: Infection, mal-union, delayed-union and non-union

Radius and ulna shaft fracture

Mechanism

Indirect fall onto outstretched hand or direct trauma to forearm (e.g. high-energy RTA).

Assessment

Examination

Deformity, crepitus, swelling. Assess for open injury and distal neurovascular status.

Imaging

AP and lateral radiographs of the forearm, including the wrist and elbow.

Management

Operative

Indication: Displaced radius and ulna fractures in adults

ORIF with two incision technique using subcutaneous border for ulna and volar Henry's approach for radius. Both fractures are exposed and provisionally reduced with the least comminuted bone (usually the ulna) fixed first. Compression or bridge (comminuted fractures) plating with bone graft for bone loss and open injuries

Complications: Infection, PIN palsy for proximal radius fractures, vascular injury, radioulnar synostosis and stiffness

Galeazzi fracture–dislocation

Distal radius fracture with a distal radioulnar joint (DRUJ) dislocation.

Mechanism

Fall onto outstretched hand.

Assessment

Examination

Swelling, deformity and crepitus at the fracture site and wrist. Assess soft-tissue and distal neurovascular status.

Imaging

Radiographs: AP and lateral views of the forearm and wrist, including the elbow. Signs of DRUJ instability: Widened DRUJ on PA view, dislocated on lateral view, >5 mm radial shortening, ulnar styloid fracture and distal radius fracture is >7.5 cm from the articular surface (55% unstable, vs 6% for <7.5 cm)

CT scan: Of both wrists in a similar position often postoperatively if there is any doubt on the DRUJ on the radiographs

Management

Initial

Emergent closed reduction and above elbow plaster 'backslab' application under sedation and analgesia.

Non-operative

Isolated DRUJ dislocations may be reduced using analgesia and/or sedation.

Operative

Indication: All unstable injuries and non-operative management results in displacement

ORIF: Plate fixation of distal radius using a volar Henry's approach. The DRUJ is reduced and assessed for stability using forearm supination. If well reduced and stable – An above elbow cast or splint in supination with early range of motion is applied. If well reduced and unstable – Percutaneous K-wire fixation into ulna and radius across DRUJ in supination with cast. If irreducible – Open dorsal approach to DRUJ to remove block (usually ECU tendon), followed by K-wire fixation and above elbow cast in supination

Complications: Infection, mal-union, non-union, DRUJ subluxation/ dislocation and stiffness (fractures treated in a cast)

Distal radius fracture

Mechanism

Fall onto a dorsiflexed or volarflexed hand, violent radial deviation of the wrist.

Assessment

Examination

Swollen, bruised and deformed (e.g. dinner fork). Assess anatomical snuff box and elbow. Assess soft-tissues and distal neurovascular status – If signs of median nerve neurapraxia perform a carpal tunnel decompression at the same sitting as ORIF. This should be using a two-incision technique to avoid damage to palmar cutaneous branch of median nerve.

Imaging

Radiographs: PA (radial shortening, DRUJ disruption, distal ulna fracture, radial styloid fracture, radial displacement and intra-articular extension) and lateral (dorsal/ volar tilt or displacement, DRUJ dislocation and intra-articular extension) views of the wrist

CT scan: For displaced intra-articular and often distal fractures to assess fragments and plan fixation

Classification

Multiple, none definitively useful.

Frykman: Based on anatomy, no correlation to treatment or outcomes

- Type I – Extra-articular
- Type III – Radiocarpal joint
- Type V – Radioulnar joint
- Type VII – Both radiocarpal and radioulnar joints
- (Above plus ulnar styloid fracture for types II, IV, VI and VIII)

Fernandez: Based on mechanism of injury

- Type I – Bending fractures, usually extra-articular metaphyseal (volar (Smith's) and dorsal (Colles'))
- Type II – Shear of the articular surface (volar or dorsal rim (Barton's), radial styloid (Chauffer's))
- Type III – Compression (e.g. die-punch)
- Type IV – Fracture–dislocations (rare, high-energy trauma – Avulsion fractures with radiocarpal fracture dislocations)
- Type V – Combined mechanism of types I–IV (severe, high-energy trauma)

Universal: Useful in treatment planning

- Extra-articular or intra-articular
- Stable or unstable

Indicators of instability are (RADIUS): Radial shortening >5 mm, **A**ngulation >20°, **D**orsal comminution, **I**ntra-articular (depression >2 mm), **U**lna styloid fracture and **S**evere displacement

Management

Initial

Emergent closed reduction of displaced fractures using analgesia and sedation or a haematoma block, with application of a below elbow plaster 'backslab'.

Non-operative

Indication: Undisplaced stable fractures or displaced fractures reduced and remain stable

Closed reduction (if appropriate) and below elbow cast.

Acceptable parameters: <10° dorsal angulation, <2 mm of articular step-off and <2 mm radial shortening with radial inclination of 21° less important. Regular (up to weekly) radiological monitoring for displacement according to stability. Indicated for type I and undisplaced type II/III fractures

Elderly patients with poor bone quality, multiple co-morbidities, cognitive impairment and low functional demands may accept more unstable/displaced fractures for casting

Operative

Indication: All unstable fractures, particularly volarly angulated/ displaced fractures

Closed reduction and K-wire fixation: Percutaneous technique using 1.6 or 1.8 mm wires into radial styloid and dorsally as positional wires. Alternatively, the *Kapandji* technique corrects dorsal displacement/angulation and then maintains reduction(Figure 27.6). Indicated in type I and II *(Chauffer)* fractures, ideally young patients with extra-articular stables fractures (e.g. no dorsal comminution)

ORIF: Volar (or dorsal in type II) anatomical periarticular locking plate or fragment specific fixation (multifragmentary) with bone graft in significantly depressed fractures. Solitary screws may be used for radial

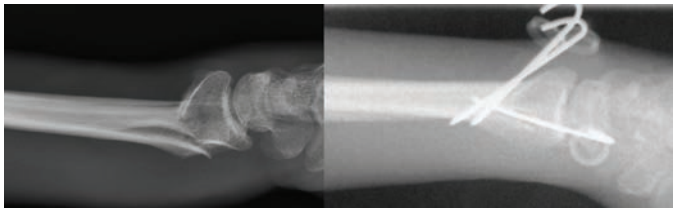

Figure 27.6 Kapandji technique of K-wire fixation of extra articular distal radius fracture

styloid or avulsion fractures. Unstable and/or displaced intra-articular fractures, articular margin fractures (buttress) and extra-articular fractures with dorsal comminution. Indicated in type II (where carpal subluxation may be present), displaced type III (disimpaction and bone graft of compressed articular surface), type IV and type V fractures

External fixation: Bridging construct for intra-articular fractures (static or dynamic), with some evidence of non-bridging construct for extra-articular fractures to allow radiocarpal motion. Adjunctive K-wire fixation may be useful for intra-articular fragments. High-energy injuries which cannot be reconstructed for bony (comminuted, often distal, intra-articular fractures) and/ or soft-tissue (severe soft-tissue and open injuries) reasons. The latter may be converted to ORIF within 2 weeks if required, particularly as there is diminishing effect of ligamentotaxis with time due to viscoelastic properties of ligaments[19]

After all surgical fixations assess pronosupination (DRUJ) and scapholunate joint stability. Intra-articular fragment reduction, TFCC integrity and carpal joints may be formally assessed with wrist arthroscopy although rarely done acutely

Outcomes: Dorsally displaced, unstable, extra-articular or simple intra-articular fractures of the distal radius treated with closed reduction and K-wires or ORIF (volar plate) had similar functional outcomes at 1-year postoperatively. However, better functional results were seen in the early postoperative period only with ORIF[20]. In addition, the multicentre randomised controlled DRAFFT (**D**istal **R**adius **A**cute **F**racture **F**ixa**t**ion) trial compared K-wire fixation to locking plate fixation in adults and found no difference in functional outcome and complications at 12 months, with K-wires being quicker and cheaper to perform[21]

Postoperative rehabilitation: ORIF (and non-bridging external fixator) allows early range of motion exercises out of a cast if the construct is stable enough. Other options require cast immobilization for a longer period so the emphasis on finger, elbow and shoulder range of motion exercises. Routine postoperative physiotherapy has not shown to improve outcomes

Complications: CRPS (one of the commonest complications and vitamin C may help in prophylaxis), infection, superficial radial nerve injury, loss of reduction/ mal-union, non-union, stiffness (particularly pronosupination if the DRUJ), median nerve neuropathy, ulnar neuropathy (DRUJ fracture), tendon injury/ adhesion/instability, EPL rupture (cast treatment), joint degeneration, compartment syndrome (leading to Volkmann ischaemic contracture) and DISI

Examination corner

Trauma case 1

EXAMINER: This is a 25-year-old patient with a midshaft humeral fracture treated with flexible intramedullary nailing 5 months ago *(shown radiograph)*.

CANDIDATE: Radiographs show a transverse fracture of midshaft of the right humerus with flexible intramedullary nails in-situ and lack of bony union.

EXAMINER: What is your plan?

CANDIDATE: I would like to take a full history and examination. History should focus on the symptoms, treatment received and complications of surgery, co-morbidities and smoking status. Examination would focus on vital signs, wound mobility at the fracture signs and neurovascular status.

EXAMINER: There is pus at the pin sites. Now what?

CANDIDATE: Pus suggests infected non-union. I would obtain a wound swab, inflammatory markers and consider a CT scan to assess for any union. If there are bridging trabeculae then the pins may be removed with pin site debridement and antibiotic therapy according to sensitivities.

EXAMINER: There is no evidence of union.

CANDIDATE: In that case I would remove the nails, debride the pin sites and the fracture site/intramedullary canal where possible. The bone ends would be debrided until there are bleeding edges with bone grafting. I would use an external fixator for skeletal stabilisation with antibiotic therapy according to microbiology advice.

EXAMINER: How would you consent the patient?

CANDIDATE: I would explain the name of the procedure and explain in lay terms, including the benefits of achieving union, treating infection, reducing symptoms and improving function. I would offer alternatives including a non-operative approach and the sequelae of persistent symptoms and infection. The risks would include infection, bleeding, nerve damage, scar, stiffness, further procedure, delayed/mal-/ non-union and failure.

- Good points – Suggested a reasonable plan and followed through with confidence. Detailed consent process made easy to understand for the patient
- Could improve – Mention the difficulties associated with an upper limb external fixator. Offer other alternative options such as retaining the metalwork until union achieved or intramedullary nailing. Mention any evidence in the literature

Trauma case 2

EXAMINER: A 45-year-old woman with this injury following a fall from height sustains this injury *(shown radiograph)*. Describe what you see.

CANDIDATE: Radiographs show right elbow fracture–dislocation with a displaced coronoid process fracture and possible radial head fracture, suggestive of a terrible triad injury.

EXAMINER: How would you manage the patient?

CANDIDATE: I would approach the patient according to ATLS® protocol.

EXAMINER: This is an isolated injury and the patient is stable.

CANDIDATE: I would like to know whether this is a closed injury and the neurovascular status.

EXAMINER: It is closed and neurovascularly intact.

CANDIDATE: Following appropriate consent and analgesia I would reduce the dislocation in casualty, placing the patient in an above elbow 'backslab' with check anteroposterior and lateral radiographs. I would check neurovascular status again.

EXAMINER: It is neurovascularly intact and the radiographs confirm reduction of the elbow joint.

CANDIDATE: I would arrange for a CT scan to make an operative plan.

EXAMINER: CT is performed. What is you approach and operative plan?

CANDIDATE: My preference is a lateral Kocher approach in between ECU and anconeus, with initial fixation of the coronoid process via the radial head fracture, then the radial head ORIF (replacement if not reconstructable) and LCL repair if needed. I would assess stability at each stage and at the end, with a temporary above elbow 'backIslab' for 2 weeks followed by an elbow brace with graduated active ROM exercises.

EXAMINER: What are the static and dynamic stabilisers of the elbow?

CANDIDATE: Static – Primary: Ulnohumeral joint, anterior MCL bundle and LCL and secondary: Radiocapitellar joint, capsule and CFO/CEO. Dynamic – Muscles crossing the elbow including anconeus, brachialis and triceps.

- Good points – Systematic and confident approach to managing an emergent situation, with good planning for definitive fixation
- Could improve – Beware of mentioning an ATLS® approach in managing isolated injuries. Perhaps describe posterior utility approach as an option. Offer evidence from the literature on management and expected outcomes

Trauma case 3

EXAMINER: A 30-year-old skateboarder fell on to left shoulder and this is the radiograph.

CANDIDATE: Radiographs show transverse fracture of midshaft left clavicle with shortening of about 2 cm.

EXAMINER: What will you do?

CANDIDATE: I will offer surgery in the form of open reduction and plate fixation.

EXAMINER: Why?

CANDIDATE: The shortening of the clavicle leads to shortening of the moment arm of shoulder and, hence, the strength of shoulder abduction.

EXAMINER: Do you have any evidence for this?

CANDIDATE: Yes. Paper published in . . . showed . . .

EXAMINER: How valid is that paper? What are its drawbacks?

CANDIDATE: Errr, don't know.

EXAMINER: OK. What are the indications for fixation of clavicle fractures?

CANDIDATE: Open fractures, polytrauma, multiple fractures in same limb, neurovascular injury, shortening >2 cm, symptomatic non-union.

EXAMINER: OK. What are the risks of plate fixation of clavicle?

CANDIDATE: Infection, wound healing problems, vascular injury, prominent plate, numbness distal to scar, plate breakage.

- Good points – Had a plan for this injury and did not beat around the bush
- Could improve – When quoting any reference, it is necessary to have read more than just the abstract and to be able to appraise the paper critically and give reasons as to why you would accept or not accept the conclusions offered. In the paper quoted there were high rates of complications in the operated group and secondary surgery to remove plate and screws

Trauma case 4

EXAMINER: A 25-year-old man suffers a grand mal seizure and presents with a painful shoulder with a limited range of motion. This is his injury – Describe what you see *(shown radiograph)*.

CANDIDATE: This is an anteroposterior and lateral 'y-view' lateral radiographs of the right shoulder, demonstrating a possible undisplaced fracture of the greater tuberosity. There appears to be a 'light-bulb sign' making me suspicious of a posterior dislocation – May I see an axillary lateral or Velpeau view?

EXAMINER: Here you go.

CANDIDATE: This is a posterior dislocation with an engaging reverse Hill–Sachs lesion in the region of 20%.

EXAMINER: What is your management?

CANDIDATE: Assuming this is an isolated injury and the patient is stable I would take a full history, when they are starved from, co-morbidities, allergies and medications, in preparation for theatre. On examination, I wish to confirm this is a closed injury and it is neurvascularly intact.

EXAMINER: He is starved and healthy, with no allergies and no regular medications. It is closed and neurovascularly intact.

CANDIDATE: I would take this man to theatre, where under a general anaesthetic (and muscle relaxation if required) and an image intensifier attempt a closed reduction using a traction-counter traction technique with a pillow case across the axilla. I would also consent him for an open reduction ± stabilisation in case the

closed reduction is unsuccessful or the shoulder is highly unstable.

EXAMINER: Would you not attempt this in casualty?

CANDIDATE: No in my experience these injuries are very difficult to reduce under sedation, particularly as there is an engaging Hill–Sachs lesion with possible fracture to the greater tuberosity and/or the posterior glenoid rim.

EXAMINER: Carry on . . .

CANDIDATE: Assuming I have a closed reduction, I would assess the shoulder for fractures and stability.

EXAMINER: The shoulder is stable and there is only a Hill–Sachs lesion.

CANDIDATE: I would place in a sling with the elbow at the side for 6 weeks. In the meantime I would obtain a CT if there is any doubt about bony injury to the humeral head or glenoid. This followed by physiotherapy (rotator cuff strengthening and periscapular stabilisation) and activity modification.

EXAMINER: What if he continues to suffer from posterior instability?

CANDIDATE: I would obtain an MR arthrogram to assess. If there is posterior Bankart lesion, I would perform an open or arthroscopic Bankart repair with a posterior capsular shift, which has outcomes of 80–85% success at 5–7 years. If there is <50% Hill–Sachs lesion I would consider a subscapularis and lesser tuberosity transfer to the defect (McLaughlin's procedure).

- Good points – Clear experience of managing these injuries and good knowledge of the surgical indications and options
- Could improve – Remember to enquire whether this is a first-time dislocation and consider control of epilepsy. Volunteer early why not to reduce in casualty and describe the deltopectoral approach. Useful to name authors/study groups of the outcome data mentioned, as well as the McLaughlin's procedure

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Chapter 28

Lower limb trauma oral topics

Jonathan R. A. Phillips and Gunasekaran Kumar

Introduction

The trauma viva component of the FRCS (Tr & Orth) examination is a 30-minute slot with 2 examiners. There may sometimes be an observer, trainee examiner or external examiner assessor also present and, if so, the examiners will clearly explain this to the candidate before the start of the viva.

In view of the large variations in cases discussed and the possibility of biases, the examination has being structured and standardized so that examiners now have a previously agreed set of cases and questions. Laptops have been discouraged and in its place printed images that have gone through a rigorous process of standardisation to ensure good quality images are used. The same sets of pictures are used by all the examiners, which ensures each candidate will see exactly the same pictures as well.

In general, there will be three scenarios from each examiner. This means that there will be lot more scope for in depth probing into a candidates' knowledge base. In general most candidates feel confident about their trauma knowledge due to good exposure to a variety of cases in fracture clinics, ward rounds and operation theatres. In addition, many candidates will spend time as part of their orthopaedic rotation working in a Major Trauma Centre (MTC). As a good working knowledge of trauma is expected, the exam is structured to test this knowledge in detail. A good solid start to the trauma viva is needed. As with any exam scenario, the start should be as non-controversial as possible, in order to avoid getting side-tracked. Candidates should discuss cases as if they were a first year Consultant Orthopaedic Surgeon. The general principles for any viva are not to get into an argument with the examiners or be controversial. The aim is to present yourself as a safe surgeon with sound rationale behind your decision making process that will lead to you scoring high marks. Quoting literature is not essential for every case that is discussed. However, evidence in support of managing common injuries like low-energy femoral neck fractures, wrist fractures, etc, will go towards passing the section with flying colours. Complex trauma may require specialist input, but the principles of managing these patients in the acute phase and their definitive management has to be part of the discussion.

The viva pattern

The spectrum of case discussed can be from a 'straightforward' intracapsular femoral neck fracture in a young adult to polytrauma involving multiple long bone fractures ± visceral injuries. The anticipated responses to these two scenarios are different. In the intracapsular femoral neck fracture case, the candidate is expected to discuss in detail the various management options available, complications (early and late) and how to manage each of these complications. In the polytrauma case, the candidate is expected to have a logical and coherent plan with respect to management in Casualty, team effort, logical sequence of investigations and surgical interventions (principles of Damage Control Orthopaedics (DCO) and Early Total Care (ETC)). The discussion should be tailored to save life, limb, prevent infection and achieve good function, in that order. In general, the discussion is going in the right direction, if the patient has reached the Intensive Care Unit (ITU). The primary aim of saving life has been achieved, at least temporarily and there is more time available for planning definitive care.

Each topic will start off with a radiograph or clinical photograph with a relevant history. The description of the radiograph or clinical photograph has to be clear and concise. This does not mean cutting corners. This is where practicing again and again describing a radiograph helps. It would be safer not to say, 'the obvious pathology is . . .' as in the tension of the examination circumstances; you might miss something that is more obvious. Time is of the essence; there is no point going on and on about what is normal. Move swiftly onto describing the problem to score the maximum possible marks. A typical scenario would be either a single bone/limb fracture or a polytrauma. After describing the radiograph/clinical photograph, ask for more radiographs if the radiograph shown is not adequate. For example, if the radiograph is of a pelvis showing a hip dislocation, further radiographs required would be – a lateral view of hip, full-length femur (to rule out an associated femoral shaft fracture).

When approaching a trauma scenario, it is vital to say that you would approach the patient using ATLS® principles. Practice your opening sentence so that it sounds slick and polished.

An example of a good opening sentence would be:

> 'I would approach the patient using ATLS® principles, firstly ensuring there were no life-threatening injuries that require treatment before assessing for any limb-threatening injuries. I would ensure that the injury is isolated, closed and there is no neurovascular deficit.'

The examiners need to hear you say this once at the beginning of your viva. Once it has been established that you understand the principles of ATLS®, do not repeat the same sentence for each scenario (especially if the scenario is a low-energy wrist fracture). A simple 'I would approach the patient using ATLS® principles' should suffice. If the examiners say it is 'an isolated injury' at the beginning of a question take the hint and do not go on about ATLS® management, as the examiners want you to quickly get to the main thrust of the question[a].

In the examination, if the candidate needs an investigation as part of management, then, they have to ask for it. For example, if you think the situation requires an MRI scan, you would be shown an MRI scan (provided you are right in asking for an MRI!!).

An AMPLE history (**A**llergies, **M**edication, **P**ast history, **L**ast meal and **E**nvironment of injury) should be taken from the patient and then further enquiries depend on the scenario. For example, a pathological fracture requires a detailed history with respect to weight loss, systemic features, sources of primary, etc. A candidate will not score many marks saying, 'you could do . . .' or 'you would . . .'. Your management plan should be logical and clear, including a sequence of investigations. There will always be more than one way to manage any given injury. A candidate has to have a particular mode of treatment as the first-line choice and should be able to rationalize it. It is also preferable not to mention any names.

The examiners have a prepared set of questions to ask. They will be bored asking the same questions again and again. It would be best not to give them a chance to ask something different. There are a few scenarios that the candidate should have practiced several times and should be able to go through with the examiners without a hitch.

In the following case scenarios, the discussion starts after ATLS® management, AMPLE history, essential investigations and after the first line of management like pain relief, splinting have been discussed. Antibiotic prophylaxis for surgical intervention in closed fractures is generally a one-off dose of an antibiotic that is broad spectrum and has a half-life of minimum inhibitory concentration (MIC) to cover the operative time. For open fractures detailed information on type of antibiotics and length of treatment is given in British Orthopaedic Association (BOA) guidelines published in October 2009[1]. Deep vein thrombosis (DVT) prophylaxis, irrespective of upper or lower limb injuries should be on assessment of patient as a whole, age, co-morbidities, previous DVT, multiple injuries, mobility status, malignancy, venous thromboembolism (VTE), etc., risk assessment form is available at the Department of Health website[2].

Be aware of the published guidelines to common conditions; for example, the BOA Standards for Trauma (BOAST) guidelines for severe open lower limb fractures. Many more BOAST guidelines can be found on the BOA website. Also be aware of the NICE guidelines for the treatment of hip fractures and VTE prophylaxis.

Fracture description

Always look at or ask for two orthogonal views that are well-centered, adequate, with good exposure and of the correct patient.

Anteroposterior and lateral view

- Displacement – Shortening, medial or lateral translation as a percentage of bone width
- Angulation – Varus or valgus. Another method of describing angulation is 'angulation with apex medial or lateral'

Lateral view

- Displacement – Shortening, anterior or posterior translation as a percentage of bone width
- Angulation – Procurvatum or recurvatum. Another method of describing angulation is 'angulation with apex anterior or posterior'
- Look for associated injuries. For example, in femoral shaft fracture, look for patella fracture, femoral neck fracture and hip dislocation
- Indirect evidence of soft-tissue injury includes gauze dressing and soft-tissue swelling
- If there is a classification for the fracture pattern, then, describe it clearly. For example, an ankle fracture – Supination external rotation injury stage IV according to Lauge–Hansen classification
- Always consider non-accidental injury (NAI) in children

Fracture patterns that provide information on mechanism of injury in long bones

- Transverse – Three-point bending
- Butterfly fragment – Three-point bending with axial loading
- Spiral – Torsion
- Multifragmentary or segmental – HIGH-ENERGY injury with possible combination of direct and indirect forces

[a] It is difficult to give absolutes, but if the examiners say 'isolated' it means that they don't want you to discuss ATLS®. Examiners can moan about candidates who repeatedly mention ATLS® at the beginning of each scenario, but this is a bit unfair as candidates are nervous and don't want to slip up and miss an associated life-threatening injury that will end up causing them to fail the oral.

Polytrauma

Key viva themes

Major Trauma Centres

Acute care and resuscitation of the polytraumatized patient

Damage control orthopaedics vs early total care

Mechanism of injury: HIGH-ENERGY injury (road traffic accident (RTA), fall from height)

Assessment: At present, patients with suspected polytrauma are taken to the nearest MTC. Patients with suspected pelvic injuries have a pelvic binder put on by the paramedics. In the Casualty department (which has been pre-warned by the paramedic team), the polytraumatized patient is received by a trauma team who use the ATLS® principles of primary survey, secondary survey and resuscitation. The trauma team should, at least, consist of senior members of the Emergency Department (ED) staff, Orthopaedics and General Surgeons. Many large trauma centres now have other members of staff present at trauma calls including radiologists and radiographers, and specialist paediatric surgeons and nurses for paediatric cases. Even though primary survey and resuscitation is described in sequence, the steps occur simultaneously with different members of the trauma call team performing their duties in tandem at the appropriate stage, not unlike a symphony.

The 'Trauma Triad of Death' has been described as a combination of coagulopathy, hypothermia and acidosis (Figure 28.1). There is a complex relationship between the three with each factor compounding the other. Severe haemorrhage diminishes oxygen delivery and may lead to hypothermaia. This can interfere with the coagulation cascade preventing blood from clotting. Anaerobic metabolism due to hypoperfusion will lead to lactic acid generation and metabolic acidosis. Lactic acidosis damages tissues and may lead to myocardial hypoperfusion. Aggressive resuscitation should be undertaken to prevent the development of these conditions.

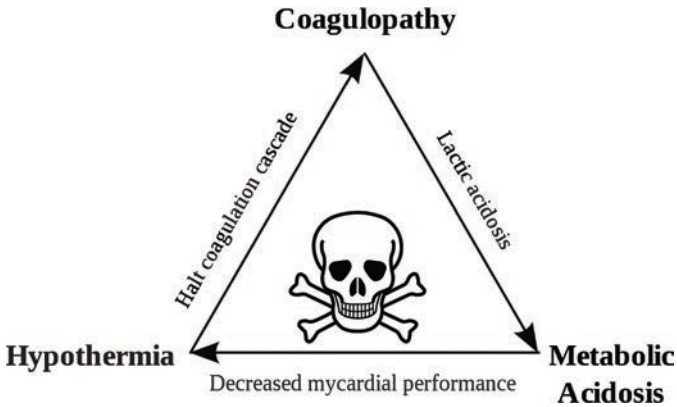

Figure 28.1 Trauma triad of death

The approach of 'Balanced Resuscitation' is now used in most trauma units. Recent literature has shown that resuscitation with large amounts of crystalloids is not beneficial due to dilution, loss of first clot and an imbalance in coagulation homeostasis. The aim of balanced resuscitation is to reduce the risk of tissue hypo-perfusion, thereby avoiding metabolic acidosis, coagulopathy and hypothermia which in turn reduce the systemic inflammatory response.

Aggressive management is undertaken to compensate for blood loss and to stop the bleeding, to maintain circulatory volume:

Compensation of blood loss: Major transfusion protocols are now in place in most Trauma Centres. The Major Transfusion Protocol is activated by the trauma team on the basis of mechanism of injury, physiological status of patient and identified and suspected injuries. The urgency of the blood product transfusion based on patient status will decide whether O-negative blood or type-specific or cross-matched blood is used. Major trauma transfusion is in the form of, packed cells, fresh frozen plasma and platelets (1 : 1 : 1)

Stopping the blood loss: Permissive hypotension, haemostatic resuscitation and damage control surgery

Permissive hypotension

Systolic blood pressure is maintained at 90 . This level keeps the balance between losing too much blood due to high pressure vs tissue hypo-perfusion due to low pressure.

Haemostatic resuscitation

The primary clot that is formed is the best clot and has to be maintained to promote further consolidation of this clot. If a pelvic binder has not been applied by paramedics, then, a pelvic binder should be applied with gentle internal rotation of both hips and flexion of knees. Intravenous tranexemic acid as bolus of 2 g or bolus of 1 g and another 1 g over a period of 8 hours has been shown to assist control of bleeding. Inotropes should be avoided. The primary cause of falling cardiac output in the trauma patient is low circulatory volume. Hence, flogging the heart will not improve the circulatory status.

Similarly, crystalloid and colloids should be avoided as they dilute and increase the risk of loss of the first clot.

Tranexamic acid and the CRASH-2 trial

This was a large international, randomised multicentre trial that showed that tranexamic acid, when given to trauma patients, reduced the risk of death in bleeding patients (relative risk (RR) 0.85; 95% confidence interval (CI) 0.76–0.96; P = 0.0077), and reduced the risk of all cause mortality (RR 0.91; 95% CI 0.85–0.97; P = 0.0035). Further in-depth analysis of the data has shown that tranexamic acid should be given as early as possible to bleeding patients – Ideally <1 hour after injury. Tranexamic acid given >3 hours after injury was less effective and could be harmful.

The CRASH-2 trial collaborators. Effects of tranexamic acid on death, vascular occlusive events, and blood transfusion in trauma patients with significant haemorrhage (CRASH-2): a randomised, placebo-controlled trial. *Lancet*. 2010;376: 23–32.

Traditionally, the diagnosis of coagulopathy was made using Activated Partial Thromboplastin Time (APTT), Prothrombin Time (PT) and International Normalized Ratio (INR). However, these take time to be reported. Bedside viscoelastic tests of blood can now identify the imbalance between thrombosis and bleeding. Two commonly used tests are thromboelastography and rotational thromboelastometry (ROTEM). These tests can identify whether packed cells or FFP or platelets are required in 5 minutes and assess fibrinolysis in about 30 minutes (Figure 28.2).

Damage control surgery

In order to control bleeding surgically, we need to identify the source of bleeding. Depending on the patient's status, CT with intravenous contrast from head to mid-thigh is the best investigation for identifying major injuries. In casualty, a FAST scan can identify free blood in the abdomen and chest. Bleeding from open wounds in the torso are controlled by direct pressure and bleeding from open wounds in the limbs are controlled by direct pressure or by use of a tourniquet (tourniquet inflation time should be recorded clearly and a named person should be monitoring it).

Interventional radiology: If contrast CT does show an arterial bleed in the pelvis that is amenable for selective embolisation, then it should be undertaken if such facilities are available.

If visceral bleeding is identified then laparotomy or thoracotomy to control bleeding may be considered by the surgical team.

Orthopaedic trauma damage control:

- Closed reduction of dislocations
- Reduction and splinting of fractures
- Traction
- External fixation of long bones
- Open fracture – Debridement and external fixation
- Amputation (only when 100% certain)

Damage control orthopaedics (DCO) vs early trauma care (ETC):

- It is a dynamic situation. As resuscitation progresses, the patient could be moving in and out of haemodynamic stability
- DCO is considered when resuscitation is deemed adequate. Tests that help to assess adequate perfusion include: Stable haemodynamics, no hypoxemia or hypercapnia, serum lactate ≤2.0 mmol/l, normal coagulation, normothermia and normal renal function (urinary output >1 ml/kg/h)
- Second hit: This is a phenomenon where the patient's condition worsens after a surgical intervention. There is controversy as to whether this is a reaction to a second trauma or whether a subclinical under-resuscitation is revealed by the surgery

When resuscitation is adequate and damage control surgery is completed the patient is taken to the ICU for further close monitoring and reassessment including hyperfibrinolysis and hypocalcaemia. Over the next few days, injuries are definitively managed as appropriate.

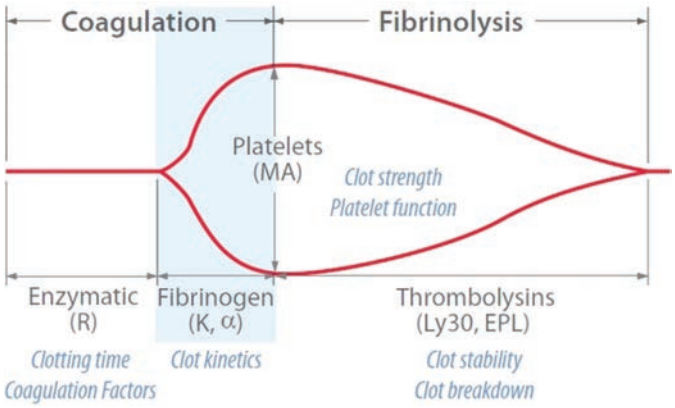

Figure 28.2 Thromboelastogram

Venous lactate

The venous lactate provides a real-time marker of the adequacy of tissue perfusion and, therefore ,of resuscitation in the trauma patient. It can be measured in most blood–gas machines. When anaerobic metabolism occurs (ie when the patient is under-resuscitated), pyruvate metabolises to lactate. Venous lactate is now used as a marker in come large trauma centres in the decision making for when to proceed with definitive surgery, or to undertake damage control surgery and continue resuscitation in the ICU.

Venous lactate:

<2.0 mmol/l – Safe to proceed with definitive surgery

>2.5 mmol/l – Damage control surgery, continue resuscitation on ITU

2.0–2.5 mmol/l – Observe for the trend. If worsening, continue to resuscitate, if improving it may be safe to proceed with care

Venous lactate is also being used for decision-making intraoperatively for the polytraumatized patient. If after one procedure is completed, the venous lactate is measured and is >2.5 mml/l, further definitive procedures should be postponed and the patient should undergo further aggressive resuscitation. Once lactate markers improve again, surgery can continue.

Intracapsular hip fracture in the young adult

Key viva themes: Reduction techniques. Surgical approach. Decision making and timing of treatment

Mechanism of injury – HIGH-ENERGY injury (RTA, fall from height)

Assessment – Closed or open fracture, soft-tissue injury, any distal neurovascular deficits and identify any other injuries

Radiological assessment –AP view of pelvis with both hips and lateral view of affected hip

Radiographic classification – Garden's I–IV on AP view but poor inter- and intraobserver reliability. Hence, it is better classified as undisplaced and displaced

Fracture orientation – The more vertical the fracture, more shearing forces at the fracture site which potentially increases risk of loss of fracture reduction and non-union

Timing – Literature has not shown much difference in outcome (non-union or avascular necrosis) when considering time to surgery as within 12 hours or beyond 12 hours. Traditionally these injuries were treated urgently. It is now reasonable to say that this should be taken to theatre first thing in the morning with an appropriate surgeon and theatre staff

Management options – All attempts should be made to preserve the femoral head. There is no role for prosthetic replacement unless radiographs show associated advanced osteoarthritis

Closed reduction technique – Leadbetter manoeuvre. On a fracture table, gentle flexion, adduction, axial traction and the hip is brought back into extension and abduction maintaining traction. Reduction is assessed clinically by the heels on palm test and by image intensifier. The heel palm test involves the surgeon holding both heels in their palms with both legs in abduction and internal rotation. Internal rotation is then released, and if the fractured site has significantly more external rotation than the non injured side, suggests reduction is not satisfactory. Whichever technique is used, it should be minimally traumatic to avoid further damage to femoral head blood supply. Repeated attempts at closed reduction could increase risk of damage to femoral head blood supply and should be avoided. Only anatomical reduction is acceptable and therefore be prepared to go on and perform an open reduction if necessary

Quality of fracture reduction – Garden Alignment Index is based on angle between compression trabeculae and long axis of femoral shaft in both anteroposterior (160°) and lateral (180°) views and has moderate interobserver reliability

Open reduction technique – Chose either the anterolateral (Watson–Jones approach) or anterior (vertical limb of Smith–Petersen) approach. Direct reduction under vision by manipulating the leg and two K-wires in the femoral head as a joystick to control rotation and reduce the fracture

Definitive treatment – This is with cannulated screws or sliding hip screw with derotation screw. Short- or long-threaded cannulated screws may be used but all threads must cross the fracture to achieve compression. Anterior capsulotomy is controversial; capsulotomy may reduce intra-articular pressure but there is no evidence to show any significant difference.

Factors that increase the risk of avascular necrosis (AVN)[3].

- Degree of initial fracture displacement (disruption of blood supply)
- Quality of fracture reduction
- Loss of reduction post surgery
- Fracture non-union

Postoperative regimen is either toe touch weight-bearing for 6–12 weeks or full weight-bearing from day 1[b]. Range of movement exercises is started from day 1.

Radiological follow up is for at least 2 years to assess for avascular necrosis of femoral head.

Serious complications – Loss of reduction and implant failure, AVN femoral head 6.6–45%; non-union 10–30%; and secondary osteoarthritis. Loss of reduction often follows when the fracture has not been reduced adequately. Avascular necrosis is often obvious or when suspected after fracture healing, removal of implants and an MRI scan will confirm the diagnosis and assess the volume of femoral head involvement. Non-union is associated with pain on weight-bearing and often obvious on radiographs with implant cut out or, if not obvious, a CT scan will confirm the diagnosis. One possible surgical option for non union in a young person following fixation failure is valgus intertrochanteric osteotomy described by Pauwels. Surgery involves ruling out infection, valgus trochanteric osteotomy (as described by Pauwels), stabilisation with 135° angled blade plate and bone graft. If fixation is deemed to be sound, then, options of augmenting fracture healing with bone graft or vascularized quadratus femoris bone graft. If all else fails consider total hip arthroplasty. Managing complications of femoral neck fracture fixation should be undertaken by a subspecialist but principles of that management should be discussed.

Femoral neck fractures in the elderly

Key viva themes: NICE guidelines

Mechanism of injury – Fall from standing height

Clinical assessment – Limb shortening, external rotation, distal neurovascular status, co-morbidities, ambulatory status, mini mental status, domestic circumstances

Radiological assessment – AP view of pelvis and lateral view of the affected hip

Full-length femoral view if concern that fracture may be pathological

[b] What is it going to be? Make up your mind. It will depend to a certain extent on quality of fixation, presence of osteoporosis and any coexisting co-morbidities

Management options

Management of all elderly patients with proximal femoral fractures should follow NICE guidance[4].

The key priorities of the NICE guidelines are:

- Timing of surgery
  - On the day of or day after admission
  - Identify and treat correctable co-morbidities immediately to avoid delay in surgical treatment
- Hip fracture surgery should be performed on a planned trauma list
- Perform replacement arthroplasty for displaced intracapsular fracture. Use extrameduallary implants for trochanteric fractures for AO classification types A1 and A2
- Mobilize patients on the day after surgery and daily thereafter with physiotherapy
- Multidisciplinary management
  - Orthogeratric assessment with rapid optimisation of fitness for surgery
  - Early identification of individual goals for rehabilitation to recover mobility and independence and facilitate return to pre-fracture residence and long-term well-being
  - Integration with related services including mental health, falls prevention, bone health and social services
  - Consider early supported discharge

In the UK there are currently six standards for hip fracture care (Blue Book). Trusts are audited against these targets, and if successful the Trust receives additional payments (a carrot rather than a stick to drive quality of care).

1. All patients with hip fracture should be admitted to an acute orthopaedic ward **within 4 hours** of presentation
2. All who are medically fit should have **surgery within 48 hours** of admission and during normal working hours
3. All should be assessed and cared for to **minimize the risk of pressure ulcers**
4. All presenting with a fragility fracture should have routine access to acute **orthogeriatric medical support**
5. All presenting with a fragility fracture should have **bone health assessment**
6. All presenting with a fragility fracture should be offered multidisciplinary assessment and intervention to **prevent future falls**

Intracapsular femoral neck fracture

Garden classification – Type I – Abduction valgus impacted. Type II –Undisplaced complete fracture. Type III– Complete fracture that is not completely displaced. Type IV – Complete fracture that is completely displaced.

Undisplaced

Surgical management – Fixation in situ using percutaneous cannulated screws fixation or two-holed sliding hip screw with a derotation screw (Figure 28.3)

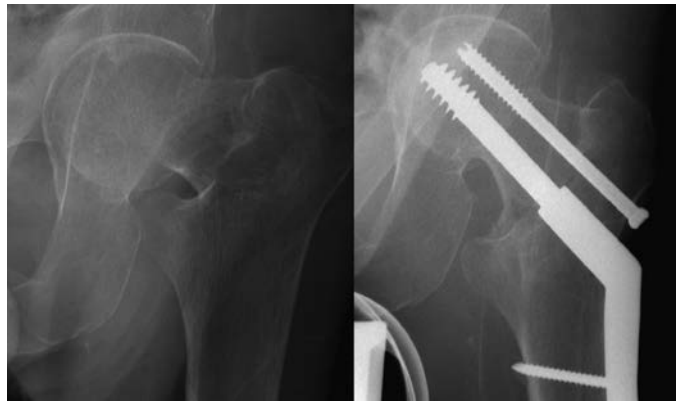

Figure 28.3 Basic cervical fracture stabilised with derotation screw and sliding hip screw

Complications – AVN, non-union, subtrochanteric fracture if cannulated screws inserted below level of lesser trochanter, chondrolysis if screws penetrate joint, screw cut-out

Displaced

Surgical management – High rates of non-union or AVN when fixed in elderly patients. Treated with cemented hemiarthroplasty or total hip arthroplasty

Complications (hemiarthroplasty) – Dislocation, infection, periprosthetic fracture (higher if uncemented), leg length discrepancy, acetabular erosion, aseptic loosening (long term)

Complications (total hip arthroplasty (THA)) – Dislocation (higher risk), infection, periprosthetic fracture, leg length discrepancy, aseptic loosening (long term)

Viva question – What type of surgery will you perform for a patient with a displaced intracapsular hip fracture?

Answer – Current NICE guidelines suggest that elderly patients with a displaced intracapsular fracture should receive replacement arthroplasty rather than fixation. Cemented hemiarthroplasty should be performed rather than an uncemented prostheses as they offer lower rates of periprosthetic fracture, lower incidences of thigh pain and improved hip scores and function. Patients who are able to walk independently out of doors with no more than the use of a stick and are not cognitively impaired should be offered a THA. However, this is balanced against the increased risk of dislocation (RR 1.48) and general complications (RR 1.1). The choice of treatment mainly depends on the general and biological condition of the patient. In general, the biological rather than chronological age should determine management.

Viva question – What are the advantages of THA over hemiarthroplasty in the treatment of elderly patients with hip fracture?

Answer – THA offers the patient a better functional outcome. A systematic review in 2010 showed that functional scores have shown consistently higher results after total hip replacement (THR) (mean Harris Hip Score 5 points better after THA than hemiarthroplasty). Rates of re-operation are also lower (RR 0.57). However, this must be balanced against an increased rate of dislocation (RR 1.5) and general complications (RR 1.1). A THA is a more technically difficult operation that takes longer with higher amounts of blood loss . A recent paper suggested that surgeons performing fewer than 35 THAs each year have higher complications . Surgeons who perform THR for fracture should, therefore, be performing this surgery regularly.

Extracapsular femoral neck fracture

Intertrochanteric fracture – Classically described as being two to four part, with increasing instability of fracture configuration with increased number of fragments

Stable and unstable fractures with small lesser trochanter fragment – Fracture reduction on traction table and insertion of sliding hip screw to compress across fracture site.

Radiological assessment of position of femoral neck screw is by Tip Apex Distance (TAD) – Risk of fixation failure increases when the TAD (sum of distance from screw tip to the midpoint of the femoral head in both the anteroposterior and lateral views after correction for magnification) is >25 mm[5].

Unstable fracture with large posteromedial fragment – Sliding hip screw or consider cephalomedullary nail (short) (Figure 28.4)

Reverse oblique fracture – This is an unstable variant. The fracture line extends from proximal–medial to distal–lateral in the trochanter. The biomechanics work against using a sliding hip screw. Treatment is with a cephalo-meduallary nail after fracture reduction. If closed reduction of fracture is not possible due to flexion, external rotation and abduction of the proximal fragment then, open reduction of fracture is performed before nail insertion.

Subtrochanteric fracture – This fracture may be either principally a trochanteric fracture with subtrochanteric extension or the reverse. Consider mechanism as differential diagnosis includes pathological fracture secondary to malignancy and fracture related to bisphosphonate treatment

Surgical treatment – Fracture reduction on fracture table. May require open reduction and cable reduction. Long cephalomedullay nail fixation

Technique – Entry point challenging in obese patients, ensure correct entry point for specific nail and consider the anterior bow of the femur, ensure correct rotation of fracture prior to distal locking, more difficult reduction in coexisting osteoarthritis, avoid fixing in varus

Complications of internal fixation– Mortality, infection, avascular necrosis of femoral head, non-union, implant failure (cut out), increased rate of revision surgery, periprosthetic fracture around the tip of a short nail

Trochanteric fracture in an arthritic hip

Mechanism of injury – Fall on to hip from standing height

Assessment – History of pre-existing groin pain and stiffness, limb deformity, external rotation and or shortening and distal neurovascular deficit

Radiographic assessment – Usually two part intertrochanteric fracture of proximal femur with features of moderate to advanced osteoarthritis

Proximal femoral fractures in an osteoarthritic hip is most often a pertrochanteric fracture. In a stiff hip, it is easier for the femur to fracture in pertrochanteric rather than subcapital region due to a longer lever arm

Management options – In a patient who is fit and healthy with a trochanteric fracture with advanced osteoarthritis, THA with a calcar replacing femoral prosthesis is a good option. The advantage is that one operation solves both problems. The disadvantage is that it is a major procedure with increased surgical risks, including infection and dislocation.

In moderate osteoarthritis in a fit and healthy patient or a patient with significant medical co-morbidities, a dynamic hip screw fixation is preferred. The advantage is that it is a short surgical procedure with limited risks. The disadvantage is that there is an increased risk of non-union, which may need further procedures in a staged fashion if osteoarthritis symptoms get worse (removal of implants followed by total hip arthroplasty).

Femoral shaft fracture

Key viva themes: Timing of treatment, ETC vs DCO, femoral shaft fractures with chest injuries

Mechanism of injury – Motor vehicle accident or fall from height

Initial management – ATLS® principles of resuscitation, traction splint, analgesia, IV fluids

Radiographic assessment – AP and lateral radiographs. Whole length femoral views. AP pelvis to assess femoral necks

Surgical treatment – Rule out pathological fracture due to malignancy or bisphosphonate treatment. If young patient, reduce fracture and use intramedullary nail (superior biomechanics). If older patient or presence of metastases, reduce fracture and use cephalomedullary nail to protect femoral neck

At the end of procedure – Assess knee joint for ligamentous integrity and rotational alignment of hip

Complications – Non/mal-union, rotational mal-alignment and shortening if comminuted or bone loss, fat embolism

Viva question – Would you perform intramedullary nailing of bilateral femoral shaft fractures where there is an associated chest injury?

Answer – On the basis of animal experiments, it is safe to perform bilateral nailing in a haemodynamically stable patient but the patient requires close monitoring prior to the second nailing. When the patient has poor oxygenation in association with a chest injury, external fixation should be performed regardless of whether there is an isolated or bilateral femoral fracture. In haemodynamically stable patients with bilateral femoral fractures and pulmonary contusions, consider intramedullary nailing of one fracture and either plating or external fixation of the other fracture[6].

Viva question – Would you perform intrameduallary nailing of a femoral shaft fracture overnight in a case of polytrauma?

Answer – This opens the debate of early total care vs damage control orthopaedics. I would ensure firstly that the patient was adequately resuscitated according to ATLS® principles. I would monitor physiological markers such as haemodynamic status, coagulation profile and venous lactate prior to making a decision. Operating at night with non-specialist theatre staff who are not familiar with the implants can be challenging. A retrospective study by Morshed[7] in the American *JBJS* in 2009 demonstrated that delayed fixation of femoral shaft fractures beyond 12 hours in polytraumatized patients may reduce mortality by 50%, especially in cases associated with serious abdominal injury. It is likely that this finding was due to time being taken to adequately resuscitate the patient prior to performing surgery to the fracture.

Femoral shaft fracture associated with femoral neck fracture

About 5% of femoral shaft fractures are associated with femoral neck fractures. If you are given a patient with a femoral shaft fracture in the exam always look carefully at the femoral neck!

Surgical treatment options

Both fractures can be stabilised by using one device, or two separate devices can be used for each fracture.

Single device – Cephalomedullary nail. This is more technically challenging, to achieve reduction of the neck fracture at the same time as fixation of the femoral shaft. Insertion of the nail may displace the femoral neck fracture. Technique includes fracture table with temporary wire fixation of neck fracture followed by nail insertion and neck screw with or without a second de-rotation screw[8]

Two devices – Cannulated screws or sliding hip screw for femoral neck fracture and retrograde nail for femoral shaft fracture. Plate fixation for femoral shaft fracture is generally not recommended. It is a load bearing device with a higher rate of implant failure and non-union. There is a need for an extensive surgical approach with associated blood loss, risk of infection and soft-tissue insult

At the end of procedure – Assess knee joint for ligamentous integrity, rotational alignment of hip in comparison with other hip and compartment syndrome

Postoperative rehabilitation – Varies from non-weight-bearing for 6 weeks to allowing immediate weight-bearing as tolerated depending on fracture pattern, fixation techniques and associated injuries

Complications – Fat embolism, acute respiratory distress syndrome (ARDS), missed femoral neck fracture, non-union, rotational mal-alignment

Viva question – Laminated radiograph shown of a communited femoral shaft fracture with part of the lateral cortex displaced and associated displaced intracapsular hip fracture. **This is a 27-year-old motorcyclist who has come off his motorbike sustaining these isolated injuries. The femoral shaft fracture is open with a large skin laceration. How will you manage the patient?**

Answer – The patient should be managed according to ATLS® protocols. I would ensure airway maintenance with cervical spine protection, adequate breathing and ventilation, circulation with haemorrhage control, insert two large-bore cannulas into his antecubital fossa, take blood off for cross-matching, FBC, U&Es and run in a bolus of IV crystalloid

COMMENT: The initial management of this patient using ATLS® principles wasn't asked for by the examiner so that although the candidate answered this part well he didn't score any points. The key words used by the examiner were 'isolated injuries'. If the examiner hasn't said this and you have already gone through ATLS® management in an earlier question you could play safe and say 'assuming these are isolated injuries'.

EXAMINER: This is his only injury.

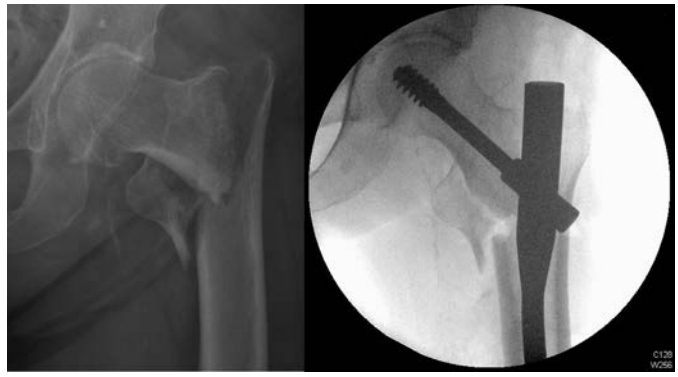

Figure 28.4 Unstable trochanteric fracture with large posteromedial fragment treated with an intramedullary nail

CANDIDATE: This is a difficult problem. I would go for two devices for fixation rather than one. I would treat the displaced intracapsular fracture with a two-hole DHS and supplementary screw. I would manage the femoral shaft fracture with a plate.

EXAMINER: Why do you want to do this? What fracture will you fix first?

CANDIDATE: This is a difficult problem.

EXAMINER: This is real life. The FRCS (Tr & Orth) is a difficult exam to pass with standards to maintain.

COMMENT: This is a difficult clinical problem and a candidate may have some difficulty thinking up a sensible safe management in the heat of the moment. Mentioning the complexities of the case once to the examiners is acceptable but twice was inviting trouble.

EXAMINER: You would use a plate for the femoral shaft fracture even though it is open fracture?

CANDIDATE: If there is a large wound there already then this may help me as I will have to debride, extend and irrigate the wound. A retrograde nailing may introduce infection into the knee joint.

EXAMINER: There is quite a lot of bone loss around the lateral cortex and when you debride the wound it a large piece becomes loose and you end up with a big defect in the femoral cortex. What are you going to do?

CANDIDATE: I would replace the bony fragment back into the femur to make the fracture more stable which will allow a better plate fixation and more chances of healing.

EXAMINER: Are you sure? Would the devitalized bone not be an infection risk.

CANDIDATE: I would remove the bone.

EXAMINER: What do you want to do? Do you want to put the bony fragment back into the femur or take it out?

CANDIDATE: I would take it out as it is dead and devitalized.

EXAMINER: Are you sure and then leave a big defect to heal?

CANDIDATE: Yes.

EXAMINER: Would you bone graft the defect?

CANDIDATE: No. The risks of infection would be increased.

COMMENT: You should know about the principles of dealing with bone loss associated with open fractures and moreover be confident with your answer. The candidate was unsure but recovered with their answer.

With the femoral fracture being open and life threatening this should generally be fixed first. If the femoral shaft fracture was closed you can debate which one to fix first. The arguments for fixing the femoral head fracture first is that if this injury is not perfectly reduced and fixed well this may lead to life long disability and it may be easier to get this more difficult fracture out of the way first and then manage the more straightforward femoral shaft fracture with a retrograde nail. The opposite argument is that you should fix the more life threatening fracture first and then afterwards deal with the femoral neck fracture.

Femoral shaft fracture with hip dislocation

Mechanism of injury – Motor vehicle accident, fall from motorcycle, fall from height

Radiological assessment – Direction of hip dislocation, level and type of femoral shaft fracture, rule out any associated femoral neck fracture or acetabular fracture

Initial management – Thomas splint, analgesia, frequent assessment for sciatic nerve and distal vascular status

Definitive management – Hip reduction takes priority. If closed reduction is not possible, which is often the case, then, a couple of Schanz pins are inserted into the proximal femoral fragment in the linea aspera, avoiding the femoral canal, if possible. A rod is used to connect the pins and use it reduce the hip joint. Options for treating femoral shaft – External fixation (as part of damage control orthopaedics), femoral plate fixation (if significant chest injury), femoral nail fixation. External fixation can be safely converted to nail fixation up to 2 weeks without increased risk of infection

If hip joint cannot be manipulated, then, open reduction is performed depending on the direction of dislocation. Anterior dislocation – Smith–Petersen approach; posterior dislocation – Posterior approach.

Hip dislocation ± posterior wall fracture or femoral head fracture

Key viva themes – neurovascular injury. Treatment of associated acetabular or femoral head fracture

Mechanism of injury – Fall from motorbike, motor vehicle accident (dashboard injury)

Initial management – ATLS® principles and resuscitation, assess position of limb, sciatic nerve, analgesia

Radiographs – Pelvis with both hips, full-length femur radiographs including knee. Assess for posterior or anterior acetabular wall fracture (Judet views), femoral head or neck or shaft fracture, patella fractures. Preoperative CT scan if it will not delay treatment. If there are large intra-articular fragments, definitive open debridement could be planned

Classification – posterior dislocation of hip (Thompson and Epstein classification)

Type I – Dislocation with or without minor posterior wall fracture

Type II – Dislocation with a large posterior wall fracture

Type III – Dislocation with comminuted posterior wall fracture

Type IV – Dislocation with acetabular floor fracture

Type V – Dislocation with femoral head fracture

Classification – Femoral head fracture (Pipkin)

Type I – Femoral head fracture below fovea. These fractures occur in the non-weight-bearing surface

Type II – Femoral head fracture above fovea. The weight-bearing surface is involved
Type III – Femoral head fracture with associated femoral neck fracture
Type IV – Femoral head fracture with associated acetabular fracture
Definitive management – Requires urgent closed reduction under anaesthetic. Do not leave overnight. Anterior dislocation – Traction in line with lateral traction of thigh. Posterior dislocation – Traction in line with adduction and flexion. Once the hip is reduced, dynamic assessment for instability is done under anaesthetic by figure of four for anterior dislocation and flexion with adduction and axial loading for posterior dislocation
If closed reduction fails, then open reduction depending on the direction of dislocation. Posterior approach for posterior dislocation and Watson–Jones(anterolateral) approach for anterior dislocation
Post-reduction management – Skin traction or skeletal traction (if associated with fractures or unstable). CT should be performed post-reduction to assess for retained intra-articular fragments
If stable following closed reduction, range of movement exercises should be started with weight-bearing as tolerated.

If unstable or associated with dislocations:

Posterior wall fracture – Posterior approach, reduction and plate fixation of fracture
Anterior wall fracture – Ilioinguinal approach, reduction and plate fixation of fracture
Femoral head fracture – Often associated with anterior dislocation. Watson–Jones approach
Large fragment – Reduction and fixation with screws with heads buried below level of articular cartilage
Multiple small fragments <50% femoral head size below level of fovea centralis – Removal of fragments
Non-reconstructable fragments involving >50% – Total hip replacement

Femoral neck fracture – Either a Watson–Jones or Smith–Peterson approach. The neck–head fragment must first be reduced into the acetabulum. If the femoral head fragment is small, fixation of the femoral neck first allows manipulation of the leg to assist in the reduction of the femoral head fragment. Use cannulated screws or DHS to fix the femoral neck fracture followed by counter sunk screw fixation of the femoral head. Consider THA with displaced femoral neck fracture in the elderly
Complications – Avascular necrosis of femoral head, sciatic nerve palsy, recurrent instability, heterotrophic ossification, secondary osteoarthritis. Risk factors for post-traumatic osteoarthritis include transchondral fracture, indentation fracture >4 mm depth and AVN

Pathological fracture of femur

Key viva themes – Diagnosis of metastatic disease. BOA guidelines

Typical history – Fall from standing height, pre-existing pain in the injured limb. History of malignancy, infection, metabolic disease, Paget's disease or bisphosphonate use

History of malignancy – Site, type, treatment (curative or palliative), previous pain in the injured limb, weight loss, appetite loss, under regular follow-up
Common malignancies that metastasize to bone – Thyroid, renal, GI, lung, breast, prostate, myeloma

Initial management – Splint, analgesia, check distal neurovascular status, observe for compartment syndrome
Blood tests – FBC, renal function tests, calcium, liver function tests, clotting profile, peripheral smear, group and save, serum electrophoresis
Staging – Local: Full-length radiographs of fractured bone, CT ± MRI
Systemic: Staging CT chest, abdomen and pelvis, Isotope bone scan or Positron emission tomography (PET). Seek oncology opinion regarding life expectancy and postoperative radiotherapy
Key Points from BOA *Blue Book on Metastatic Bone Disease*[9]:

- The prognosis for patients with bone metastases is improving. Many will survive >3 years
- Never assume that a lytic lesion, particularly if solitary is a metastasis
- Metastatic pathological fractures rarely unite
- Prophylactic fixation on long bone metastases is easier for the surgeon and less traumatic for the patient
- Fixation of pathological fractures or lytic lesions around the hip or proximal femur have high failure rates. Cemented hip prostheses have lower failure rates
- Never rush to fix a pathological fracture. Ensure investigations are performed and surgical intervention is discussed with appropriate colleagues
- Constructs should allow immediate weight-bearing and aim to last the lifetime of the patient
- Each trauma group requires a lead clinician for metastatic bone disease
- Management should be within the context of a multidisciplinary team[10]
- Definitive management is planned after all investigations and life expectancy is known.
- Known primary with multiple metastases – Stabilisation with locked nail unless life expectancy is very limited then, palliative management
- Known primary with solitary metastasis or unknown primary with solitary metastasis or primary bone tumour – Referral to bone tumour centre. Principles of management are wide or radical excision and endoprosthetic replacement, postoperative radiotherapy ± chemotherapy and surveillance

Identification of metastatic deposit in bone (no fracture)

Mirel's score is helpful to decide management of a metastatic deposit, based on radiological and clinical symptoms[11].

- Site – Upper limb, lower limb, pertrochanteric region (score 1–3)
- Size – Cortical involvement <1/3, 1/3 to 2/3, >2/3 (1–3)
- Type – Osteoblastic, mixed, osteoclastic (1–3)
- Pain – Mild, moderate, severe (1–3)

Total score >8 indicates surgical stabilisation of the involved bone but specificity and sensitivity is poor.

- Fixation of an isolated metastatic deposit should only be performed when the primary is known

Atypical fractures related to bisphosphonate treatment

Several publications have shown association between long-term bisphosphonates (especially Alendronate) and femoral fractures[12].

Characteristic features include:

- Transverse fracture pattern
- Fracture in proximal or mid-femoral shaft
- Localized thickening of lateral femoral cortex (beaking)
- Follow minimal or no trauma
- Prodromal symptoms of thigh pain

Treatment options include cephalomedullary nail, modification of osteoporosis treatment and surveillance of the contralateral side or staged prophylactic nail fixation of the uninvolved side in presence of radiological features and pain.

Periprosthetic femoral fracture around a THA

Key viva themes – treatment based on stability of prosthesis. Treatment in presence of infection

Mechanism of injury – Usually low-energy injury (fall from standing height)

Assessment – Pre-injury symptoms (thigh pain worsening on weight-bearing, night pain). Time since primary or revision THA. Type of THA (company, model, femoral taper size, femoral head size, cup size). Indication for THR. Perioperative issues. Postoperative complications including infection, dislocation and persistent pain. Associated co-morbidities. Pre-injury ambulatory status. Presence or absence of ipsilateral TKR

Radiological assessment – Level of fracture, fracture pattern, loosening of prosthesis (compare serial radiographs) and bone stock. All these factors are combined together in Vancouver classification[13]

- Type A – Fracture of greater (A^G) or lesser (A^L) trochanters
- Type B – Fracture around the femoral prosthesis
  - B1 – Well-fixed stem
  - B2 – Loose stem with good bone stock
  - B3 – Loose stem with inadequate bone stock
- Type C – Fracture well-distal to the tip of the stem

Management

- The stability of the femoral stem is the most important factor in decision making. If the implant is stable, fixation can be performed. If the implant is loose, the prosthesis should be revised
- Type A – Non-operative management provided the prosthesis is stable and in a satisfactory position without any signs of loosening or infection
- Type B1 – Open reduction internal fixation. Depending upon fracture configuration, fixation either with a single plate (four bicortical screws distal to stem with combination of unicortical screws, cables and bicortical screws (where possible) around the stem), or double orthogonal plating (anterior and lateral) with or without a strut allograft
- Type B2 – Revision of femoral stem (and acetabular cup if required)
- Type B3 – Revision; may require proximal femoral replacement
- Type C – Fracture stabilisation based on fracture pattern

In all periprosthetic fractures, infection must be ruled out. If any suspicion of infection, two-staged revision should be performed. First stage should consist of removal of all implants, insertion of antibiotic cement spacer, with 6–8 weeks of antibiotics based on microbiology cultures. Second stage should consist of removal of the antibiotic spacer, samples for culture

Routine follow up of joint replacements help identify loose prosthesis. Vancouver type B fracture subtype is often difficult to identify, hence, assessing fixation of femoral stem is essential[14].

Periprosthetic fracture around a TKR

Key viva themes: Fixation vs revision, extensor mechanism problems

1. **Fracture around femoral component**

Typical history – Elderly patient with low-energy fall

Assessment – Neurovascular status. Time since TKR, type of TKR (primary or revision, posterior cruciate sacrificing or retaining, company that made it and the model of TKR). Previous skin incisions. Any postoperative issues: Infection, stiffness, persistent pain. Associated co-morbidities. Pre-injury ambulatory status. Presence of ipsilateral THR

Radiological assessment – Is the TKR implant now loose as a result of the fracture? Is the fracture very low (little

bone left to fix distally), or in the metaphyseal region?
Is there comminution (especially medially)?
Review previous films – Was the implant loose before the periprosthetic fracture?
Early management – Splint, analgesia, record neurovascular status, check radiographs in splint
Definitive surgical management options – Very similar to periprosthetic fractures of the hip. Rule out infection. If the implant is loose either as a result of a low fracture or prior to fracture, revision surgery should be performed.
Consider referral to specialist arthroplasty unit for distal femoral hinged replacement (collateral ligaments are likely to be compromised)

If the implant is well fixed and the distal fracture fragment has adequate space for fracture stabilisation then internal fixation of the fracture is preferred. Consider the surgical approach. Either direct lateral approach – Closed reduction and bridge fixation with lateral distal femoral locking plate. Or midline incision and lateral parapatellar approach (use this approach if there is concern that the fracture is unreconstructable and endoprosthetic replacement may be required). Minimally invasive lateral approaches may be used as long as fracture reduction is adequate. Where there is medial comminution, the failure rate of lateral plate fixation is higher; consider additional medial plate fixation. Bridge the fracture with locking screws in the distal fragment with a long plate that spreads the stress across the bone (Figure 28.5).

Retrograde nail fixation is technically possible but careful preoperative planning is required to ensure this is possible (check with knee implant company), and to ensure that appropriate sized nails are available. Clearly nailing is not possible where a posterior cruciate sacrificing implant with a box has been used.

In the presence of ipsilateral THA, retrograde nail is not recommended due to the stress riser effect of a short length of 'unprotected' femoral shaft between tip of retrograde nail and tip of femoral stem. In this situation, the lateral locking plate fixation should overlap the femoral stem. If the fracture has dictated a stemmed revision TKR in the presence of an ipsilateral hip replacement, careful planning is required to ensure the stems fit within the remaining femoral bone; consider piggy-back plating across the unprotected femoral shaft to prevent future fracture.

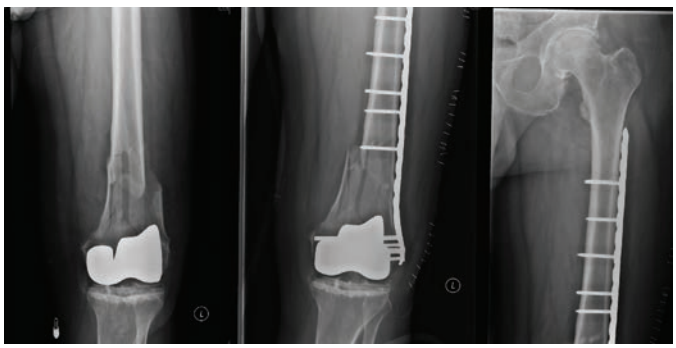

Figure 28.5 Periprosthetic fracture treated with a long locking plate

Viva question – Should a periprosthetic fracture around a well-fixed TKR in an elderly patient always be fixed?

Answer – There is a growing opinion that patients who sustain this fracture pattern are similar to the hip fracture group of patients. As such, fixation and protected weight-bearing may leave them bedbound and at risk of the complications of prolonged hospitalization (and financial cost). Revision TKR using a cemented, stemmed hinged prosthesis is, therefore, an option. There are the risks of a larger initial operation, but these may be outweighed by the benefits of early full weight-bearing mobilization and discharge (such as in the hip fracture patients). Financial costs may also be outweighed by reduced length of stay. The Knee fix or replacement (KFORT) trial is currently underway assessing DFR surgery or surgical fixation on the recovery of elderly patients with a distal femur fracture.

2. **Around the tibial component**

   Less common. Most vital is the integrity of the extensor mechanism and tibial tuberosity.

   If tibial component is stable, then fix in situ. If the tibial component is unstable, revise to a stemmed component. Sleeves and/or cones may be used to achieve stable fixation within intact tibial bone, otherwise fix distally with stem in diaphysis or cement stem. If the collateral ligaments are compromised, a hinge is required.

3. **Disruption of the extensor mechanism**

   This may include fractures of the tibial tuberosity or patella, or rupture of the patellar tendon or quadriceps mechanism. Extensor failure leads to poor results after knee replacement. If the patella is unresurfaced, primary fixation should be performed. Fixation in the presence of a patella resurfacing is likely to fail, especially if the button is loose. Excision of the patella and suturing of the remnants may be required.

   Any primary repair (of the tendons or patella) may benefit from augmentation with the ipsilateral hamstring tendons or an artificial graft

The dislocated knee and multiligament knee injury

Key viva themes – Management of neurovascular injury, anatomy of posterolateral corner

Mechanism of injury – High energy injury. A second cohort of morbidly obese patients sustain knee dislocations after low-energy falls

Structures that may be injured (in any combination) – Both cruciate ligaments, medial collateral ligament, posteromedial structures, posterolateral structures, tibial artery or vein, common peroneal nerve, menisci, patellar tendon

Initial assessment – ATLS® principles and resuscitation. Assess for neurovascular injury (high risk of injury). Reduce knee and splint

Vascular injuries should be managed according to the BOAST guideline for arterial injuries. These can be applied to any vascular injury associated with a fracture or dislocation

- Re-align the pulseless limb. Detailed documentation of neurovascular status before and after intervention
- Early referral to vascular surgeon
- Revascularization should take place within 3–4 hours
- The sequence of surgical interventions can be crucial. In general, vascular perfusion should be restored using temporary shunts followed by assessment of viability. Skeletal stabilisation should then be performed, followed by definitive reconstruction with autologous vein grafts. In the case of the dislocated knee with a vascular injury, temporary stabilisation with a bridging external fixator should be performed
- Observe for compartment syndrome
- Postoperative care should be by nursing and medical staff competent in the assessment of the critically injured limb

Once the knee is temporarily stabilised (normally in a splint if there is no neurovascular deficit), an MRI scan is required to evaluate the extent of injury and plan for definitive stabilisation.

Surgical treatment – Non-operative management historically yields poor results. Surgery should be within the first three weeks, and there is no consensus on the optimal graft choice (autograft, allograft or synthetic). Structures may be directly repaired, reconstructed or both. Two studies have found surgical repair alone leads to a higher incidence of failure with recurrent instability and, therefore, reconstruction is recommended[15,16].

Anatomy of posterolateral corner

Layer 1 (most superficial) – Superficial fascia (fascia lata), iliotibial tract, biceps femoris

Layer 2 – Anterior: Quadriceps retinaculum; Posterior: Two patellofemoral ligaments

Layer 3 – (most deep) – Superficial: Lateral collateral ligament, fabellofibular ligament; Deep: Arcuate ligament, coronary ligament, popliteus tendon, popliteofibular ligament, capsule

The 'floating' knee

Key viva themes: Surgical decision making

This is the presence of ipsilateral femoral and tibial fractures.

High-energy injury. Associated with polytrauma. Femoral or tibial fractures may be open with an associated neurovascular injury. High risk of compartment syndrome

Early management – ATLS® principles and resuscitate. Splint the limb after reducing any deformities

Practical difficulties in applying the external fixator – If the arterial injury is in the popliteal fossa, the surgical approach requires a prone position. Pins in the femoral shaft can be applied laterally with patient prone though the orientation can be confusing. However, tibial shaft pins application is difficult if not impossible in the prone position. The knee can carefully be flexed and pins inserted. After the vascular reconstruction is performed, rods can be attached to the pins in femur and tibia to reduce the fractures and maintain a tension free vascular repair/reconstruction

Definitive surgical management

- **Fractures in the diaphyseal regions** – Retrograde reamed locked femoral nail and antegrade reamed locked tibial nail through same incision[17]
- **One or both fractures in the metaphyseal region** – Fixation with precontoured plates as nail fixation of metaphyseal fractures more challenging
- After stabilisation of both fractures, assess the knee for ligamentous injury

Distal femoral fractures

Mechanism of injury – High energy

Initial assessment – ATLS® principles and resuscitation. Assess neurovascular status, analgesia, splint after reducing the deformity

Definitive management depends upon the fracture pattern

Supracondylar femoral fracture (extra-articular) – Retrograde nail or precontoured distal femoral locking plate fixation (can be performed using minimally invasive incision is satisfactory reduction can be achieved)

Intra-articular fracture – Open reduction of intra-articular fragments usually via anterolateral approach (or midline incision and lateral parapatellar approach if patient is elderly or has pre-existing arthritis). If severe arthritis present in elderly patient consider endoprosthetic replacement. Anatomic reduction and stabilisation is required to fix the intra-articular fragments. The plate acts as a neutralisation device. Care must be taken to achieve correct alignment, length and rotation, especially in comminuted fractures. The gastrocnemius will act to push the distal fragment into extension. This can be overcome through the use of a bolster under the knee and precise surgical technique

Patella fracture

Key viva themes: Fixation methods, description of tension-band principle

Mechanism of injury – Indirect through pull of quadriceps (transverse fracture), direct blow (multi-fragmentary fracture), special cases (following patella tendon graft for ACL reconstruction, following TKR or MPFL reconstruction)

Assessment – Soft-tissue status. Ability to straight leg raise
Fracture pattern – Level of fracture (superior or inferior pole, body), type of fracture (transverse, multifragmentary).
Differential diagnosis: Bipartite patella

Management

Non-operative if undisplaced (due to intact patellar retinaculum). Cylinder cast for 2 weeks followed by hinged knee brace with progressive increase in range of movements and serial radiographs to assess for fracture displacement.

Displaced fractures – Operative treatment. Transverse fractures can be treated with tension-band wire fixation (Figure 28.6), screws or cannulated screws with tension-band wire
Multifragmentary fractures – Tension band wire with cerclage wire fixation
Displaced inferior pole of patella fracture – If fragment is small, it can be excised and the patella tendon reattached to the main fragment
Patellectomy is not considered as a primary procedure for patella fractures.
Complications – Implant related prominent wires (often require subsequent removal), knee stiffness, wound healing issues

Tibial plateau fracture

Key viva themes: Surgical approach, care of soft tissues

Mechanism of injury – High energy injury (RTA, fall from height) in young or low-energy injury (fall from standing height) in osteoporotic bone
Initial assessment – Closed or open fracture, soft-tissue status, distal neurovascular status, compartment syndrome
Radiological assessment – AP and lateral views of knee and proximal tibia. Schatzker classification[18]

Type I – Lateral split fracture
Type II – Lateral split-depression fracture
Type III – Lateral pure depression fracture
Type IV – Medial fracture
Type V – Bicondylar fracture
Type VI –Fracture extending into metaphysis

The classification increases with increasing severity of injury, energy and soft-tissue damage.

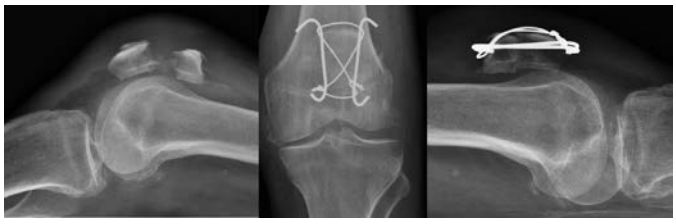

Figure 28.6 Transverse fracture of the patella stabilised by tension-band fixation

Types IV–VI fractures are associated with higher levels of neurovascular injury and compartment syndrome and can be associated with knee dislocation or subluxation.
Initial management – ATLS® principles and resuscitation if high-energy injury. Reduce and splint fracture. Evaluate for neurovascular injury and compartment syndrome. High level injuries may require spanning external fixation. Once stable, CT scan to assess fracture pattern and configuration. The CT is useful for identifying the degree of displacement and joint depression and for planning definitive surgery (Figure 28.7).

Definitive surgical management

This should only be undertaken once the soft-tissue injury has settled, which may take up to 3 weeks. The principle of surgical treatment is to reduce and stabilise the fracture and articular surface, bone graft any defects and allow early range of movement exercises. Surgical incisions should be planned to allow access to the fracture and articular surface. Care should be taken to avoid excessive stripping of soft tissues during exposure, and a wide skin bridge of at least 5–7 cm is required between incisions. A single incision to expose both the medial and lateral sides of the knee is generally not recommended.

Operative management options include:

- Percutaneous lag screws or anterolateral plate fixation can be used for undisplaced or minimally depressed fractures
- For depressed fractures, a window may be made in the anterolateral tibia to then 'punch' up the articular surface, followed by bone grafting and raft screw fixation
- Bone graft may include autograft, allograft or bone substitute
- Lateral fractures may be fixed using either open reduction internal fixation (ORIF) using precontoured plates, often as a buttress plate after articular surface reconstruction, or using a circular frame

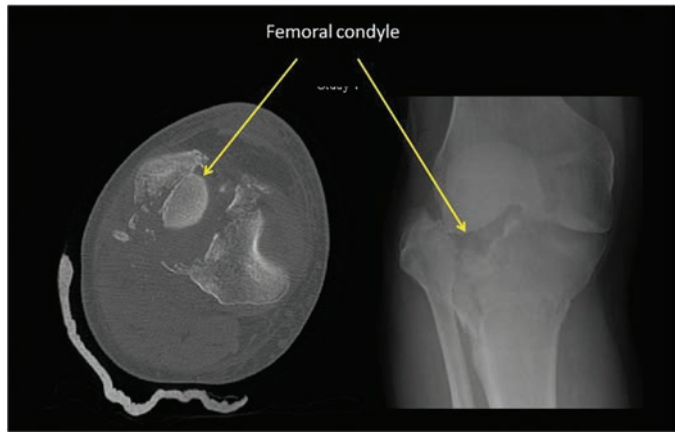

Figure 28.7 CT scan of tibial plateau fracture showing severe depression of the lateral tibial plateau, caused by the lateral femoral condyle driving through the lateral tibial plateau

- The anterolateral approach to the knee allows access to most lateral fractures. The meniscus may be elevated to visualize the joint and remove any incarcerated meniscus that may prevent fracture reduction. The joint may then be elevated, bone graft inserted and the fracture fixed
- Arthroscopy has been described to aid fracture reduction. This is often technically difficult due to the presence of bleeding, and is associated with a risk of compartment syndrome
- Once the fracture has been fixed, check for any ligamentous instability within the knee

Types I–III: Undisplaced or depression <2 mm – Examination under anaesthesia, if stable hinged knee brace
Types I–III: Displaced, unstable or depression >2 mm – Operative management
Types IV–VI: These fractures require careful surgical planning and evaluation of the fracture configuration (Figure 28.8). Posterior fragments where there has been posterior subluxation require a posterior approach and buttressing of the fracture. This may be accessible via a posteromedial incision but a posterior approach to the knee may be required. An additional anterior or anterolateral incision may also be required to reduce and hold all of the fragments. This may require the patient to start prone to achieve posterior buttressing of the fracture followed by turning the patient supine to perform the remainder of the procedure
Adjuncts to fracture fixation – If the fracture is unstable, a femoral distractor may be used, or the external fixator may be left on during definitive surgery. Remember that fractures are easier to reduce when body weight is working in your favour (such as choosing to go prone to reduce posterior fractures)

Viva question – Which is better? Circular frame or ORIF to treat severe tibial plateau fractures?

Answer – The evidence suggests that the outcomes using either approach are similar as long as the level of expertise is high . . .

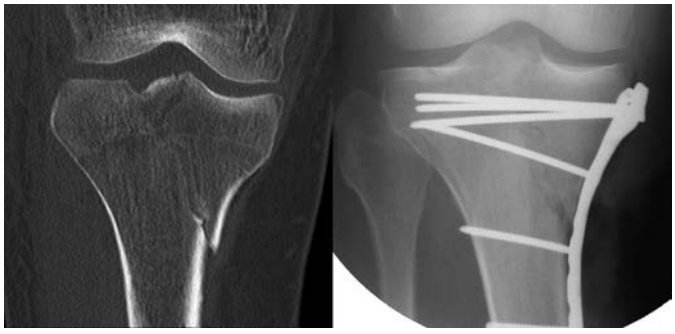

Figure 28.8 Medial tibial condylar fracture stabilised by a buttress plate

However, pragmatically, certain fracture such as those with significant soft-tissue injury may benefit from a less invasive approach. In the same way there may be other fractures, such as those with significant instability that buttressing the fracture with a plate may be preferable.

Tibial shaft fractures

Key viva themes: Management of open fracture, compartment syndrome, fixation of proximal or distal tibial shaft fractures

Mechanism of injury – May be high-energy injury, often isolated sporting injury in young adult
Initial assessment – Assess level of soft-tissue or neurovascular injury
Initial management – Above knee back slab, analgesia, observe for compartment syndrome

Definitive management

Non-operative management

Stable isolated fractures, <10° angulation or rotational mal-alignment, <50% translation may be treated non-operatively in plaster. Allow swelling and soft tissues to settle then apply above knee full cast either in theatre or plaster room and observe regularly to check for fracture displacement. Wedging may be used to correct small amounts of fracture displacement or angulation. At 4–6 weeks the above knee cast may then be converted to a Sarmiento or patella tendon bearing cast until the fracture is united[8].

Some authors recommend patella tendon bearing cast as the definitive management[8]. If the fracture starts to angulate in the cast, then, wedging of the fracture can be performed to correct it.

Operative management

Indications – Open fracture, associated vascular injury, segmental fracture, tibia and fibula fractures at same level, multiple fractures, compartment syndrome, and failed non-operative management
Surgical treatment options –

- Closed reamed interlocked nail fixation is the treatment of choice in tibial shaft fractures
- In fractures at metaphyseo-diaphyseal junction (either proximal or distal), nail fixation is possible but is technically demanding
  - Distal shaft fractures require tibial nails with multiple locking screw holes near the tip of the nail and nail length increments in 10 mm to allow accurate nail length. Distal fractures often
  - Proximal tibial fractures can be stabilised with a nail via a suprapatellar entry portal which requires special instruments and is technically demanding. There is a high incidence of valgus, apex anterior (precurvatum) mal-union in proximal tibial shaft fractures so

measures need to be taken intraoperatively to prevent this
  - Special techniques like use of blocking screws (Figure 28.9), fixation of the fibula fracture, external fixator assisted or plate assisted fracture reduction and angular stable locking screws all have extended the indications for nail fixation of tibial shaft fractures
  - Minimally invasive precontoured plate fixation is also an option in shaft fractures that are either in the proximal or distal metaphyseo-diaphyseal junction

- External fixation may be used either as a temporary measure such as in cases of severe open fracture, or can be used as a definitive treatment (using a circular frame or monolateral fixator)

Complications – Compartment syndrome, rotational deformities, anterior knee pain (after tibial nailing), non-union, mal-union

Open tibial fractures

Regarding the BOA guidelines in the treatment of severe open fractures, the principle recommendations are:

- A multidisciplinary team, including orthopaedic and plastic surgeons with appropriate experience, is required for the treatment of complex open fractures
- Hospitals that lack a team with requisite expertise to treat complex open fractures have arrangements for immediate referral to the nearest specialist centre
- The primary surgical treatment (wound debridement/ excision and skeletal stabilisation) of these complex injuries takes place at the specialist centre (normally the regional MTC) whenever possible

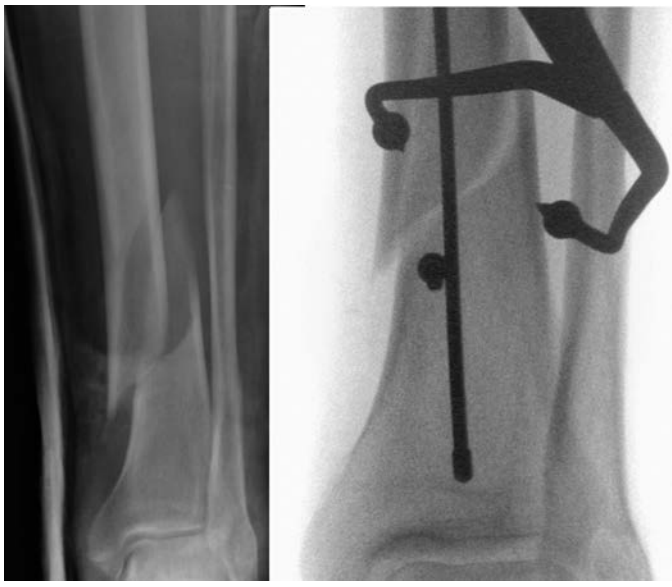

Figure 28.9 Distal tibial fracture – guidewire insertion aided by a blocking screw

These guidelines have a huge amount of information that are essential reading for any surgeon managing trauma. Other significant messages include:

- The specialist centre should include expertise in orthopaedic and plastic surgery, microbiology, radiology, limb fitting and rehabilitation services and intensive care facilities. Dedicated theatre sessions during the working day are necessary for the combined orthoplastic management of these patients
- ATLS® principles should be used. Wounds should not be 'provisionally cleaned'; only gross contaminents should be removed. Immobilization of the limb using simple splintage should be performed. A photograph of the wound should be taken and the sterile dressing applied
- Antibiotics should be administered as soon as possible, certainly within 3 hours. Anti-tetanus prohylaxis is given
- The only reasons for immediate surgical exploration are the presence of gross wound contamination, compartment syndrome, a devascularized limb and in the multiply injured patient. In the absence of these criteria, debridement should be performed on a scheduled trauma list by senior orthopaedic and plastic surgeons within 24 hours of injury
- A typical sequence of events should be followed
  - Initially the limb is washed with a soapy solution and a tourniquet applied
  - The limb is prepped with alcoholic chlorhexidine (avoiding the open wound)
  - Soft-tissue debridement and excision is performed under tourniquet control. Debridement means the excision of all devitalized tissue (except for neurovascular bundles)
  - Visualisation of the deeper structures is faciliatated by wound extensions along the fasciotomy lines. Tissues are assessed systematically in turn. Bone ends should be carefully delivered through the wound. Loose fragments that fail the 'tug test' should be removed, including large unviable fragments. Use copious lavage (but high pressure pulsatile lavage is not recommended). Negative pressure dressings may be used temporarily but not as a definitive treatment. Antibiotic impregnated bone cement beads are recommended in cases with segmental bone loss, gross contamination or established infection
  - After debridement the injury can be classified and definitive reconstruction can be planned. This may be as a single or multiple stage procedure
- Spanning external fixation is recommended when definitive fracture stabilisation and immediate wound cover is not carried out at the time of primary debridement. Internal fixation is safe if there is minimal

contamination and soft-tissue coverage is achieved at the same time as insertion of the implant. Exchange from spanning fixator to internal fixation is done as early as possible. Definitive external fixation can be used if there is significant contamination, bone loss and multilevel tibial fractures

- Definitive soft-tissue reconstruction should be undertaken within the first 7 days after injury. All open fractures should be covered with vascularized soft-tissue. Muscle flaps are better to treat diaphyseal tibial fractures while fasciocutaneous flaps are better to treat metaphyseal fractures especially around the ankle
- Primary amputation should only be performed as a damage control procedure if there is uncontrollable haemorrhage (usually from multiple levels of arterial/venous damage in blast injuries), for crush injuries exceeding a warm ischaemic period of 6 hours, or for incomplete traumatic amputations where the distal remnant is significantly injured. The decision to amputate should be taken by two consultants with, if possible, patient and family involvement

Classification of open fractures (Gustillo–Anderson)

Type I – LOW-ENERGY trauma, wound <1 cm
Type II – Wound 1–10 cm in size, without extensive soft-tissue damage
Type IIIA – Any high-energy open injury. Extensive soft-tissue damage with adequate soft-tissue coverage
Type IIIB – Any high-energy open injury with extensive soft-tissue loss where soft-tissue coverage is not possible
Type IIIC – Any open injury associated with an arterial injury requiring repair

Viva question – How would you evaluate and manage a clinical case of compartment syndrome at midnight in a tibial fracture that had undergone tibial nailing that morning?

Answer – The main clinical feature of compartment syndrome is pain out of proportion to the injury. It is a surgical emergency that I would diagnose and treat urgently. I would clinically evaluate the patient, looking in particular for pain of passive stretching of the muscles that pass through the tibial compartments. I would review the anaesthetic chart to see if the patient had received any nerve blocks during surgery that may mask symptoms of compartment syndrome. I would then split all dressings to the skin to relieve any external pressure and re-evaluate the situation. If available, I would measure intracompartment pressures. A difference of 30 mmHg or less between the measured pressure and the diastolic pressure is a reasonable threshold for decompression. Compartment syndrome, however, is a clinical diagnosis and if I can any cause for suspicion, such as in this case, I would proceed to urgent fasciotomy to decompress of the compartments.

Question – How would you perform the fasciotomy?

Answer – The BOA guidelines for the treatment of open fractures recommend a two-incision technique. I would perform long longitudinal anterolateral and a medial incisions. Through these incisions I am able to expose all four tibial compartments, and I would split the fascia longitudinally to release the pressure within each of the compartments. I would take care with my anterolateral incision to preserve the superficial peroneal nerve distally. I would leave the wounds open, covering them with a sterile dressing and I would arrange for the patient to return to theatre in 48–72 hours for a 'second look' with a plastic surgeon, where any devitalised tissue may be debrided or the wound closed if possible. If the wound does not close at this point I would liaise with the plastic surgeons to arrange soft-tissue coverage, likely in the form of a skin graft.

Pilon fracture

Key viva themes – ORIF vs external fixator, soft-tissue management

Mechanism of injury – HIGH-ENERGY injury (axial compression) – Fall from heights, road traffic accidents, skiing injuries) in the young. Low-energy injury(rotational) twisting injury – Fall from standing height in osteoporotic bone

Initial assessment – ATLS® principles and resuscitation if associated injuries (calcaneus, tibial plateau, pelvis, vertebral spine), status of soft tissues, neurovascular status, assess for compartment syndrome. Reduce fracture and stabilise with temporary cast. If fracture is open, refer to the BOA guidelines for severe lower limb open fracture. Admit and elevate limb

Radiological assessment – AP, lateral and mortice radiographs of ankle. Full length tibia/fibula and foot radiographs to assess alignment and look for fracture extension. CT scan to evaluate fracture pattern and to plan definitive surgery

Classification – Ruedi and Allgower[19]. Based on the severity of comminution and displacement of the articular surface

I – No comminution or displacement
II – Some displacement but no comminution or impaction
III – Comminution and/or impaction of the joint surface

Classification – AO

AO 43A – Extra-articular fracture
AO 43B – Partial articular fracture
AO 43C – Complete articular fracture

43C1 – No comminution

43C2 – Comminution of metaphysis
43C3 – Comminution of epiphysis and/or metaphysis (roughly corresponds to R&A group III)

Initial management – If alignment and articular fragments are reasonable then back slab, elevation, analgesia and CT scan. In presence of significant fracture displacement and/or ankle is subluxed or dislocated or significant soft-tissue damage is present, then, closed reduction and spanning external fixation is performed followed by CT scan

Classification – Soft tissues based on Tscherne classification. Fracture – Partial articular or complete articular (further subdivided based on degree on metaphyseal and or articular comminution)

Management – This depends upon the patient's co-morbidities, soft-tissue status and fracture pattern
Undisplaced fractures may be treated non-operatively in a cast for 6 weeks
Displaced fractures – The conventional treatment of this injury to perform an early spanning external fixation (using an 'A or Delta frame' leaving pins clear of the zone of injury and from potential incisions). This allows better control and visualisation and treatment of the soft tissues. The CT scan should be performed at this point. Only when soft-tissue swelling has subsided can surgery safely be performed(7–14 days on average)

Surgical options

- ORIF. The principles as described by Ruedi and Allgower are fibula length restoration, precise articular restoration, bone graft of the metaphyseal defect and stabilisation of the fracture. The axial CT provides information on how best to choose the fixation method. The fixation method then dictates the approach. Many approaches may be used (anteromedial, posteromedial or anterolateral to the tibia; posterolateral to fibula), but it is vital that the soft tissues are handled carefully. Skin bridges should be at least 7 cm
- Closed reduction and percutaneous fixation of articular surface. A minimally invasive approach may then be used to apply a percutaneous plate
- Acute limited fixation and external fixation with a ring fixator. The articular surface is reconstructed using K-wires (with or without olives) and/or lag screws. A ring fixator is then applied

Complications – Wound complications (soft-tissue slough, necrosis, haematoma), ankle stiffness and infection. Non-union if significant comminution, bone loss, hypovscularity and infection. Mal-union common with non-anatomic reduction, inadequate buttressing followed by collapse or premature weight-bearing). Reported incidence of up to 25%. Tibial shortening, secondary osteoarthritis, ankle stiffness, chronic pain, association with significantly poorer general health scores after injury

Viva question – Is there any evidence for early definitive treatment of pilon fractures?

Answer – The Vancouver group (White et al. *JOT* 2010)[20] published their results of early definitive fixation within 48 hours. They demonstrated excellent results with low complication rates. However, all fractures were treated by experienced high volume trauma surgeons. Historically early fixation has been associated with high complication rates, and in view of this surgeons should proceed with care and the generally accepted practice is to perform surgery once the soft tissues and swelling have improved.

Ankle fractures

Key viva themes – Mechanism of injury, methods of fixation

Mechanism of injury – Most commonly twisting injury

Immediate assessment – Assess neurovascular status, an x-ray of the displaced fracture provides a lot of information on the pattern of the fracture but should only be performed if it can be done immediately, a displaced fracture should then be reduced under sedation with analgesia, placed in a split plaster or backslab, x-rays repeated and the limb should be elevated. Gross dislocation clinically should be reduced and not delayed for radiographs as skin necrosis can progress rapidly. If left unreduced there is a risk of vascular compromise, pressure necrosis, articular damage and prolonged ankle soft-tissue swelling

Classification – Weber. The easiest and simplest classification, but only of the fibula and are based on the relationship to the syndesmosis: A = distal; B = at the level of the syndesmosis; C = proximal. Associated medial and posterior injuiries should be described
Lauge–Hansen classification is based on mechanism of injury and is well worth learning as it helps you to understand the direction of the forces involved and the structures injured
Supination external rotation (SER) injury is the commonest type. A candidate should be able to discuss the four stages of SER injury and differentiate stable and unstable SER injuries. Local features of medial injury include bruising, tenderness and swelling but these are not 100% sensitive

Definitive treatment

Stable fractures may be treated non-operatively. These fractures consist of Weber A injuries, and fractures where there is no evidence of joint incongruity. Stress testing may be used to aid in this diagnosis.

Unstable fractures are more often treated operatively.

Ankle joint incongruity of >2 mm has the effect of reducing articular surface contact by 60%. If elected to treat non-operatively, there must be no evidence of joint incongruity and serial radiographs are required until fracture union has occurred, possibly with changes of plaster as the ankle swelling subsides. An ankle that has been dislocated or previously displaced is an unstable injury. Weber B fractures *may* be unstable if the joint has ever been incongruent, or has been associated with a medial-sided injury. A Weber C fracture is an unstable injury.

Viva question – How do you assess for ankle joint incongruity?

Answer – The mortice view allows assessment of the fibula length by talocrural angle, talar shift by medial clear space >4 mm, syndesmosis by tibiofibular clear space and a broken circle sign. A circle sign is the curve between the lateral process of the talus and the recess of the distal tip of the lateral malleolus. It is vital that the ankle is fully dorsiflexed as the talus is cuboidal shaped and a plantarflexed ankle may look abnormal. I may also choose to compare x-rays of the other ankle or perform stress testing under image guidance. I am fully aware that an undisplaced fracture may still represent an unstable injury.

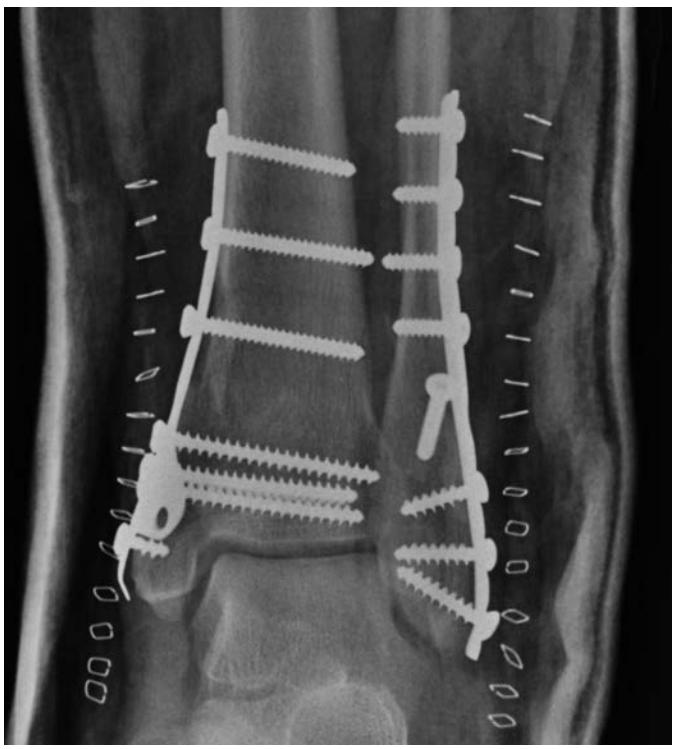

Figure 28.10 Medial malleolus treated with a plate

If an ankle is too swollen and/or has blisters, then elevate the leg. Surgery is delayed until skin wrinkles start to appear and blisters settle. If the ankle is grossly swollen or significant blisters develop or the fracture redisplaces in the back slab, consider a spanning external fixation which that allow blisters to be daily dressed and swelling more easily assessed.

Standard AO techniques of anatomic reduction and absolute stability should be lateral malleolus fracture:

Lag screw and contoured one third tubular neutralization plate or pre-contoured locked plate applied laterally
A posterior gliding plate with possible lag screw has the potential advantage of a buttress plate and allows bicortical fixation for screws in the distal fragment but peroneal tendons irritation is a potential issue
Other options include Rush pin, fibula nail with locking screw option. In the elderly, with significant soft-tissue and/or vascular pathology, a tibio-calcaneal nail can be considered to maintain alignment and allow early weight-bearing

Medial malleolus fracture:

Lag screws with or without washers (either uni or bicortical). If fragment is small or comminuted then, consider tension-band wire fixation or fixation with suture anchors. Vertical medial malleolus fracture (in supination–adduction type fracture) cannot be adequately stabilised with just screws. A buttress plate provides satisfactory stability to the fracture (Figure 28.10). The site of plate application depends on the plane of posteromedial fragment (coronal or oblique)

Posterior malleolus fracture

In injury patterns involving the posterior malleolus, the PITFL is usually still attached to the posterior malleolus fragment. A CT scan may be required for further evaluation. Posterior malleoli fractures benefit from fixation when they represent >25% of the articular surface, or when the fragment is displaced as ankle stability is restored. Fixation options include anteroposterior lag screws fixation after reduction with a pointed reduction clamp or a posterior buttress plate via posterolateral approach (Figure 28.11).

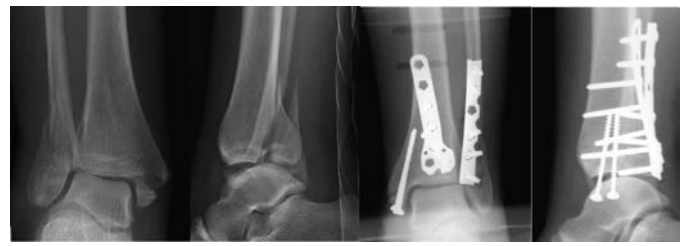

Figure 28.11 Posterior buttress plate fixation of a large posterior malleolus fracture

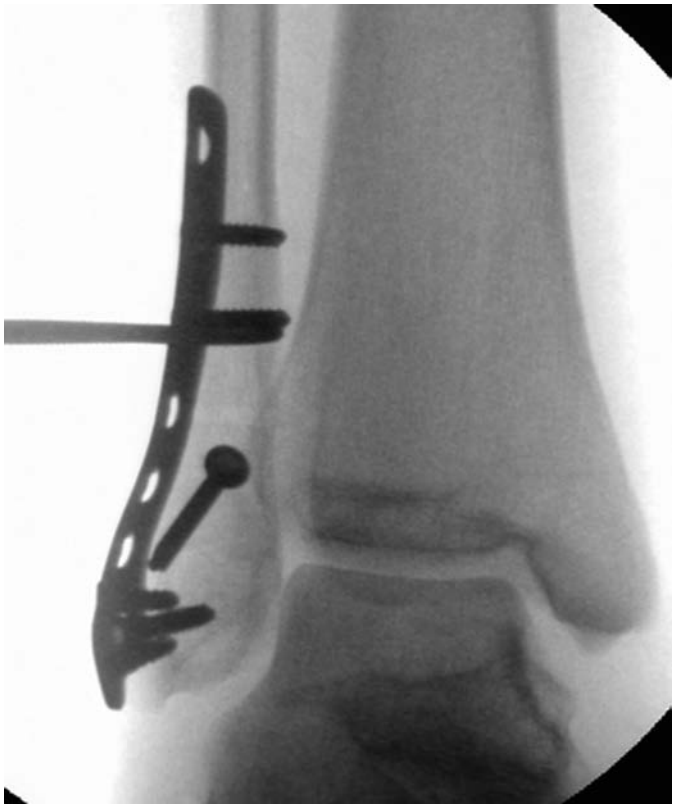

Figure 28.12 Hook test after lateral malleolus fixation

Syndesmosis Injury

Distal tibio-fibular syndesmosis injury can be subtle or obvious on radiographs. The commonest mechanism of syndesmosis injury is pronation external rotation (as per Lauge–Hansen classification). However, syndesmosis injury is possible in Weber B fractures. The integrity of the syndesmosis should be tested in all ankle fracture fixations by the hook test (Figure 28.12) or stressing the ankle in external rotation (thought more reliable).

There is no single preferred method of treating syndesmosis injuries[21]. One or two 3.5 mm or 4.5 mm fully threaded screw at 2 - 5 cm proximal to the ankle joint, running posterolateral to anteromedial, parallel to the ankle joint, across three or four cortices with ankle held in neutral. It is probably preferable to use two screws to reduce and hold the syndesmosis in an isolated syndesmotic injury such as in a Maisonneuve fracture The screw (s) should be used as positional screws not compression screws. Other options include suture techniques using a 'Tightrope' or bioabsorbable screws. If the syndesmosis injury is part of a bimalleolar fracture, then a syndesmosis screw is introduced through the plate fixation of the lateral malleolus. If it is a high pronation-external rotation (PER) type injury, then fibula length is first restored by traction and then a large pointed reduction clamp is applied across the lateral malleolus and tibia to maintain syndesmotic reduction. The syndesmosis is checked under image intensifier (talocrural angle, Shenton line, etc) before application of syndesmosis screws. It is important that the syndesmosis is reduced and the fibula is in the correct length and rotation in relation to the talus.

Screws need not be removed prior to mobilization In such injuries, the screw should not be removed early as that may lead to late displacement. There is currently controversy on what is considered to be a 'reduced' syndesmosis, as the syndesmosis is a mobile joint and, therefore, does not have a single position where is considered 'reduced'[22]. Regular removal of syndesmosis screw is controversial. An intact syndesmosis screw is associated with a worse functional outcome compared with loose, fractured, or removed screws[23]. However, there were no differences in functional outcomes comparing loose or fractured screws with removed screws. Screw removal is unlikely to benefit patients with loose or fractured screws but may be indicated in patients with intact syndesmosis screws.

In special cases such as in very elderly patients, patients with severe neuropathy or as a salvage procedure, hindfoot nailing may be performed.

There is also no considered optimal protocol for the post-operative management of ankle fractures. Braces may be used; however, cast immobilization and gentle increase in weight-bearing and mobilization over a 6-week period is considered usual practice.

Complications – Wound breakdown and infection (1–3%). Outcomes are worse in the elderly and diabetic patients. Post-traumatic arthritis has been described to occur in 14% of cases, most likely as a result of chondral injury sustained at time of the initial injury. Nerve injury (superficial peroneal nerve), ankle tiffness, RSD and compartment syndrome (rare)

Talus neck fracture

Key viva themes – Surgical approach, complications

Mechanism of injury – Usually high-energy trauma. Hyperdorsiflexion with axial loading of the ankle that causes impingement of talus neck against the anterior tibial lip

Radiological assessment – AP and lateral views of ankle. Preoperatively – Canale view (Plantar flexion, pronated and fluoroscopy directed 75° cephalad)

Hawkin's radiological classification

Based on the amount of displacement and the associated dislocations or subluxations. Rates of dislocation shown in brackets:

Type I – Undisplaced with no subtalar incongruity (up to 15%)

Type II – Displaced with subluxed or dislocated subtalar joint (up to 50%)

Type III – Displaced with dislocated subtalar and ankle joints (up to 100%)

Type IV – Displaced with dislocation of subtalar, ankle and talonavicular joints (100%). This category was not included in Hawkin's original paper but later added by Canale and Kelly[24]

Management

Undisplaced – Non-weight-bearing cast for 6 weeks. Serial radiographs to assess for displacement. If any doubt, CT scan

Displaced – If a closed reduction is achieved and the alignment is satisfactory by Canale's view, percutaneous screws fixation anterior to posterior. This fracture is often associated with medial comminution, and varus mal-union should be avoided. If the fracture cannot reduced using closed means, proceed to an open reduction

Open reduction

Approaches to the talus are a compromise of visualizing enough to achieve a satisfactory reduction while not further damaging the tenuous blood supply to the bone. The following approaches may be used – Anterolateral (with minimal vascular risk), anteromedial (risk to artery of tarsal canal) or posterolateral. Combined anterolateral and anteromedial approaches maybe needed. A medial malleolus osteotomy may help to visualize proximal fractures while also preserving the deltoid branches of the posterior tibial artery.

Viva question – What is the blood supply to the talus?

Answer – The talus is supplied by the posterior tibial artery (artery of tarsal canal, calcaneal branches), anterior tibial artery (branch to artery of tarsal sinus, medial tarsal branches), and the peroneal artery (branch to artery of tarsal sinus). The talar head is supplied by branches of dorsalis pedis artery and anastomosis of arteries of tarsal canal and tarsal sinus. The talar body is supplied by anastomosis of arteries of tarsal canal and tarsal sinus. The talus is an unusual bone in that 70% of it is covered by articular cartilage. The proximal body of the talus is particularly vulnerable to avascular necrosis after displaced talar neck fractures, which can be made further worse through the final remaining blood supply being damaged (entering medially) by surgical approaches to reduce and fix fractures.

Complications

Due to its unique anatomy, the tenuous blood supply, and the multiple complex articulations in the hindfoot, these injuries are associated with varus mal-union, non-union, avascular necrosis, secondary osteoarthritis of subtalar and or ankle joints[25].

Avascular necrosis – The talus is at risk of avascular necrosis after displaced talar neck fractures. Postoperative serial anteroposterior radiographs will show indirect signs of vascular viability. Hawkin's sign – Subchondral lucency in the talus on anteroposterior view of ankle at 6–8 weeks demonstrates vascular viability. The lucency occurs secondary to an intact blood supply causing bony resorption during fracture healing. Sclerosis is an indication of an inadequate blood supply and may be associated with non-union

Classic reference

Hawkins LG Fractures of neck of talus. *J Bone Joint Surg Am.* 1970;52:991–1002.

The Hawkin's classification is still one of the most widely used and accepted classification for talar neck fractures. It is based on displacement and dislocation, and therefore, presumed damage to the blood supply of the talus. The paper reviewed the outcome of 57 such fractures following various treatment methods.

Talar body fracture

Talar body fractures are commonly caused by axial compression, such as a fall from a height, with a worse prognosis than talar neck fractures (rate of avascular necrosis around 50%).

Surgical management – Medial malleolus osteotomy provides visual access to accurately reduce the fracture and stabilisation could be performed percutaneously with screws running posterolateral to anteromedial.

Talar lateral process fracture

Usually lower energy axial loading/inversion injuries to ankle frequently occurring with sporting activities. An intra-articular fracture that may be difficult to identify on plain ankle radiographs. A CT scan is often necessary to appreciate extent of the fracture. Increased incidence with snowboarding.

Undisplaced fracture – Non-weight-bearing cast for 6 weeks
Displaced fracture – Reduction and headless screw fixation
Comminuted fracture – Excision of fragments

Talar posterior process fracture

Mechanism of injury – Hyperplantar flexion
Radiographs – Anteroposterior and lateral views of ankle and CT scan. Differential diagnosis is os trigonum
Non-operative management with non-weight-bearing cast for 6 weeks followed by weight-bearing as tolerated and range of movement exercises
Complications – Non-union
Symptomatic non-union – If the fragment is large, then, either bone graft and fixation or excision. Smaller or comminuted fragments are excised

Talus dislocation

Mechanism of injury – High energy with associated rotational forces
Assessment – May be closed or open dislocation, assess local soft-tissue and neurovascular status. Could be medial (most common) or lateral dislocation (associated with distal fibula fracture)
Management – Closed reduction under anaesthetic as soon as possible. If unsuccessful, there may be tendon interposition (extensor digitorum brevis/peroneals in

medial dislocation or tibialis posterior in lateral dislocation). If closed reduction is unsuccessful then open reduction is performed using anteromedial and/or anterolateral approaches. Once reduced, check stability; if unstable a spanning external fixator or K-wires may be used to reduce the talo-calcaneal and talo-navicular joints

Complications – Soft-tissue problems, AVN (consider calcaneotibial arthrodesis), secondary arthritis (consider subtalar arthrodesis)

Calcaneal fracture

Key viva theme – Discussion of management options, factors associated with poor outcomes

Mechanism of injury – Axial loading (fall from height)

Immediate assessment – Can be associated with limbs, pelvis and spine related to the mechanism. Assess soft tissues and observe for compartment syndrome of the foot

Radiological assessment – Ankle lateral view (angle of Gissane, Bohler's angle) and calcaneal axial view (widening, lateral impingement). Broden view – Posterior facet of subtalar joint

CT scans in the axial and coronal planes help to fully understand the fracture pattern and plan for surgery.

Classification – The Essex–Lopresti classifies intra-articular fractures into joint-depression types, where the primary fracture line exits close to the subtalar joint, and tongue-types where the primary fracture line exits posteriorly. Sander's classification is based on the coronal section of the posterior facet of a CT scan and number of fracture lines

Treatment options

A recent multicentre randomised controlled trial has been published questioning the surgical treatment of calcaneum fractures[26]. This paper suggests that non-operative treatment of calcaneum fractures is associated with fewer complications and no difference in outcome scores in the short term. The strengths of its study were in its design and that surgery was performed by specialist surgeons. Its weaknesses include the large number of patients identified as suitable but then not included in the study, and its exclusion criteria – Patients were excluded if the fracture had gross deformity of the hindfoot (this is the criteria that many surgeons use as an indication for surgery). However, what this paper does suggest is that operative treatment of displaced calcaneal fractures does not lead to improved outcomes at 2 years

Undisplaced fracture – Elevation, maintain subtalar and ankle movements. Non-weight-bearing for 6–12 weeks

Displaced fractures – Fractures with gross hindfoot deformity (fibular impingement), open fractures or fractures causing soft-tissue compromise may benefit from surgery. The aim of surgery is to achieve reduction of posterior facet and reduce the widening of calcaneum. Surgery should wait until the soft tissues and swelling have subsided. An extended lateral approach is performed (maintaining thick soft-tissue flaps and meticulous surgical technique), with interfragmentory screws and a neutralisation plate to the lateral wall of the calcaneus. Broden's view intraoperatively confirms posterior facet articular reduction. The aim of surgical treatment is to reduce the calcaneo-cuboid and subtalar joints and to restore the shape and normal width of the foot. Again, gentle tissue handing with wound close avoiding tension must be performed to avoid soft-tissue problems and skin necrosis

Percutaneous techniques have also been developed. These are performed primarily for Essex–Lopresti tongue type fractures, using closed reduction and screw fixation, although techniques are now described for joint-depression fractures using leverage techniques to achieve fracture reduction with percutaneous screw fixation.

Factors associated with less satisfactory outcomes after surgical treatment are male sex, manual work, diabetes, steroids, smoking, alcohol, non-compliance and worker's compensation (medico-legal claims)[27].

Complications – Wound-healing problems, infection, prominent metalwork, subtalar arthritis, widened heel and difficulty fitting shoes (with non-operative treatment)

Calcaneal tuberosity fracture

Key viva theme – Skin necrosis

Mechanism of injury – Eccentric contraction of gastrosoleus causing calcaneal tuberosity avulsion fracture

Initial assessment – This injury is associated with skin necrosis and, therefore, requires prompt reduction and fixation

Management – Initial treatment is a cast in equinus. Undisplaced fractures may be treated non-operatively. Displaced fractures should be reduced and held with interfragmentory screws (Figure 28.13)

Lisfranc fracture dislocation

Key viva theme – Assessment of displacement and stability

Mechanism of injury: plantar flexion with axial loading or crushing injury

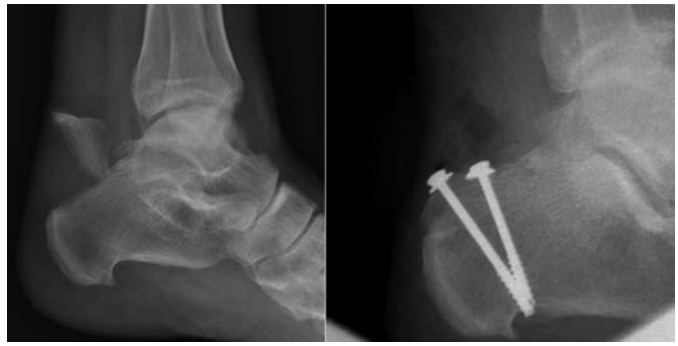

Figure 28.13 Calcaneal tuberosity fracture

Assessment: Local soft-tissue status, ensuring correct diagnosis (as this is a commonly missed injury). It is important to exclude diabetes and peripheral neuropathy as treatment of a Charcot injury is different

Viva question – What is the Lisfranc joint?

Answer – The Lisfranc joint consists of three cuneiform and two metatarsal articulations – The first and second. The Lisfranc ligament runs obliquely from the plantar aspect of the base of the second metatarsal to the plantar surface of the medial cuneiform.

Radiological assessment – Three views required – dorso-plantar view (medial border of second metatarsal in line with medial border of middle cuneiform), oblique view (medial border of fourth metatarsal in line with medial border of cuboid) and lateral view (dorsal displacement of metatarsal bases). If uncertain, obtain CT scan, MRI scan or stress view

Classification (Hardcastle)

- Total incongruity (medial or lateral)
- Partial incongruity (medial or lateral)
- Divergent (total or partial)

Management options

If truly undisplaced or stable on stress views – Plaster with non-weight-bearing and serial radiographs

Displaced/unstable fracture – Surgical fixation after soft tissues have settled. CT scan will help plan surgery. The principle of surgery is that the second metatarsal base is the 'keystone' fitting into the mortice between the cuneiforms. The second metatarsal base connects to the medial cuneiform via the plantar (Lisfranc) ligament. The medial column is less mobile than the lateral column. Two dorsal incisions are routinely used to visualize the joint (the first over the first/second TMT joints and the other over the fourth metatarsal). The joint should be visualized and reduced. K-wires may achieve a temporary reduction. The medial column may be fixed with screws; however, more recently bridging plates may be used to hold the reduction without compromise the articular surface. These are particularly useful in more comminuted fractures. The lateral rays are more mobile and may be held with K-wires. Primary arthrodesis of the TMT joints may be performed in more comminuted cases or in cases of delayed presentation

Charcot midfoot fractures – Treatment is primarily non-operative. In the acute phase, immobilize (total contact casting) with non-weight-bearing. This treatment may be necessary for 3–6 months. The underlying diagnosis including diabetic control and vascular supply should be optimized

Prognosis – This is a serious injury. Post-traumatic arthritis may occur. It is frequently missed at first presentation and may lead to long-term foot dysfunction, pain, stiffness and disability

Tendoachilles rupture

Key viva themes – What are the benefits of surgery?

Mechanism of injury – Eccentric loading of gastrosoleus, in middle-aged 'weekend warriors' often presents as a sudden pop or crack at the back of the ankle while playing sport

Associated factors include the use of steroids use, gout, quinolones and chronic renal failure

Assessment – Gap in the tendoachilles. Symond's or Thompson's test (Squeezing calf produces passive plantar flexion in intact tendoachilles.) Often ruptures about 5–6 cm from insertion. If diagnosis uncertain, ultrasound can be used

There is controversy with incomplete tendoachilles ruptures. Often, when ultrasound examination demonstrates an incomplete rupture, at surgery, the rest of tendon that appeared to be in continuity is, in fact, stretched and often non-functional

Treatment options

The treatment of Achilles tendon ruptures remains controversial. Good functional outcomes can be achieved using serial casting or functional bracing. Non-operative treatment has been associated traditionally with higher rates of rerupture when compared to surgery (8.8% vs 3.6% in a recent meta-analysis of level 1 trials). Surgery, however, is obviously associated with higher rates of deep infection, problems with the scar and sural nerve dysfunction. This has also been confirmed by a recent Cochrane review.

Surgery may be performed open or percutaneously. Percutaneous repair was traditionally associated with high rates of sural nerve injury (13%); the sural nerve runs posterolaterally down the calf in close proximation to the lateral border of the Achilles tendon. More modern techniques have demonstrated lower rates of sural nerve injury, with fewer complication rates when compared to open repair[28].

The non-operative treatment of Achilles tendon ruptures has improved and now functional bracing with intense rehabilitation may now achieve similar low rates of re-rupture when compared to surgical repair. Many historical papers with high rerupture rates consisted of casting for prolonged periods of time. Willits et al.[29] published an RCT comparing functional bracing with intense rehabilitation to surgical repair (with the same rehabiliation regime) and found similar low rates of re-rupture with fewer complications in the non-operative group.

Delayed presentation – Open repair may be augmented using fascia, the plantaris tendon, flexor hallucis longus, flexor digitorum longus or peroneus brevis. Techniques to lengthen the gastro-soleus complex may also be required. Chronic injuries are associated with worse outcomes and higher rates of complications.

Classic reference

Willits K, Amendola A, Bryant D, et al. Operative versus nonoperative treatment of acute Achilles tendon ruptures: A multicenter randomised trial using accelerated functional rehabilitation *J Bone Joint Surg Am*. 2010;92:2767–75.

Operative treatment of acute Achilles tendon rupture does not offer any clinically significant benefit over accelerated functional rehabilitation alone and is associated with a higher complication rate.

This is one of the largest multicentered RCTs using the functional rehabilitation protocol. It refutes the previous notions of very high rerupture rates after conventional non-operative treatment of plaster immobilization of the lower limb for 6–8 weeks.

Strengths included being a well-conducted large multicentered RCT that had clearly defined aims and inclusion/exclusion criteria.

In this multicentre study, 144 patients were randomised to two groups. Both groups underwent a prescribed accelerated rehabilitation protocol. One group also had operative repair. Primary outcome measure was rerupture rate.

Open fractures

Definition – A fracture that communicates with the external environment through a skin wound. Open tibia fracture is the commonest open fracture

Goals of management – Prevent sepsis, achieve skin cover, promote healing and regain good function

Local factors – Mechanism of injury, soft-tissue injury, contamination, fracture pattern

Gustilo and Anderson classification (after debridement) – Type I – low energy injury, <1 cm wound, minimal contamination, simple fracture. Type II – Higher energy, >1 cm wound, moderate contamination and comminution. Type III – High energy, > 10 cm wound, severe contamination (including sewage, marine and farm yard injuries) and comminution. Further subdivided into IIIA – Soft-tissue stripping but periosteal cover maintained (delayed primary closure or split skin graft), IIIB – Soft-tissue stripping with exposed bone (local or remote flaps) and IIIC – Associated arterial injury. This classification does not take into account presence or absence of bone loss. Interobserver reliability is about 60%

Management

The BOA has published guidelines on managing open fractures.

Initial management

Remove any gross contamination, photograph the wound, apply clean saline soaked wet swab to the wound, cover the wound with impregnable film, reduce the fracture and splint the limb, radiographs of the limb including joint above and below are obtained.

Tetanus cover – If patient was fully covered but booster dose was >5 years back, then tetanus toxoid 0.5 ml is administered. Intramuscular tetanus immunoglobulin is administered when tetanus cover is not known or in severe contamination and/or severe muscle damage.

Intravenous antibiotics – Co-amoxiclav (1.2 g) or cefuroxime (1.5 g) 8 hourly for 72 hours or till wound closure. Clindamycin 600 mg, 6 hourly is administered in cases of penicillin allergy. Metronidazole is added in cases of severe contamination with sewage or farmyard injuries.

Debridement can be done at up to 24 hours from injury unless significant contamination with sewage or farmyard injuries or associated injuries requires surgical management. Debridement includes excising the wound edges, extension of wounds in line with compartment release incision, bone ends are delivered, fragment edges are gently curreted to remove any debris and any loose fragments removed. Soft tissues that are not viable are also excised. Lavage of the wound is performed with warmed Normal saline via a giving set with a large bore. Pulse lavage is not used as pressurised fluid can push the debris further into the wound. Once the surgeon is happy that the wound is clean photographs are taken (for plastic surgeon to review). External fixator pins are introduced away from zone of injury in a 'near/far' configuration and fracture reduction is maintained with rods away from the wound. In cases of bone loss Gentamicin beads or calcium sulphate with antibiotics are introduced and vacuum-assisted wound closure is performed. Rest of the rods are connected to increase stability of external fixation.

Combined orthopaedic and plastic approach.

Definitive stabilisation of fracture and wound cover should be achieved within 3–7 days.

Usually, in type I, II and IIIA definitive stabilisation with intramedullary device and wound cover can be achieved.

In IIIB – Nail fixation can be performed if soft tissues and bone are in a satisfactory condition. If there is any doubt, circular frame fixation of the fracture is performed after wound cover is achieved.

Type IIIC injuries are to be dealt with as an emergency. Arteriography is not essential unless there are other proximal injuries and delay in achieving arteriography does not compromise ischemic time. On table arteriography is another option. Temporary vascular shunt, external fixation, debridement of the wound, vascular repair or reconstruction is performed. Definitive stabilisation and skin cover is based on the available vascular branches.

BOAST guidelines for severe lower limb open fractures are available online[1].

Mangled Extremity Severity Score

Mangled Extremity Severity Score (MESS):

Energy – Low, moderate, high, very high (1–4)

Ischaemia – Pulse present/absent but normal perfusion; pulseless with reduced capillary refill; cool, paralysed, insensate, numb (1–3)

Shock – Systolic >90 mmHg, transient hypotension, persistent hypotension (0–2)

Age – <30 years, 30–50 years, >50 years (0–2)

There are other scores but all the scores have high specificity (high probability of a low score leading to limb salvage) and low sensitivity (low probability of high score leading to amputation).

Decision to salvage or amputate the limb depends on presence or absence of other limb injuries or life threatening injuries, MESS, associated co-morbidities. If in doubt, salvage is a safe option with low threshold for later amputation in consultation with another surgeon which also gives patient more time for informed consent.

Injury Severity Score

The Injury Severity Score (ISS) provides an overall score for multiply injured patients using an anatomical scoring system. Body is divided into six regions (head, face, chest, abdomen, extremities (including pelvis) and external) and each injury is given an Abbreviated Injury Scale (AIS) score (0–6). Only the highest AIS score in each body region is used. The square of the three most severely injured body regions are added together to produce the ISS score. The ISS ranges from 0 to 75. An AIS of 6 (unsurvivable injury) in 1 body region automatically brings the ISS to 75. The ISS correlates with morbidity, mortality and hospital stay. However, several different injuries can add up to same ISS as different body regions' scores are not weighted.

Damage control orthopaedics (DCO) vs early total care (ETC)

DCO is performing just minimal surgical intervention that would assist achieving haemodynamic stability and nursing care[30].

Pathophysiology

Response to injury

Ebb phase – Fight or fright response and usually lasts for <24 hours. During this period release of catecholamines, activation of hypothalamus–pituitary–aldosterone and rennin–angiotensin–aldosterone axis occurs. All these responses tend to maintain circulating blood volume.

Flow phase is divided into catabolic and anabolic phases.

Catabolic phase usually lasts for 3–10 days and is characterized by increased BMR, increased temperature, breakdown of fats and protein (negative nitrogen balance) and insulin resistance. The purpose of this phase is continued maintenance of energy available.

Anabolic phase usually lasts 10–60 days. This phase is the recovery period with positive nitrogen balance when there is rebuilding of lost tissues.

Concept of first hit and second hit. The initial trauma of injury produces inflammatory response which is essentially the first stage of repair process but this can be aggravated by a second hit in the form of trauma due to surgical intervention. This second hit can compound the first hit and push the patient over to irreversible physiological damage including multiple organ failure and acute respiratory distress syndrome.

DCO is based on avoiding the second hit or minimizing the effect of second hit by performing only essential surgery which will allow overall care of the patient, control bleeding, remove devitalised tissues, reperfuse ischaemic tissues.

DCO is considered when the ISS is >20 and associated with chest trauma or >40 without chest trauma in difficult resuscitation, hypothermia and massive blood transfusion.

Cytokines, vascular endothelial cells interact to increase permeability of the capillary bed, especially in lungs, gut and muscles. In lungs interstitial fluid increases leading to pulmonary dysfunction, in gut there is translocation of bacteria leading to sepsis and in muscles increased interstitial fluid can lead to compartment syndrome. Commonly used biomarkers are serum lactate >2.5 mmol/l and IL-6 >5 μg/l which indicate that patient still not completely resuscitated.

Management (DCO)

Total surgical time is kept below 90 minutes. External fixation, cast application, reduction of dislocations and plate fixation all play a role in achieving temporary stabilisation of fractures.

Timing of definitive surgery is based on achieving haemodynamic resuscitation, serum lactate <2 mmol/l, no coagulopathy, normothermic, urine output >1 mg/kg/h, negative fluid balance and no inotropic support.

In general terms, polytrauma patients can be classified as stable, borderline, unstable and in extremis. Stable patients can undergo definitive surgical stabilisation of fractures as soon as possible. Borderline patients can undergo ETC, but plan can be changed to DCO if patient's condition deteriorates. Unstable patients are treated by DCO. In extremis patients require ITU care and possibly closed reductions of dislocations and external fixation of long bone fractures.

Principles of management of non-union

Generally, long bone fractures are considered to be non-unions if they have not healed by double the time required for the fracture to heal usually. Non-progression of radiological healing, pain and mobility at fracture site all point to non-union.

Patient factors

- Co-morbidities – Diabetes, steroids, smoking, rheumatoid arthritis. Type of host
- Previous surgery – Number of surgeries, approaches, infection, implant type, bone graft
- Local soft-tissue status – Poor skin, open wounds

Weber–Cech classification

- Hypertrophic (intact blood supply and mechanical instability)
- Atrophic (loss of blood supply and mechanical instability)

Host type in infected non-unions

- Cierny classification
  - Type A – Good immune system
  - Type B – Compromised systemically or locally

- Type C – Significant immune compromise that surgery is not contemplated

Goals of non-union management
- Achieve fracture union
- No shortening or mal-alignment
- Good function of the limb

Investigations
- Blood tests – FBC, ESR, CRP
- Imaging – Radiographs, CT and or MRI scans – To assess bony defects, osteomyelitis, arteriography in cases where plastic surgery is anticipated due to poor soft-tissue status

Planning management
- Host type
- Soft-tissue status – Coordinate with plastic surgeon
- Bone factors – Alignment, shortening, bone loss, joint stiffness

Options

No previous surgery
- Hypertrophic non-unions in good alignment and no shortening or gap
  - Functional cast brace and or electromagnetic stimulation or LIPUS (**L**ow **I**ntensity **P**ulsed **U**ltrasound)
  - Nail fixation for diaphyseal non-union
  - Plate fixation for metaphyseal/diaphyseal junction non-union
- Atrophic non-union, shortening, gap, mal alignment, poor soft tissues
  - Circular frame fixation for bone transport or lengthening or gradual correction of mal-alignment

Previous surgery
- Investigations to rule out infection and other investigations as described above
- Aseptic non-union
  - Single-stage revision fixation (fixation options as above) with or without augmentation with autologous bone graft or demineralised bone graft or recombinant bone morphogenic protein (BMP)
- Septic non-union – Staged procedure
  - First stage – Removal of implants, resection of devitalised bone and soft tissues, local and systemic antibiotics, temporary spanning external fixation. Serial blood markers of infection, blood cultures, Repeat debridement if required. Temporary vacuum-assisted dressing
  - Second stage – Soft-tissue cover by plastic surgery, if required
  - Third stage – Definitive procedure with either plate or nail or circular frame as discussed above

Fracture non-union management is a subspecialty and needs a dedicated multidisciplinary team including surgical team, physiotherapists, occupational therapists, specialist nurses, microbiologist, pathologist and radiologist.

Other trauma scenarios to consider

- THA presents with a first-time dislocation
- Osteochondral fracture of the knee
- 15-year-old boy club football player with acute ACL injury
- Tibial plateau fracture with associated tibial pilon feature
- Draw Hamilton–Russell traction for a femoral fracture
- High-energy midshaft tibial fracture and chest injury
- Inferior shoulder dislocation
- Treatment of distal biceps rupture and describe surgical approach
- Tibial tuberosity avulsion in a child
- Segond fracture
- Secondary prevention of osteoporosis and effects on bone healing
- Ankle instability
- Intertrochanteric fracture non-union
- Pathological fracture of humerus
- Femoral neck fracture in Paget's disease
- Late presentation of shoulder dislocation
- Multiple metatarsal fractures and calcaneal fractures
- Tibial spine fracture
- Posterolateral corner injury of knee
- Dislocated knee
- Long bone non-union with in situ implant

Examination corner

Case 1

Radiograph of pelvis and right femur in a 20-year-old female passenger involved in a RTA.

CANDIDATE: Described the radiographic findings, displaced right femoral midshaft fracture, minor comminution.

EXAMINER: What will you do?

CANDIDATE: Assess patient according to ATLS® protocols.

Before candidate could proceed . . .

EXAMINER: Patient is stable, no head, neck, spine, chest or abdominal injury.

CANDIDATE: Is it a closed injury? Any distal deficits? Any other areas of tenderness in knee, tibia, ankle or foot?

EXAMINER: Closed injury and no other distal injuries.

CANDIDATE: Femoral shaft fractures are best treated operatively. I will stabilise this fracture with a reamed femoral locked nail.

EXAMINER: OK. This is the postoperative radiograph. What do you think?

CANDIDATE: Radiographs show locked femoral nail fixation. Alignment, nail and screws lengths are satisfactory.

EXAMINER: You go to see the patient on the first postoperative day and patient is breathless. What do you do?

CANDIDATE: My worry is whether this is fat embolism. I will assess patient's respiratory status by getting history as to when the breathlessness started any chest pain or palpitations. I will examine patient for respiratory rate, air entry bilaterally, tracheal position, Oxygen saturation, pulse rate, blood pressure, what percentage of oxygen patient is on. Obtain ECG, chest radiograph, ABG immediately. Contact medical on call team and ITU for further medical assessment.

EXAMINER: Patient did have PE but recovered and was sent back to orthopaedic ward 4 days later. In the ward physiotherapist is concerned that the patient is not weight-bearing due to pain and is limping. What will you do?

CANDIDATE: I will get a full length radiograph of right femur AP and lateral views.

EXAMINER: This is the radiograph.

CANDIDATE: Radiographs show a displaced subcapital femoral neck fracture.

EXAMINER: What to do now? Do you want to look at the first radiograph?

CANDIDATE : Yes *(after looking at the first radiograph)*. Even retrospectively looking at this radiograph I am not able to identify a femoral neck fracture.

EXAMINER: Yes, why is there a fracture now and what to do now?

CANDIDATE: It may have been an undisplaced fracture that has displaced on weight-bearing or iatrogenic fracture as the femoral nail entry point is in the piriformis fossa. Even though it may be a few days since femoral neck fracture due to patient's age I will still try to reduce and stabilise the fracture. Options are:

1. Closed reduction and screw fixation with miss a nail technique exchange the femoral nail for a reconstruction type nail
2. If closed reduction fails, then, open reduction via anterolateral approach and fixation as above

EXAMINER: Could not do miss a nail screw fixation so I exchanged the nail *(shows radiographs)*. What will you do?

CANDIDATE: I will allow toe touch weight-bearing for 6 weeks followed by increasing weight-bearing over the next 6 weeks to full weight-bearing and obtain serial radiographs at 2, 6, 12 weeks and 6 monthly till 2 years follow-up looking for AVN of femoral head and non-union of femoral neck fracture.

Good points – Answered all questions directly without beating around the bush. Candidate had practised for a similiar viva scenario
Could have done better – Looked for femoral neck fracture but failed to mention that I looked for it

Opportunities for other questions – If femoral neck fracture was identified prior to surgery, then surgical options should be discussed. Either single or two implants but femoral neck fracture is the more important of the two fractures. Timing of nail fixation of femoral shaft fractures. Reamed or unreamed nail. Dynamic or static locking.

Case 2

EXAMINER: This 20-year-old injured his left tibia while playing football.

CANDIDATE: Transverse fracture of left tibia midshaft and fibula with >50% cortical contact in both views. Angulation appears to be around 20°.

EXAMINER: How will you treat this fracture?

CANDIDATE: If this is an isolated closed fracture with no distal neurovascular deficits and no compartment syndrome, then, options of management are manipulation and above knee cast or locked nail fixation. I will discuss the advantages and disadvantages of both with patient.

EXAMINER: What are the important advantages and disadvantages of both treatments?

CANDIDATE: In cast treatment there are no risks of infection or anaesthetic-related complications but prolonged immobilization of limb, frequent radiographs and mal-union are risks. In nail fixation, advantages include early mobilization of knee and ankle, reduced risk of mal-union but risks include infection, compartment syndrome and anterior knee pain.

EXAMINER: Patient opts for cast treatment. What will you do?

CANDIDATE: If patient can tolerate it, I will reduce and apply a moulded above knee cast in plaster room under entonox. If not, then in theatre. Weekly radiographs for first 3–4 weeks and if there is satisfactory early callus formation, I will apply patellar tendon weight-bearing cast and allow full weight-bearing. Cast treatment will continue till there is three out of four cortical bridging callus and there is no pain or abnormal mobility at fracture site.

EXAMINER: This is the radiograph at first follow-up.

CANDIDATE: Radiographs show satisfactory bony contact but there is valgus angulation of about 20°.

EXAMINER: What will you do now?

CANDIDATE: Since it is only angulation deformity, I will wedge the cast laterally and get another radiograph.

EXAMINER: This is the radiograph after wedging.

CANDIDATE: Alignment and angulation is satisfactory. So, will continue cast treatment with weekly radiographs.

EXAMINER: What do you mean by satisfactory alignment?

CANDIDATE: There is >50% cortical contact, angulation <10° and no shortening. Rotational alignment has to be checked clinically.

EXAMINER: Yes. This is radiograph of another tibia fracture. What do you think?

CANDIDATE: Locked nail fixation with satisfactory alignment but there is minimal distraction at fracture site.

EXAMINER: this is the clinical photograph of leg at 2 weeks post surgery.

CANDIDATE: There is external rotational deformity of the tibia.

EXAMINER: What will you do?

CANDIDATE: I will assess the external rotation deformity and compare it to the opposite leg. If thigh foot angle difference is >15–20°

EXAMINER: Between two sides I will offer revision surgery after ruling out infection by examination and blood tests with FBC, ESR and CRP.

EXAMINER: What surgery will you do?

CANDIDATE: I will remove the distal locking screws and correct the rotational alignment by rotating the distal fragment on the nail and insert the locking screws again.

Good points – Candidate had prepared for this scenario and had decided on what his/her cut off points would be for acceptable fracture alignment and had back up plans if Plan A did not work

Case 3

EXAMINER: A 30-year-old skate boarder fell onto left shoulder and this is the radiograph.

CANDIDATE: Radiographs show transverse fracture of midshaft left clavicle with shortening of about 2 cm.

EXAMINER: What will you do?

CANDIDATE: I will offer surgery in the form of open reduction and plate fixation.

EXAMINER: Why?

CANDIDATE: The shortening of clavicle leads to shortening of the moment arm of shoulder and hence, the strength of shoulder abduction.

EXAMINER: Do you have any evidence for this?

CANDIDATE: Yes. Paper published in . . . showed . . .

EXAMINER: How valid is that paper? What are its drawbacks?

CANDIDATE: Err, don't know.

EXAMINER: OK. What are the indications for fixation of clavicle fractures?

CANDIDATE: Open fractures, polytrauma, multiple fractures in same limb, neurovascular injury, shortening >2 cm, symptomatic non-union.

EXAMINER: OK. What are the risks of plate fixation of clavicle?

CANDIDATE: Infection, wound healing problems, vascular injury, prominent plate, numbness distal to scar, plate breakage.

Good points – Had a plan for this injury and did not beat around the bush
Could improve – When quoting any reference, it is necessary to have read more than just the abstract and to be able to critically appraise the paper and give reasons as to why you would accept or not accept the conclusions offered. In the paper quoted there were high rates of complications in the operated group, secondary surgery to remove plate and screws

Case 4

EXAMINER: This 40-year-old motor bike rider fell off the bike at 50 miles/hour. This is the pelvis radiograph.

CANDIDATE: Pelvic radiograph is not adequate but from the available radiograph there is pubic symphysis widening. I will assess the patient according to ATLS® protocol and get an AMPLE history.

EXAMINER: If the patient is haemodynamically unstable what will you do?

CANDIDATE: Obtain intravenous access with two large cannulae and get blood samples for FBC, U&E, Grouping and cross-match 6 units urgently and infuse 2 litres of crystalloids. I will look for areas where patient could be losing blood – Chest, abdomen, long bone fractures, and open injuries. Chest x-ray will show presence of haemothorax or mediastinal injury. FAST scan will show any free fluid in the abdomen.

EXAMINER: FAST scan shows free fluid in abdomen and surgeons want to take patient to theatre straight away. What will you do?

CANDIDATE: Before they perform laparotomy I will apply an external fixator such that it will not interfere with laparotomy incision.

EXAMINER: Let's go back to assessing patient in A&E. Patient is haemodynamically stable. How will you assess the pelvic injury?

CANDIDATE: I will look for any areas of bruising or skin injuries, perineal or urethral injuries and perform per rectal examination to assess for any rectal injuries.

EXAMINER: Will you do anything else? How will you know whether pelvis is stable or not?

CANDIDATE: Compression distraction test will show pelvic instability but it can dislodge any pelvic clots and make patient haemodynamically unstable.

EXAMINER: What first-aid measures can be done for this pelvic injury?

CANDIDATE: Pelvic binder.

EXAMINER: How will you apply the binder and what will you do after applying the binder?

CANDIDATE: Binder is applied around the greater trochanter area and I will get a pelvic x-ray to check whether the pubic symphysis is reduced.

EXAMINER: What is the definitive management of this injury?

CANDIDATE: Definitive management of this injury is pubic symphysis plate fixation.

Good points – Candidate had good insight into immediate assessment and management
Could improve on – In discussing definitive management, CT scan should have been mentioned as it will assist in deciding whether posterior stabilisation is also needed or not

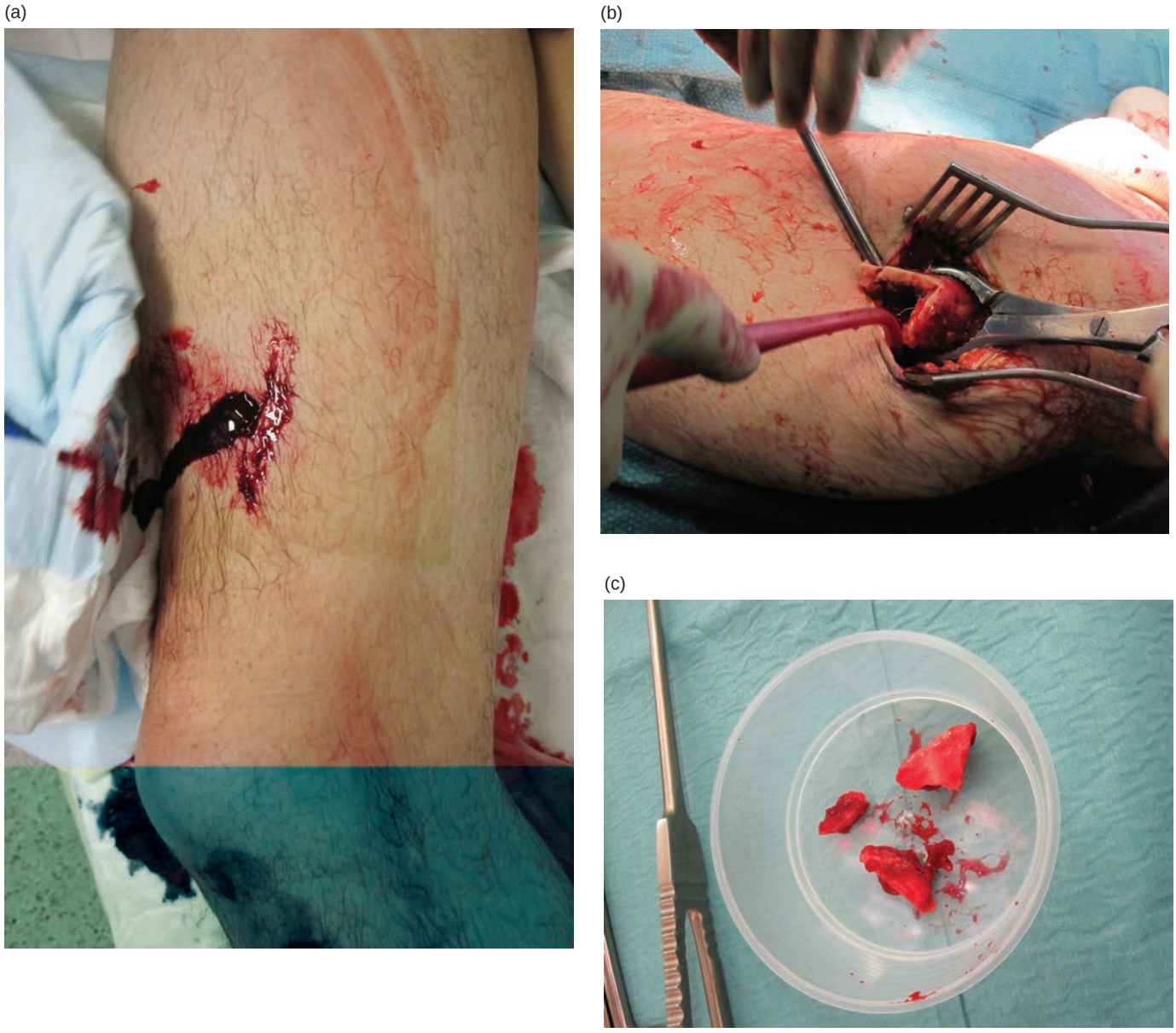

Figure 28.14 (a) Open femoral shaft fracture. (b) Fracture end is delivered and debrided. (c) Photograph showing comminuted bone fragments with no soft-tissue attachments that were removed

Further discussion – Uncontrolled haemodynamic instability protocol, massive transfusion protocol, pelvic fracture classification – Tile, Young and Burgess

Trauma case 5

EXAMINER: A 28-year-old motorcyclist travelling at about 40 miles/hour skidded and came off the bike. These are his right femur radiographs.

CANDIDATE: Radiographs show a distal third shaft of femur fracture that is completely displaced. It is mainly transverse type with some comminution.

EXAMINER: What will you do next?

CANDIDATE: I will assess patient according to ATLS® protocol. If patient is stable and with no other injuries, it will require internal fixation with nail fixation.

EXAMINER: This is the clinical photo of patient's thigh (Figure 28.14 a–c)

CANDIDATE: It shows a possible puncture wound that is oozing blood. This is an open fracture.

EXAMINER: How will you grade it?

CANDIDATE: According to Gustilo and Anderson grading it will be III whether it is A or B will be decided after debridement.

EXAMINER: How will you manage this patient?

CANDIDATE: IV fluids, analgesia, IV antibiotics, splint, early debridement of the wound and stabilising the fracture.

EXAMINER: How soon will you do the surgery and what are the steps involved?

CANDIDATE: BOA guidelines do not recommend the 6-hour rule anymore. I will do surgery as soon as possible. It will involve extending the wound, debriding the wound and removing any non-viable tissues. After changing instruments and rescrubbing I will stabilise the fracture with a femoral nail, if the wound is clean. If there are any signs of contamination, I will perform external fixation to stabilise the fracture temporarily and continue IV antibiotics.

EXAMINER: Wound is clean, what type of femoral nail will you use – Reamed or undreamed, antegrade or retrograde and why?

CANDIDATE: If wound is clean I will use a reamed antegrade nail. Reamed nail will allow me to use a nail that is thicker and hence, stronger. Antegrade nail to avoid involving the knee joint.

EXAMINER: What do you do with the wound?

CANDIDATE: I will leave the wound open and check the wound again in theatre at 48 hours. If it is clean I will close the wound.

Good points –Covered most of the aspects of open fracture management
Could have improved –Instead of waiting to be prompted could have explained in detail about surgical management options depending on the wound status and definitive stabilisation. Mention the possibility of associated femoral neck fracture and assessment of knee joint at the end of the procedure
Further discussion –Type of antibiotic, length of antibiotic cover, BOA guidelines in depth, recent advances in open fracture assessment[31], associated femoral neck fracture

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Chapter 29

Applied trauma oral topics

Jonathan R. A. Phillips, William Eardley and Paul Fearon

The trauma viva is sometimes regarded by candidates as 'the one' that caught them out. This really should not be the case as with a bit of preparation, familiarity with the type of question asked, refining your technique and understanding general trauma principles this viva should be nailed. This chapter drills down onto more specific questions that regularly pitch up in the viva. We begin by exploring the principles of intra-articular fracture management.

Distal femoral fractures, tibial plateau fractures and pilon fractures

Intra-articular lower limb fractures are grouped together to avoid repetition. As with all intra-articular injuries, there are many avenues that the examiners may take a candidate in the viva through. Applied anatomy, classification, early management and fracture healing principles are all fair topics on which to concentrate. The candidate must not jump straight into 'This is a Schatzker III fracture, I would plate it'. This is categorically not what is required. Clear descriptions, acceptance of potential for other bony and soft-tissue injuries, sensible early management and a sound grasp on how you wish to encourage this fracture to heal and how to rehabilitate your patient is paramount.

The following points will assist in developing this framework, taking the tibial plateau as an example. Like many fractures, it may be divided into low-energy (insufficiency) injuries and high-energy (traumatic) injuries. Whilst seemingly basic, this approach allows for a reasoned evaluation of the injury in front of an examiner.

High-energy injuries – The young motorcyclist with a medial tibial plateau fracture – Are a spectrum of injury intensity away from the osteoporotic elderly lady who stumbles getting off the bus, sustaining a depressed lateral plateau injury.

The unwary candidate will neither assess nor deal with these injury profiles differently. They are both fractures of the tibial plateau, but this is the only similarity they share.

The medial tibial plateau is stronger and its fracture carries a greater association with soft-tissue injury. Young adult bone more often demonstrates a simple split with associated ligament damage where the osteoporotic yields depressed fracture types without ligament injury in the generality. The position of the leg, the direction and quantity of force and the degree of knee flexion at time of injury also contributes.

All of these factors need to be considered and the candidate should be ready to volunteer this information early in the question whilst discussing the fracture. This will impress the examiners and result in a smoother experience overall. (Figure 29.1 a and b)

Evaluation

The energy of injury will dictate the approach. A full ATLS® workup is required for the high-energy fracture – This may be overkill for a fracture resulting from a stumble, however. The examiners are looking for this distinction as it identifies the strong candidate from one with a purely formulaic approach.

The skin condition should be assessed and the distal pulses should be documented with a hand held Doppler. Documentation of the integrity of the common peroneal branches and the tibial nerve in the foot is essential. Compartment syndrome may occur and appropriate assessment for this is required. A high index of suspicion for meniscal and ligament injury must be maintained. Similar points need to be addressed with pilon fractures also.

Following initial resuscitation and evaluation, stabilisation with above knee plaster and analgesia, two plane radiographs of the joint including the full tibia and ankle are taken. A CT scan is a necessity as it helps to evaluate the joint surfaces, delineating the articular fracture fragments and revealing occult fracture lines, unseen on plain films (Figure 29.1c–f). This rationale should be given to the examiners, don't just state that you would *'get a CT'*. If the skin/soft-tissue envelop is contused/bruised and swollen, the application of a spanning external fixator is highly desirable, in order to stabilise the fracture configuration, and allow the soft tissues to recover. Be prepared to talk through how you would apply a spanning external fixator to the knee/ankle region. It's only then should a CT scan be requested for surgical planning.

Classification

Another favourite – The Schatzker method of classifying these injuries must be committed to memory. Types IV–VI are

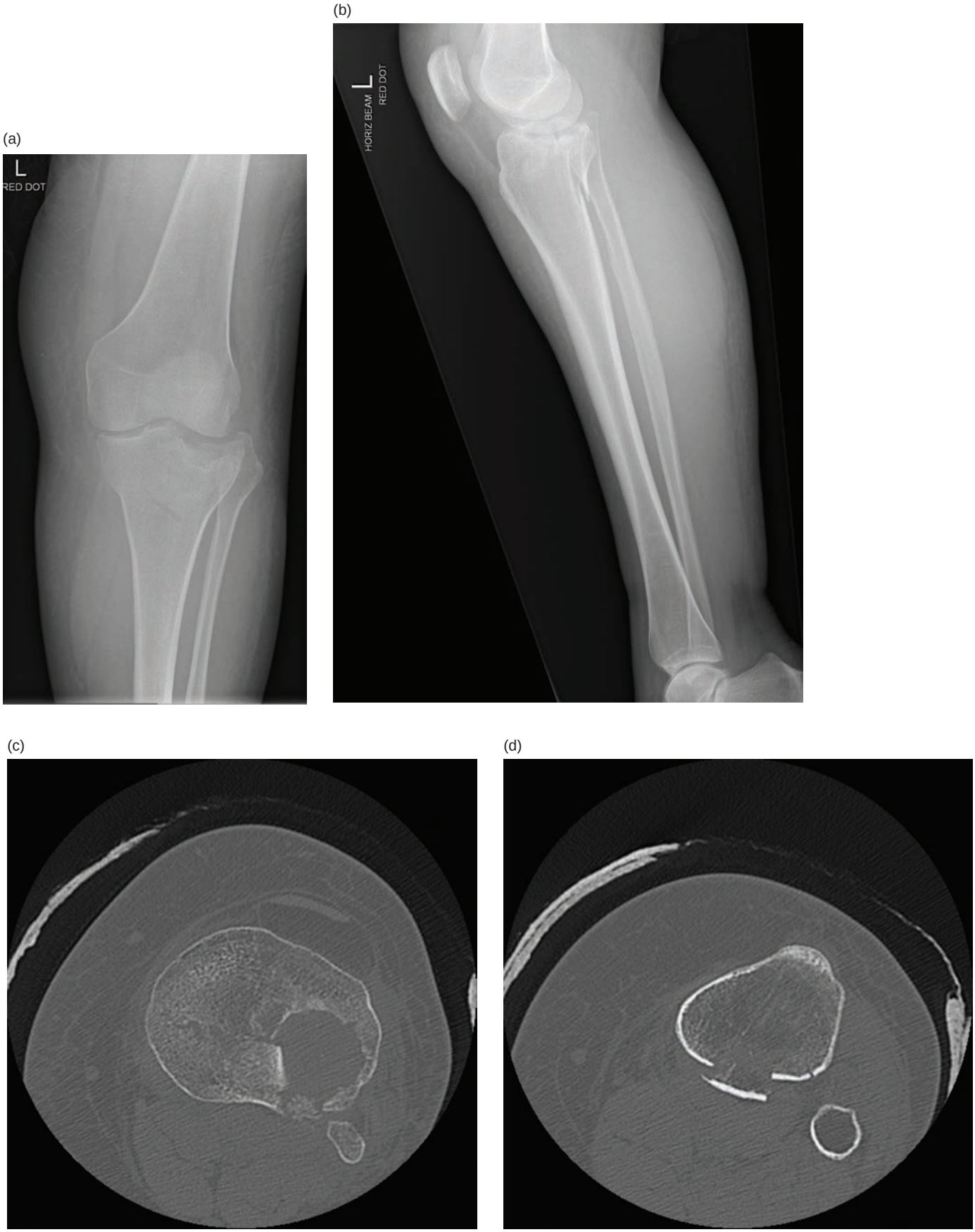
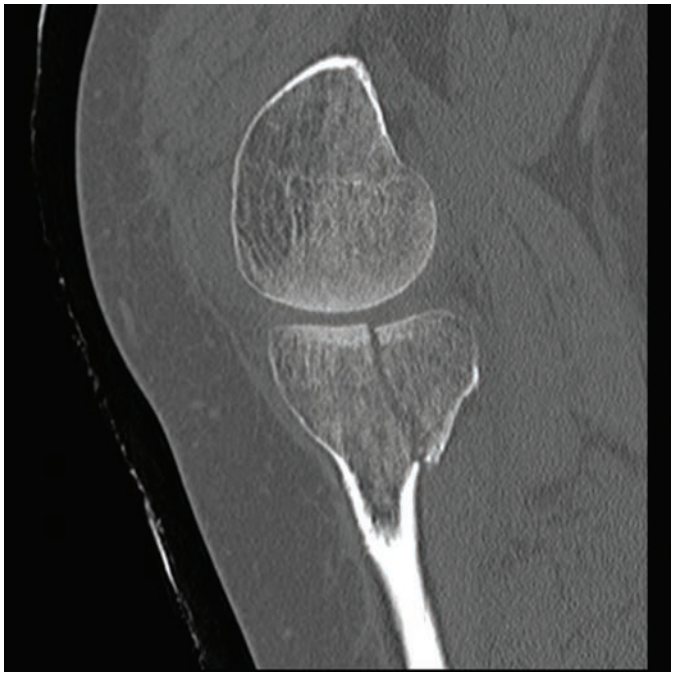


Figure 29.1 AP (a) and lateral (b) radiographs of complex tibial plateau fracture in an obese middle-aged patient. (c–f) CT of knee in bone window in axial (c, d), sagittal (e) and coronal (f) reformations. (g, h) AP (g) and lateral (h) postoperative radiographs showing dual plating of tibial plateau fracture

(e)



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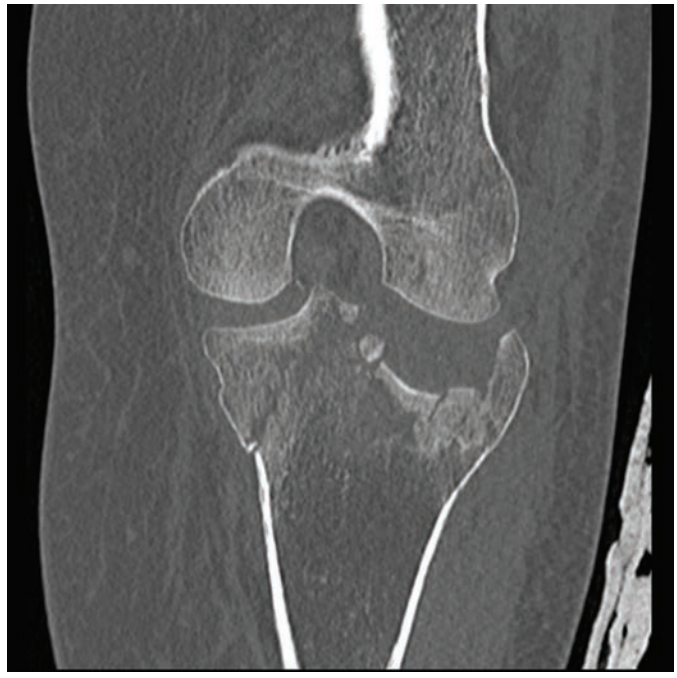
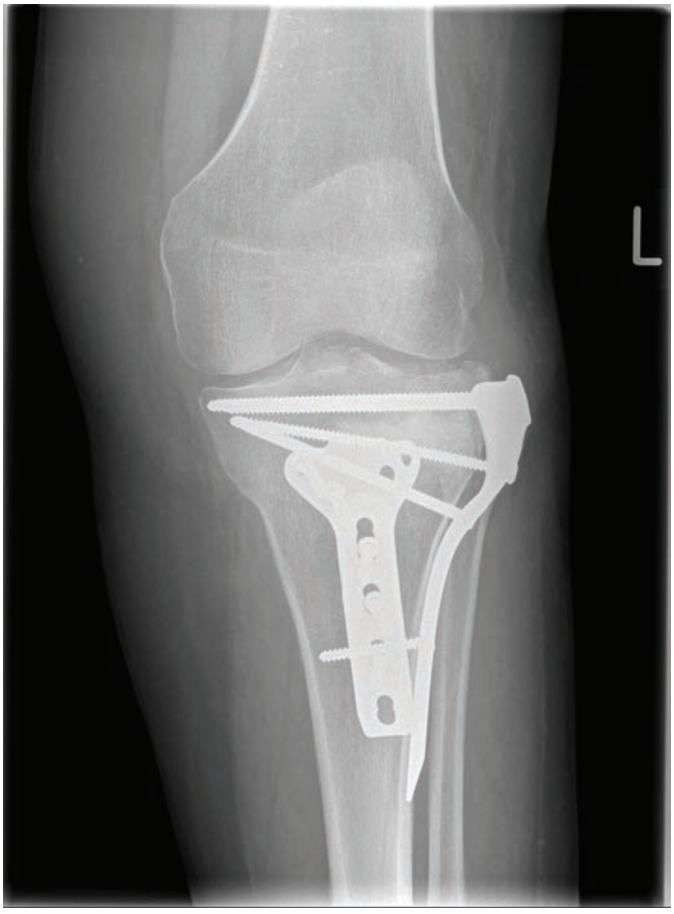


(g)



(h)

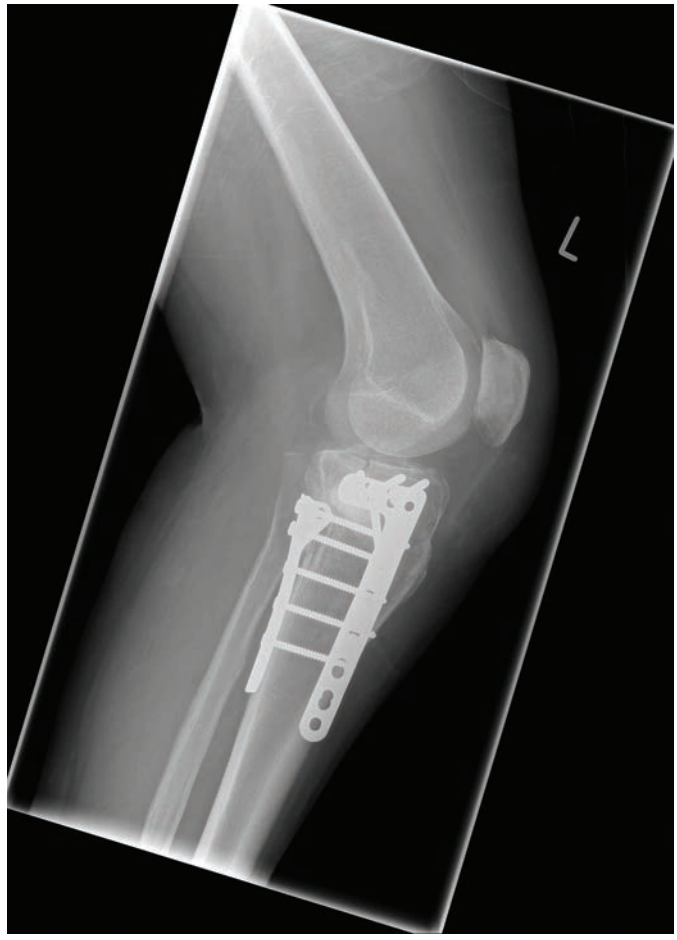


Figure 29.1 (*cont.*)

associated with increased energy and neurovascular injury. Medial meniscal tears are most commonly seen with type IV and lateral meniscal injury with type II, the overall most common fracture type.

I Split of the lateral plateau
II Split and depression of the lateral plateau
III Depression of the lateral plateau
IV Medial plateau fracture
V Fracture involving both the medial and lateral plateau
VI Tibial plateau fracture with an associated shaft fracture distally – A complete metaphyseal/diaphyseal separation

Associated injury is common, especially with the high-energy injuries. Overall 50% of plateau fractures will have a meniscal tear, 30% will have an associated ligament injury and up to 50% of young high-energy injuries will have disruption of the anterior cruciate ligament.

Management

The key to the treatment of intra-articular fractures, once associated injury is ruled out, is the resultant stability of the joint and articular alignment. Laboratory studies suggest tolerances of around 3 mm of articular step off in the knee but it is the effect of articular depression and widening on stability and the effect of fracture displacement on the mechanical axis that matters.

Non-operative management may be considered for some pure depression injuries due to the likely integrity of the ligaments and also the retained cortical rim of bone to prevent varus/valgus collapse. Examination under anaesthesia and long leg casting converted to protected mobilization in a hinged brace is an option for these injuries.

The more unstable fractures involving a significant joint disruption or metaphyseal discontinuity are normally treated operatively. The goals of surgery are anatomical restoration of the joint line with absolute stability, connection of the articular block to the diaphysis and early mobilization. There is cited an articular step off of 3 mm and joint widening of >5 mm and whist these are slightly arbitrary they represent increased instability and so may be used in the theme of this answer.

The surgical approaches and technique must be learned – Be able to detail in brief the approach to the knee and ankle, including the less well-known ones. Marks can be gained by demonstrating awareness of the planning needed for these cases. Review of the CT scan to enable fragment specific surgery, tourniquet application, use of a radiolucent table and correct positioning of the image intensifier, availability of a femoral distractor, etc are points that you want to get across to the examiner when detailing your operative plan. DO NOT just go straight for 'I would plate this fracture'. In this illustrated example there is a posterior and anterolateral fracture component, with a medially exiting fracture line also (Figure 29.1c–f). In this case the patient was positioned prone initially and the fracture buttressed, using a posterior plate applied after a tenotomy of the medial head of gastrocnemius. The patient then had a standard anterolateral approach allowing joint elevation, grafting and plating (Figure 29.1 g and h).

The high-energy intra-articular injury may lead to a discussion on many aspects of trauma management. Following this theme of evaluation, stay broad and flexible in the initial approach and use overall stability and the effect on the mechanical axis to guide your response. Remember, arthroplasty is always an option and discussing the case with a senior arthroplasty colleague is an appropriate management step in the elderly osteoporotic highly comminuted injury.

Diaphyseal femoral/tibial fractures

There are no 'easy' questions and so beware falling into the trap of relaxing if you get shown a fractured femur or tibia. This injury is a starter for a possible multitude of question directions. Fracture healing, implant selection, soft-tissue management, compartment syndrome and late reconstruction are all appropriate and are just some of the associated topics. The candidate must stay flexible and not dive straight into addressing the fracture in isolation without listening to the examiners and ensuring you are answering their questions.

Initial management

After detailing the salient points in the history such as the mechanism and overall injury pattern, candidates must be able to provide a concise early management plan.

Points which must be mentioned are assessment of the limb from a perfusion perspective, a neurological perspective and a soft-tissue perspective. Instead of saying 'I would assess the limb' be specific as to the action you would actually take.

Circulation is addressed through seeking and documenting pulsatile flow on hand held doppler in both the dorsalis pedis and posterior tibial arteries. Sensation in the named distribution of the branches of the common peroneal and tibial nerves is documented. Power on an MRC scale is noted in the long toe extensors and flexors. Soft-tissue trauma is detailed according to the classification of Tscherne and the 1984 modification of Gustillo–Anderson for closed and open fractures respectively. It is important to caveat that, whilst the latter is widely used, it is only intended to be used following initial surgical debridement and so it is worth mentioning this if using it to answer a question.

Detailing these points rapidly in the above manner demonstrates experience in the structured management of lower limb trauma. It establishes a baseline against which further clinical change can be contrasted should concern arise regarding compartment syndrome for instance.

Following assessment, the basic measures of splinting/immobilization, elevation, prescription of analgesia and antibiotics where appropriate and admission with serial clinical assessment for compartment syndrome completes the early management.

Definitive management

Following initial assessment, definitive orthopaedic management of the fractured long bone with consideration of the soft tissues can be addressed. Non-operative management is acceptable in closed, low-energy, minimally displaced tibial fractures. Alignment of these fractures may be considered acceptable if there is:

- <5° angulation in the coronal plane
- <10° angulation in the sagital plane
- <15° rotation
- <1 cm shortening
- >50% cortical apposition

These figures are to guide management only. It must be remembered that sagital plane deformity is much better tolerated than that in the coronal plane due to compensatory movement of the hip and knee in the sagital plane and the asymmetric joint loading seen in coronal deformity.

Non-operative management for tibial fractures consists of the application of a long leg cast in the operating theatre under general anaesthesia in which the patient remains for 4 weeks before conversion into a patellar bearing Sarmiento cast. Whilst healing times will vary, the patient must be aware that splinting may need to be continued for 4–5 months before the fracture healing occurs and that in the early phase, weekly attendance at fracture clinic is required.

Operative management

The majority of femoral fractures will require surgical stabilisation whereas a large number of tibial fractures may be treated in a cast. There are a number of situations where operative management may be chosen. These include: Patients that have failed non-operative management, those in which the initial deformity exceeds accepted tolerances or where there is a same level fibula fracture, open injuries, fractures associated with nerve or vessel injury, segmental fractures and fractures with compartment syndrome.

In addition to the above, operative management of simple tibial fractures (i.e. those that may be considered for casting) has been shown to decrease time to union, to be associated with a faster return to activities of daily living and whilst there is an increase in superficial wound problems in this group compared to non-operative management, there is minimal deep-tissue infection risk.

For unstable fractures with unacceptable alignment tolerances (Figure 29.2a and b) complex distal third tibia and fibula fracture with x-rays showing AP view of knee and lateral of ankle) comparing cast treatment to intramedullary nailing, the latter is associated with decreased time to union, decreased hospital stay, decreased time off work, lesser angular deformity, less shortening and less secondary procedures. There is a balance of increased knee pain in the operative group of around 50% of cases, which is independent of the approach used to the nail entry point.

Implant selection

Having resuscitated and ensured optimum early management, the question will come, 'So what are you going to do'? Don't fall into the trap of 'I'd nail it'.

This question requires another prompt, concise approach to the generic preoperative assessment of trauma patients including full informed consent. It is not a one-line 'implant of choice' question.

Time is short and a full preamble of preoperative work up will both bore the examiners and prevent you from scoring further marks. Again, it is useful to acknowledge a structured approach but choose an element in particular to focus on. If the examiners don't want this, they will simply guide you in the right direction, it is better to offer knowledge than to have it drawn out of you.

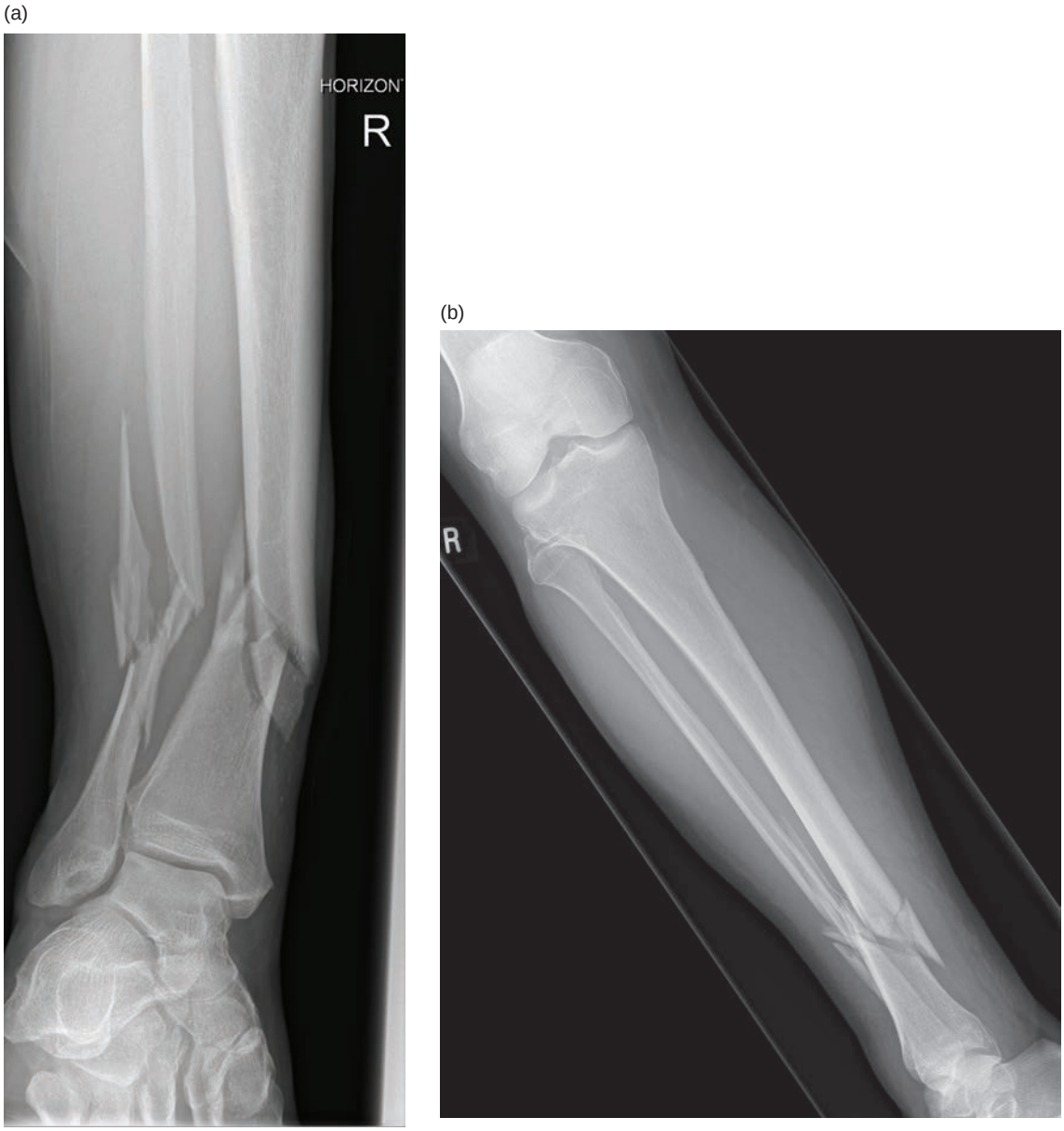
There is no right answer when it comes to fracture fixation, it is a matter of supporting your treatment philosophy with a sound rationale based on both experience and appropriate literature.

Be careful, therefore, not to appear inflexible with regard to implant choice. It is important to show an awareness of the range of options but to quickly focus on your particular management plan. This demonstrates to examiners a level of experience and reassures them that you have more than one way of dealing with tibial fractures whilst quickly moving you on to score points on areas where you can demonstrate your knowledge. The example in Figure 29.2 a and b had marked soft-tissue blistering and was not appropriate for plating, leaving the options of external fixation or intramedullary nailing as definitive treatment. However, maintaining reduction while reaming and nail insertion can be problematic and technically demanding, so be prepared to explain what works best in your experience (Figure 29.2 c and d).

In general, an interlocked intramedualllry nail is the treatment of choice for diaphyseal tibial fractures as compared to plating, there is less associated disruption of the periosteal blood supply whilst the implant affords a load sharing capacity of relative stability and faster union time. Caution must prevail if the fracture involves the distal third of the tibia where the evidence for implant choice is less conclusive and is currently the subject of a multicenter randomised controlled trial (FIXDT).

When using an intramedullary nail for diaphyseal fracture fixation, the nature of reaming is an important point to address and is an area of opportunity to demonstrate a wider knowledge base.

Reaming the medullary canal enables increased contact between the nail and the bone and allows for the use of implants of greater diameter and potentially larger locking bolts, both factors which increase stability. Biologically however, the potential benefits of 'reaming debris' in an osteogenic capacity, whilst often cited are not proven. In addition, reaming further disrupts the internal, endosteal blood supply although this is reversible. Entry point is crucial, depending on the type of nail (trochanteric femoral nail will have a lateral bend proximally, compared to the straight piriformis nail); however, examples of



Figures 29.2 (a, b) AP and lateral radiographs of complex distal third tibia and fibula fracture. (c, d) AP and lateral radiographs following reamed tibial IM nailing

poorly reduced fractures and complications are still frequently seen due to a lack of understanding of the implants being used.

In order to address the question of reaming in tibial shaft fractures and of use for answering questions, candidates are advised to be aware of the Study to Prospectively Evaluate Reamed Intramedullary Nails in Patients with Tibial Fractures (SPRINT) trial. To summarize, this large, multicentred study demonstrated that there was, in closed tibial fractures a possible benefit for reamed intramedullary nailing when endpoints of late surgical intervention or autodynamisation are used. No benefit is seen with open fractures.

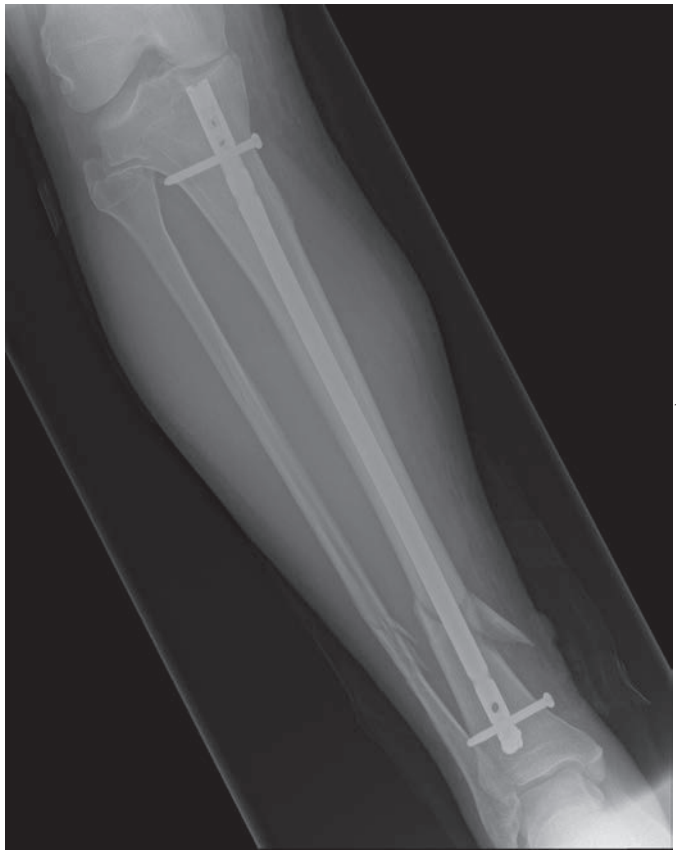
In addition to reaming, the particular behaviour of both proximal and distal shaft fractures when treated with intramedullary nailing is worth mentioning in questions relating to tibial fractures. Proximal tibial fractures when nailed have a tendency to go into procurvatum and valgus and their distal counterpart into varus and recurvatum. An awareness of this is essential and may be addressed with correct entry site position (more lateral and posterior in proximal fractures), the use of blocking screws, unicortical plates or percutaneous clamp application to maintain reduction.

Calcaneal fractures

From the history assimilate whether low or high-energy injury, age, co-morbidity (diabetic, pelvic disease (PVD)).

Fall from height (scaffolding, ladder, wall) or road traffic accident (RTA).

(c)



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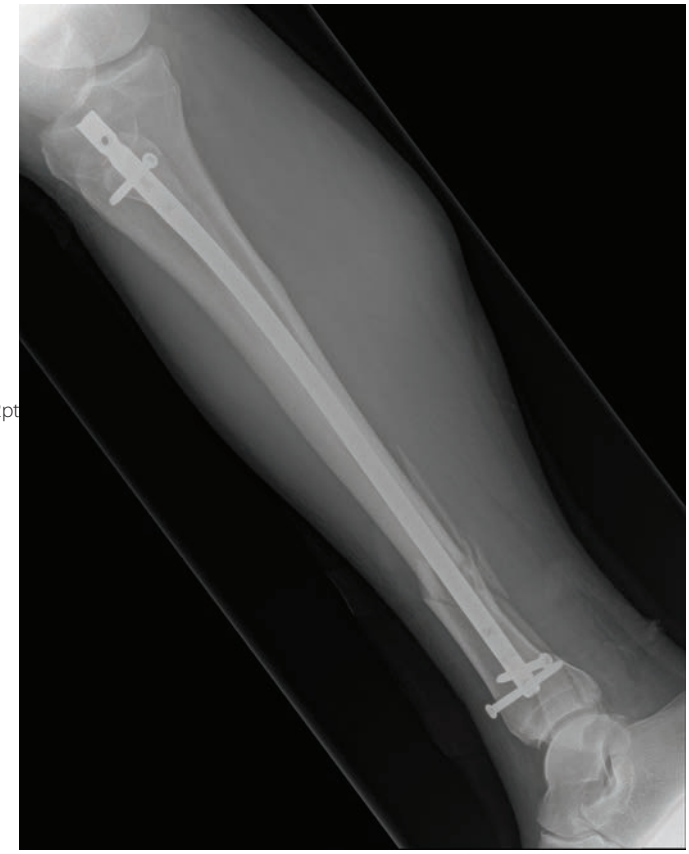

Figures 29.2 (*cont.*)

Associated injuries – Tibial plateau, tibial plafond, lumbar spine, acetabulum and contralateral os calcis.

- Following appropriate emergency care treatment and resuscitation according to ATLS® protocols, I would take a relevant history asking about . . .
- I would start by assessing the patient as a whole following ATLS® protocol[ab]. Associated injuries may include . . .

'Examination of the foot would include neurovascular status, any evidence of compartment syndrome in particular I would look for . . .'

'My initial management of this fracture would be . . . I would arrange urgent admission for elevation and more formal assessment of the fracture with an urgent CT scan.'

Be able to describe radiographs and CT scan.

- Bohler's angle is the angle between two lines joining the anterior process-posterior facet and superior tuberosity-posterior facet. This should be between 20° and 40°, decreased or even reversed with severe fractures
- Gissane's angle is formed by the intersection of two lines-the first along the downward slope of the posterior facet and the second running upwards towards the beak of the talus. Normal value is 95–105°, an increase in value suggests collapse weight-bearing posterior facet
- Be able to discuss primary and secondary fracture lines
- Know the Saunder's classification

Non-operative care includes elevation, application of ice, early mobilization, and the use of a splint.

Know some technical details about surgery[c].

Complications of conservative management include post-traumatic osteoarthritis (OA), peroneal tendinitis.

Complications of surgery include infection, skin necrosis and breakdown, non-union/mal-union.

There is a considerable controversy about the role of surgery. Grifin et al. reported the results of a pragmatic multi-centre randomised control trial comparing operative vs non-operative treatment of displaced intra-articular calcaneal fractures[1]. They concluded that no symptomatic or functional advantage existed with surgery at 2 years with the risks of complications higher following surgery. They did not

[a] This is an appropriate general statement that can equally be applied to a number of different fractures.

[b] If the examiners have already told you it is an isolated closed injury and you come out with this statement, at best you come across as not listening to what is being said and, at worst, just tactically limited in your ability to pick up clues.

[c] See minute 3, Chapter 8, Lower limb trauma, p. 116, Postgraduate Orthopaedics. Viva guide.

recommend ORIF of these fractures. They excluded open fractures and grossly displaced fractures.

In 2002 Buckley et al. published a prospective, randomised multicenter study involving 424 patients. They concluded that there was no overall benefit from surgery on the basis of two principle outcome measures: The visual analog pain scale and the SF-36.

However, stratification of the patient population distinguished certain features favouring surgery: Patients who were younger (<29 years), female, had a light or moderate workload involving the foot, those who were not receiving Workers' Compensation. The best patients to treat non-operatively are those who are 50 years or more, males, those who are receiving Workers' Compensation and have an occupation involving a heavy workload.

There are some methodological criticisms of this study including the fact that one surgeon performed the majority of operations (73%) and concerns with selection bias with the study using a pre-randomised strategy.

Agren et al. published a randomised control trial from five centres in Sweden. Outcome measures were similar to the Buckley paper. Eighty-two patients were randomised to open reduction with internal fixation (ORIF) or non-operative care. At 1 year there was no difference between the 2 groups. A recent Cochrane review concluded that there was insufficient high quality evidence to establish whether surgical or conservative treatment was better for adults with displaced intra-articular calcaneal fractures, and called for an adequately powered, multicentre controlled trial.

In summary, recent evidence-based studies suggest the results of operative intervention may be ineffective and, therefore, any patient with a significant risk of developing post-operative complications may be best managed conservatively.

Lisfranc injuries

These are rare injuries but a common topic for the trauma viva. Diagnosis and principles of treatment are important.

Examination corner

Trauma oral 1

EXAMINER: This is an AP radiograph of a cyclist who caught his foot in a toe strap and presents to casualty with forefoot pain[d]. You are called down to see him.

What do you think of the x-ray?

What do you look for on a plain film?

(Minute 1[e])

CANDIDATE: These are AP and 30° oblique radiographs of the left foot.

On the AP view:

There is diastasis of >2 mm between the base of the first and second metatarsals, features suggestive of Lisfranc tarsometatarsal fracture dislocation

There is a small avulsed fragment of bone in the Lisfranc space. This avulsion fracture is from the insertion of the Lisfranc ligament into the base of the second metatarsal-the 'fleck sign'

Medial border of the second metatarsal should be collinear/perfectly aligned with the medial border of the middle/intermediate cuneiform

The space between the first and second metatarsals bases should be equal to the space between the medial and intermediate cuneiforms[2,f]

On the 30° oblique radiograph in normal radiographs:

Medial border of the fourth metatarsal should also be in line (collinear) with the medial border of the cuboid[g]

The medial and lateral borders of the third metatarsal are perfectly aligned with the medial and lateral borders of the lateral cuniform

There is a compression fracture of the cuboid which supports the diagnosis of a Lisfranc injury[h]

Timpone described the 'intermetatarsal fat pad sign' on plain radiographs between the first and second metatarsals from oedema and hemorrhage from a Lisfranc ligamentous injury. This is similar to the sail sign of the olecranon

'I would like to see a lateral radiograph to check for dorsal displacement of the lateral metatarsals which would suggest ligamentous injury'

(Always ask for the lateral radiograph.)

(Minute 2)

EXAMINER: What is the Lisfranc joint?

CANDIDATE: This consists of three cuneiform and two metatarsal articulations. Joint stability is provided by strong ligaments and the recessing of the second metatarsal head.

EXAMINER: What is the Lisfranc ligament?

CANDIDATE: The Lisfranc ligament runs obliquely from the plantar aspect of the base of the second metatarsal to the plantar surface of the medial cuneiform.

EXAMINER: How will you manage this patient?

[d] The clue that this may be a Lisfranc injury is contained in the mechanism of injury.There is a twisting injury with forced abduction on the forefoot on the tarsus.

[e] In the lower limb trauma section of the viva book each question was treated as a chess game lasting 5 minutes and we have kept this format in this question. Some candidates found this different approach both useful and tactically challenging.

[f] This is getting into small detail.The examiners may move you along from this as they want to get to how you will manage the injury. However, it is part of the specifics of diagnosing the injury radiographically.

[g] Added for completeness. This radiographic feature is not always mentioned in textbooks.

[h] The examiners may move you along from this as they want to get to how you will manage the injury. However, it is part of the specifics of diagnosing the injury radiographically.

CANDIDATE: Following appropriate emergency care and resuscitation according to ATLS® protocols and assuming that this injury is both isolated and closed I would take relevant history: Mechanism of injury, patient's general condition, past medical history, allergies, smoking as well as occupation and previous level of activity.

Examination of the injured foot:

- Soft-tissue status, swelling, pain, tenderness and ecchymosis
- Painful passive abduction/pronation
- Neurovascular status, dorsalis pedis and posterior tibial pulse
- Tendon entrapment may be demonstrated with an altered, uncorrectable position of the toes or midfoot
- Compartment syndrome must be excluded

Following assessment, my initial management includes analgesia, elevation and splinting using a below-knee backslab.

On admission to hospital I would arrange for regular clinical examinations and monitoring in order not to miss an early developing compartment syndrome.

EXAMINER: What would you do if the radiographs were inconclusive in diagnosing this condition?

CANDIDATE: I would consider further radiographic imaging, a lateral view, stress views and a CT scan or may opt for an MRI scan.

EXAMINER: What is it – An MRI or CT scan?

CANDIDATE: An MRI allows direct visualisation of integrity of the Lisfranc ligament and surrounding soft-tissue structures. It is especially useful in low velocity injuries and in the setting of equivocal radiographic studies

A CT scan is more useful in high velocity injuries as it assesses degree of fracture comminution.

(Minute 3)

EXAMINER: How do you classify Lisfranc injury[i]?

CANDIDATE: Quenu and Kuss were the first to classify Lisfranc injuries (1909). They divided injuries into:

- Homolateral: All five metatarsals displaced in the same direction
- Isolated: One or two metatarsals displaced from the others
- Divergent: Displacement of the metatarsals in both the sagittal and coronal planes

This classification was modified by Hardcastle (1979) by dividing injuries into three categories: A, B and C. Type A injuries were complete displacement of all metatarsals (total incongruity) in the sagittal or transverse plane). Type B injuries were partially incongruous and type C injuries were divergent.

Myerson (1986) revised the classification dividing B and C injuries into subtypes 1 and 2.

Total incongruity (type A)

A – In any plane or direction

Partial incongruity (type B)

B1 – Medial (only the first ray is involved)

B2 – Lateral (one or more of the lateral rays) with the first metatarsal unaffected

Divergent displacement (type C)

C1 – Partial divergent (incongruence). The first metatarsal displaced medially and a portion of the lateral complex displaced laterally in the sagittal and/or transverse planes

C2 – Total divergent

EXAMINER: What is the mechanism of injury?

CANDIDATE: These injuries are most commonly high-energy injuries, but some can occur from low-energy twisting injuries.

Mechanism can be categorized as direct or indirect:

Direct – Crushing injury. Associated with soft-tissue injury and compartment syndrome (foot run over by a car)

Indirect – Axial loading of a plantar flexed foot or severe abduction, leading to dorsal ligamentous disruption. This typically occurs with falling on the heel of a plantar flexed foot, missing a step off a curb, falling off a horse with the foot in the stirrup

EXAMINER: How do you treat Lisfranc tarsometatarsal fracture dislocation?

CANDIDATE: This depends on severity of injury and degree of displacement of fracture. There is a role for non-operative management of an undisplaced stable injury or sprain that includes a non-weight-bearing cast for 6 weeks and regular clinical and radiological review. However, in the presence of subluxation or dislocation, then accurate reduction and stable fixation is essential. In this case, I would consider open reduction and internal fixation with screws and possible plating, as required.

Position is supine. Knee flexion allows plantarflexion of the foot for easier exposure and imaging. I would use two longitudinal dorsal incisions; one centered over the first web space and the other the fourth metatarsal. The skin bridge should be as wide as possible and the incisions should not be undermined. The key to success is to achieve initial reduction of the second metatarsal into its mortice between the 3 cuniforms. The other joints can then be reduced around it. Temporary stabilisation can be achieved with K-wires.

There has been a trend in recent years to be more aggressive in the management of this injury using compact foot plates or bridging plates instead of screw fixation. A bridging plate avoids articular cartilage damage with no loss of rigidity. This is especially helpful with a severely comminuted fracture/dislocation in which screw fixation is not possible. They can be used on the first, second and third metatarsals.

With a severely comminuted fracture or a late presentation (8–12 weeks), then primary arthrodesis of tarsometatarsal joints

[i] There has been a move away from rote-learning classification systems to concentrate more on evidence-based management.

may be required. Joint preparation is by standard methods and bone graft is often required.

Informed consent should be taken. The management options, postoperative rehabilitation, outcome and potential complications should be discussed in detail with the patient and documented in medical records.

EXAMINER: What are the complications from treatment?

CANDIDATE:

- Post-traumatic osteoarthritis
- Compartment syndrome
- Infection
- CRPS
- Neurovascular injury
- Metalwork breakage/migration

(Minute 4)

EXAMINER: What prognosis will you give this patient?

CANDIDATE: This is a serious injury with potentially a poor outcome. Residual pain and a stiff foot are not uncommon complications of this injury. Post-traumatic osteoarthritis is related to the initial injury and adequacy of reduction. It may occur in >50% of cases, even if well-fixed operatively

Patients must be informed about the length of treatment, recovery period and future implications for work and lifestyle. About 10–20% of patients will develop symptomatic arthritis requiring arthrodesis despite ORIF.

(Minute 5)

EXAMINER: If this patient developed compartment syndrome, then how would you manage it?

CANDIDATE: Once compartment syndrome has been diagnosed clinically, emergency decompression is required. Theatre staff and anaesthetic on call team should be informed, informed consent must be obtained. I will take patient to theatre as soon as it is safe to do that. There is more than 1 technique described to decompress compartment syndrome of the foot, but I have been trained to decompress the nine compartments of the foot through three incisions, two dorsal over the second and third metatarsals and one on the medial side, just under the medial border of the first metatarsal. The patient will need to go back to theatre to have the wounds closed, once the soft-tissue swelling has gone down.

EXAMINER: What is the natural history of an untreated Lisfranc injury?

CANDIDATE: The long-term consequences of the untreated injury are the development of post-traumatic TMT arthritis. This may lead to the need for midfoot arthrodesis. Progressive deformity is also a problem with midfoot collapse, forefoot abduction and development of a rocker bottom foot.

EXAMINER: What is the role of primary arthrodesis?

CANDIDATE: It is controversial as to whether the Lisfranc joint complex should be fused primary or strictly reserved for a salvage procedure. In the past arthrodesis has been reserved as a salvage procedure after failed ORIF, for a delayed or missed diagnosis and for severely comminuted intra-articular fractures of the TMT joints.

However, in the more recent literature, a strong indication for primary arthrodesis of the TMT joints has been discussed often for purely ligamentous injuries. Such injuries are less likely to stabilise following fixation and may go on to develop degenerative changes.

Ly and Coetzee have described their selective indications for primary fusion of Lisfranc joints[3]. They perform primary fusion with:

- Major ligamentous disruption and multidirectional instability
- Comminuted intra-articular fractures at the base of the first and second metatarsal
- Crush injuries of the midfoot with an intra-articular fracture–dislocation

Contraindications are:

- Lisfranc injuries in children with open physis
- Subtle Lisfranc injuries with minimal or no displacement
- Unidirectional Lisfranc instability
- Unstable extra-articular fractures of the metatarsal bases with unknown amounts of ligamentous injury

Henning et al.[4] reported a random control trial in 31 patients comparing primary fusion with ORIF in a wider range of injuries including fracture–dislocations, although 'major intra-articular fracture patterns' were excluded. Fourth and fifth TMTJs were stabilised with wires. The trial was underpowered due to problems with recruitment. There was a trend towards better Short Musculoskeletal Functional Analysis scores in the fusion group but no difference in SF-36 scores, complications and pain or return to work or to wearing normal shoes.

EXAMINER: Any new methods of fixation?

CANDIDATE: Several recent papers have reported the use of suture button fixation in the hope of allowing some physiological motion and to avoid putting screws across the articular cartilage of the first cuneiform and the second metatarsal. Ahmed et al.[5] did a cadaveric study that showed more displacement with suture button fixation of isolated Lisfranc ligament injuries in cadaver specimens, which was in contradiction to the study by Panchbhavi et al.[6] that showed equivalence. A clinical trial would be useful to compare the two methods of treatment.

EXAMINER: Should the fusion be complete or partial?

CANDIDATE: The literature suggests partial fusion of the medial column has a better outcome than complete fusion. Mulier et al.[7] compared ORIF of severe Lisfranc injuries (16 patients) with partial (5) and complete (6) arhrodesis. At the 30-month follow-up period, patients who underwent fusion had more pain than the

ORIF and partial arthrodesis group. Complete fusion causes stiffness of the relatively mobile lateral column.

Primary partial arthrodesis should be considered in patients with either severely comminuted injuries or significantly displaced purely ligamentous injuries.

Classic reference

Ly, TV, Coetzee, JC. Treatment of primarily ligamentous Lisfranc joint injuries: Primary arthrodesis compared with open reduction and internal fixation. A prospective, randomised study. *J Bone Joint Surg Am*. 1996;88:514–20.

Department of Orthopaedic Surgery, University of Minnesota
Level 1 evidence
Prospective randomised control trial of 41 patients with an isolated acute or subacute purely ligamentous injury only

Twenty patients were treated with ORIF and 21 patients with primary arthrodesis of the medial 2 or 3 rays. At 2 years after surgery the American Orthopaedic Foot and Ankle Society midfoot score was 68.6 in the ORIF group and 88 points in the arthrodesis group.

Of the 20 patients in the ORIF group, 16 underwent a secondary surgery to remove prominent or painful hardware. Follow-up radiographs showed evidence of loss of correction, increasing deformity and degenerative joint disease in 15 patients.

The study concluded that primary stable arthrodesis of the Lisfranc joint complex had a better short-term and medium-term outcome than ORIF.

Classic reference

Kuo RS, Tejwani NC, Digiovanni CW, Holt SK, Benirschke SK, Hansen ST, Sangeorzan outcome after open reduction and internal fixation of Lisfranc joint injuries. *J Bone Joint Surg Am*. 2000;82A:1609–18.

Harborview Medical Center, Seattle, Washington
Level IV evidence

This paper was a retrospective review of 48 patients with both ligamentous and combined ligamentous and oosseous injuries that were followed for outcomes for an average of 52 months. Results showed that stable anatomical reduction of the fracture dislocation leads to the best long-term outcomes. Theses patients were shown to have less arthritis and better American Orthopaedic Foot and Ankle Society ankle–hindfoot scores.

Trauma oral 2

Lisfranc injury

EXAMINER: You have been called to see a 21-year-old male who has fallen 10 feet onto his right foot. He is complaining of pain, swelling and inability to weight bear in his right foot. This is an isolated injury; he has no other injuries of note[j].

How would you proceed?

CANDIDATE: I would order an x-ray.

COMMENT: Gaff 1. This is the wrong thing to start with in this particular scenario. At the very least the candidate should have mentioned examining the foot to exclude a fracture/dislocation and to check for any compartment syndrome or developing compartment syndrome

EXAMINER: Hold on is that the first thing you would do? Would you not want to take a history first and examine the patient?

CANDIDATE: (Silence)

EXAMINER: Here are the radiographs.

CANDIDATE: This shows an abnormal area around here. (Candidate pointing to x-ray.)

COMMENT: Gaff 2. The candidate was unable to describe the radiographs correctly.

The radiograph was an anteroposterior (AP) weight-bearing radiograph of the left foot. This would have been the first thing to have mentioned. The abnormality on the radiograph was identified but the Lisfranc injury couldn't be accurately described.

The candidate didn't mention obtaining a lateral non-weight-bearing radiograph. This radiograph may have shown dorsal displacement of the proximal base of the second metatarsal. The step-off point is where the dorsal surface of the second metatarsal is higher than the dorsal surface of the middle cuneiform.

EXAMINER: What is the injury?

CANDIDATE: (Long pause and then recovery) This is a Lisfranc injury of the foot.

EXAMINER: The patient was taken to theatre and the injury fixed. These radiographs are shown to you the next day on the ward round. What do you think about the fixation?

A radiograph of a poor k-wire fixation of this injury was shown without proper reduction of the Lisfranc dislocation and the candidate asked to comment. The candidate correctly identified the poor fixation and volunteered that it needed re-fixation.

A discussion then took place about exactly what was meant by the Lisfranc ligament and joint.

This was answered extremely well by the candidate.

A radiograph was shown of the re-fixed injury and the candidate asked to comment. A discussion of the current treatment recommendations in the literature then took place. In recent years there has been a move towards more aggressive fixation of this injury with plates rather than just using K-wires and screws.

Candidate debrief

The candidate never got past a 6 because of the 2 big gaffs at the beginning.

[j] Indirectly hinting to the candidate not to jump straight into a talk about ATLS® management.

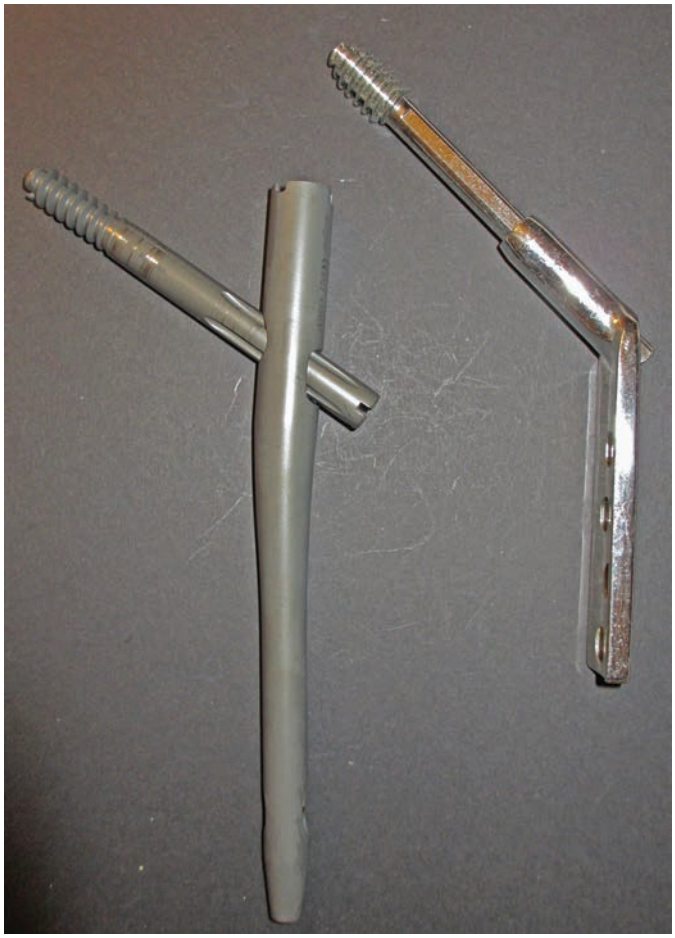

Figure 29.3 Photograph of DHS and IMHS. Lead in prop to discuss the biomechanics of both fixation devices

The candidate did, however, recover very well, identifying a poorly fixed fracture requiring re-do surgery and knowing the latest literature on fixation strategies.

Overall, a patchy performance with some very good answers later on, which saved the day for the candidate as the viva was passed, but the overall performance was a bare 6.

Without this poor start the candidate would have definitely scored a 7. The examiners gave the candidate an opportunity to recover

Trauma oral 1: Biomechanics of the intramedullary hip screw (IMHS) and dynamic hip screw (DHS)

This may be asked in the trauma viva, less likely a basic science question.

A dynamic compression hip screw uses the principle of dynamic compression that modifies functional physiologic forces into compression at the fracture site. The implant consists of two major parts: A wide-diameter cannulated lag screw which is inserted into the femoral head and a side plate with a barrel at a set angle which is attached to the femoral shaft (Figure 29.3). Weight-bearing and abductor muscle activity cause the screw

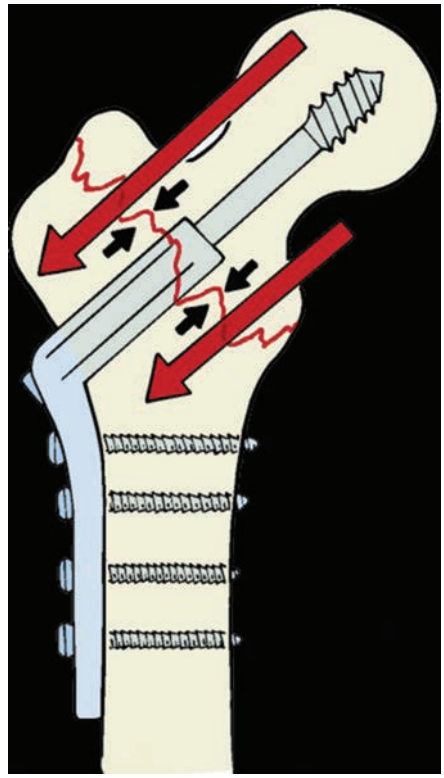

Figure 29.4 Dynamic compression-sliding hip screw. Functional loading of a sliding hip screw causes dynamic compression at the fracture site. With functional loading, the screw slides through the barrel of the side plate, allowing the fracture to impact or compress

shaft to slide through the barrel, resulting in impaction of the fracture surfaces and a stable load-sharing construct.

There are two important biomechanical principles that must be respected when using a hip screw:

1. Fracture compression can occur only if the lag screw and barrel are inserted across the fracture site. This occurs with intertrochanteric fractures but when used to fix a high subtrochanteric fracture, the lag screw and barrel are located exclusively in the proximal fragment and do not cross the fracture site. In these circumstances, the lag screw acts only as a fixation device and does not contribute to fracture compression by sliding
2. The lag screw must slide far enough through the barrel to allow the fracture gap to close sufficiently for the proximal and distal fragments to impact completely

The dynamic compression hip screw has several modes of failure, the most common being varus cut out of the lag screw from the femoral head, collapse resulting from excessive medial displacement of the femoral shaft and bone erosion in the femoral head.

Biomechanically the IMHS offers four main advantages over DHS fixation: (1) Load-sharing device rather than load bearing (DHS); (2) decreased implant bending strain because the shaft fixation is moved from the lateral cortex to the intramedullary canal, decreasing the lever arm on the implant; (3) the device acts as a robust intramedullary buttress, limiting excessive shaft medialisation in unstable and reverse oblique fractures; (4) it can allow impaction/collapse along the axis of the shaft (Figure 29.4).

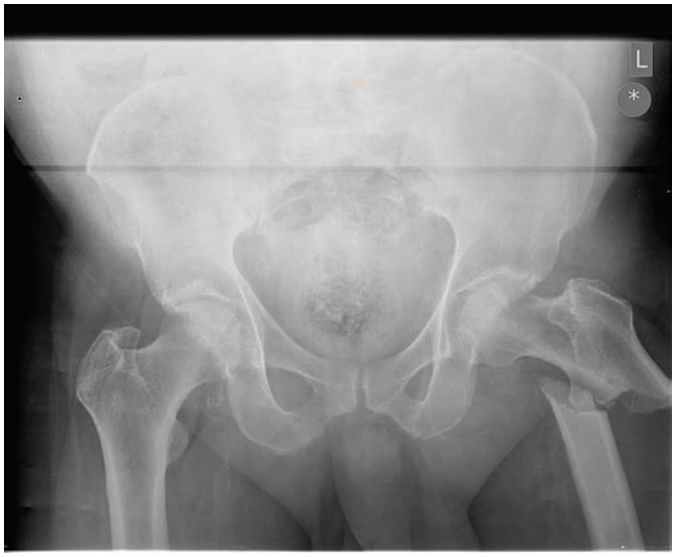

Figure 29.5 AP pelvic radiograph of subtrochanteric fracture

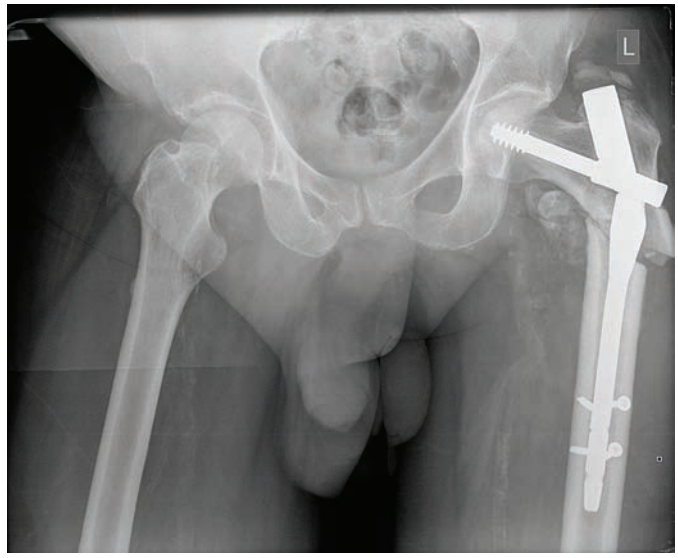

Figure 29.6 AP pelvic radiograph of broken IMHS

Randomised prospective studies for routine intertrochanteric fractures with an intact lateral buttress show no difference in operating time, complication rate, blood loss, hospital stay, time to ambulation or patient satisfaction.

However, several randomised prospective studies have reported improved results using IMHS with unstable fracture patterns including reverse obliquity intertrochanteric fractures, fractures with posterior and medial comminution, and fractures with extension into the femoral neck or subtrochanteric region.

Trauma oral 2

EXAMINER: This is a AP pelvic radiograph of a 83-year-old female who sustained a subtrochanteric fracture of her left femur (Figure 29.5) managed with IMHS which has failed (Figure 29.6). What are your thoughts?

CANDIDATE: Do we have an immediate postoperative radiograph?

EXAMINER: No. These are the only radiographs we have.

CANDIDATE: The IMHS has failed. It has fractured by metal fatigue cracking. The fatigue cracking has propagated to an extent that the remaining cross-sectional area of the nail could not bear the imposed patient loading, leading to overload fracture. Fatigue cracking is caused by the nail bearing cyclic (i.e. repeated) stresses in excess of the material endurance limit for an extended period of time. These excessive cyclic stresses may be caused by any number of conditions, including but not limited to excessive patient activity levels prior to full bone union, poor bone quality, excessive patient weight and chronic non-union or mal-union of the bone fracture.

EXAMINER: What do you think has happened here?

CANDIDATE: It is difficult to say entirely but it looks as though the fracture wasn't fully reduced before the nail was inserted and this can predispose to non-union and nail breakage.

EXAMINER: How do you prevent nail failure occurring?

CANDIDATE: A more rigid, larger implant that minimizes motion at the subtrochanteric region and minimizes stress risers may lesson the risk of implant failure and aid subtrochanteric fracture-healing. If the patient is on biphosphonates this should be stopped until the fracture has healed.

COMMENT: The candidate could have mentioned smoking, alcohol and poor nutrition as possible causative factors for the non-union developing. The easiest option would be to go through the possible local, general and fixation factors which influence fracture union.

EXAMINER: What causes the fatigue fracture?

CANDIDATE: Fatigue fracture of the nail starts at the aperture of the lag screw. It is the location of the highest von Mises stress, which is the failure criterion for ductile materials. A possible reason could be eccentric insertion of the lag screw causing a localized defect in the material which acts as a stress riser.

COMMENT: The examiner is steering the topic back to applied biomechanics rather than clinical.

EXAMINER: How would you manage this fracture now?

CANDIDATE: Re-nailing with a long IMHS and bone grafting to the non-union site is an option, but there could be a high chance of failure and I would prefer to avoid this option. This method has failed once and may easily fail again. (Figure 27.7a and b.) I would prefer to use compression plating with use of a fixed-angle device along with protected weight-bearing postoperatively.

Further lower limb topics candidates should explore for the viva would include:

Periprosthetic fracture: Hip/knee

- Unified Classification System[8] – The next stages of Vancouver classification

Intracapsular hip fracture – Young and old – THA debate – NICE Guidelines. Extracapsular hip fracture : A2 fractures – DHS vs nail, etc. Bisphosphonate fractures

(a)

(b)

Figure 29.7 (a, b) Photographs of retrieved broken IMHS

Femoral nailings set up and complications – Medial wall blow out with incorrect entry point. Freehand vs traction table

Combined intra-capsular hip fracture and associated diaphyseal fracture

Rotational profile assessment of long bone fractures

Management of delayed union – Exchange nailing/rule out infection/grafting

Femoral head fracture/native hip dislocation management

P&A – Pelvic fracture – Building on above – Resuscitation and evaluation/classification/haemodynamic and mechanical instability. Approaches, e.g. posterior approach, sciatic nerve assessment

Talk me through surgical hip dislocation, for Pipkin management!!

Floating knee/knee dislocation: Revascularization/reperfusion/stabilisation/fasciotomy. Pin positions – To allow for plating with X-fix still in situ

Pilon fracture: Staged management – Span scan plan, etc. Approaches. Fixation options

Syndesmosis injuries and late presentation

Talus fracture : Recognition/surgical anatomy/phased management/complications and follow up.

Compartment syndrome of foot

Tendon injuries – Ligamentum patella, quadriceps, acute injuries and delayed presentations

Adult upper limb

Anterior shoulder instability

A common trauma scenario, dislocation of the glenohumeral joint may function as a case from which examiners may choose to explore assessment, management, applied anatomy and rehabilitation domains of trauma care.

The humeral head, spherical in nature, is retroverted around 30° from the transepicondylar axis of the distal humerus with the articular surface inclined 130° from the shaft. This angulation is additive to the 30° that the scapula sits anterior to the coronal plane on the chest wall. These features contribute to the large freedom of movement around the joint and also in its stability.

When discussing anterior shoulder instability, a simple way of answering questions is to breakdown the restraints to dislocation into static and dynamic elements.

Static restraints of the glenohumeral joint:

1. Joint capsule
2. Negative intra-articular pressure
3. Articular congruity and joint version
4. Glenoid labrum

5. The glenohumeral ligaments

Of these structures, the glenoid labrum and the glenohumeral ligaments are key to static control of the joint. In particular the capsuloligamentous condensation of the anterior band of the inferior glenohumeral ligament (IGHL) affords static control of the joint in its most exposed position, that of maximal external rotation with 90° abduction. Rupture of this ligament along with the anterior labrum where it anchors produces the pathognomic Bankart lesion.

Dynamic restraints of the glenohumeral joint:

1. Rotator cuff muscles
2. Biceps brachii
3. Deltoid
4. The periscapular muscles

Providing dynamic control of humeral head dislocation, the rotator cuff musculature centres and compresses the humeral head against the glenoid. Both the rotator cuff and the periscapular muscles which position the scapula and orientate the glenohumeral joint in addition to affording restraint, are crucial in the rehabilitation from shoulder injury and must feature in any answer dealing with the rehabilitation of a patient with a dislocated shoulder.

Classification of shoulder dislocation

It is worth remembering that classification of an injury should guide its management. Classification systems have been applied to shoulder instability but they do not fully address the large spectrum of conditions that can masquerade as anterior instability.

The key to classifying or ordering ones thoughts when assessing instability is to fully appreciate a few significant factors: Patient age, index event or recurrent, frequency if recurrent, the degree of instability (dislocation vs subjective subluxation), associated trauma, the direction and volition. In particular, age is important for two reasons. Firstly, recurrence rate of dislocation following index traumatic injury is greatest in the teenager and young adult. Secondly, and often under appreciated, there is a significant correlation between increasing age and associated rotator cuff tear with index traumatic dislocation.

These features are important in assessing instability and will inform management. In essence, unilateral, unidirectional traumatic dislocations, particularly in the young often require surgery. By contrast, atraumatic, multidirectional, bilateral and/or volitional instability requires rotator cuff and periscapular musculature rehabilitation. This concept is delivered in the Stanmore classification as:

Traumatic structural (the young sportsman with an IGHL lesion and Bankart lesion), atraumatic structural (due to abnormal static restraint) and habitual non-structural (muscle patterning abnormality).

Management of glenohumeral joint dislocation

Following initial global assessment of the limb, including axillary nerve sensation, adequate radiographs should be obtained. Whilst in reality these are often done before orthopaedic referral is sought, it should be emphasized that a dedicated AP view of the glenohumeral joint and an axillary lateral are the minimum acceptable radiographs to allow evaluation of the joint. Single image radiographs, which include the hemithorax and neck with the glenohumeral joint on the periphery often partly obscured, are not acceptable as posterior dislocations may be missed.

There are many methods available to reduce a dislocated shoulder and can be accessed elsewhere. What is of most importance is that with any fracture dislocation of the humerus, a low threshold for controlled reduction under imaging and general anaesthesia is advised to prevent propagation of fracture and conversion to a surgical neck extension. This last point is of greatest worth and should be emphasized in the exam.

With regards immobilization, there is no evidence for immobilization further than 1 week from injury and so a simple sling and 1-week fracture clinic or subspecialist shoulder assessment clinic is satisfactory. Bracing in external rotation has been demonstrated in some series to reduce recurrence but these results are not reproducible and compliance is a real issue.

The main indicators of recurrence are: Young age, high-energy injury, glenoid bone loss, an engaging Hill–Sachs lesion and non-compliance with rehabilitation.

The 'ideal' patient for operative management is the young (<25 years), high demand primary dislocator with an acute traumatic injury with no associated bone loss, such as a soldier injured playing rugby. For these individuals, following a period of 'pre'rehabilitation, arthroscopic stabilisation with biodegradable anchors has been shown to have a similar recurrence and re-operation rate to open anterior capsulolabral reconstruction but with less pain and greater range of movement.

Success of surgery diminishes in the presence of generalized laxity (as assessed by the Beighton score[9]), multidirectional instability and abnormal glenoid morphology through bone loss.

In patients with glenoid bone loss, a large engaging Hill–Sachs lesion or humeral avulsion of the glenohumeral ligament open reconstruction and augmentation where appropriate with a Laterjet type procedure is recommended.

Fractures of the clavicle

As with the humeral shaft, the management of the vast majority of clavicle factures is no different in the viva table than it is in a busy fracture clinic setting. Non-operative management is the baseline with variance from this acceptable within certain circumstances although indications for operative management remain surgeon specific and open to some debate. These variations which attract debate affect the minority of cases but can derail the examination candidate. An appreciation of the evidence is key here.

Again as with the humerus, disruption of anatomical balance seen subsequent to clavicle fracture is a good basis on which to build an argument for the management of clavicular injury. The sternocleidomastoid causes elevation of the medial fragment upon fracture, in contrast to the relative depression of the lateral fragment consequent to the static control of the coracoclavicular and coracoacomial ligaments and the effect of the weight of the arm.

The clavicle is unique in its osteology. It is the first bone to ossify and its medial physis is the last in the body to fuse, often as late as the third decade. Its morphology is also characteristic and contributes to injury patterns and fixation strategy. It has a double curve in the coronal plane with its medial two thirds convex and its lateral third concave. The junction between these curves is a weak point and is commonly where fracture occurs.

Fractures of the clavicle are often classified according to the location of the fracture within this coronal plane as involving the medial third, the middle third or the lateral third. Distribution of fracture is roughly 80% involving the middle third, 19% the lateral third and only 1% of fractures are seen in the medial third of the bone. Although the majority of fractures involve the middle third, these afford less complication to surgeon or patient. Due to the energy required to fracture the medial clavicle with its strong ligamentous attachment to the sternum, it has a higher association with underlying injury and is less benign than fractures of the middle third. Similarly, fractures of the lateral third may be problematic, not with great vessel injury or organ damage as seen medially but in non-union. The non-union rate of lateral clavicle fractures is around 10% but it is important to note that only a small number of these patients come to require intervention.

Allman[10] classified clavicle fractures according to location with group I representing middle third fractures, group II lateral third fractures and group III representing fractures of the medial third. Subsequently, due to the range of fracture patterns seen and the impact of exact fracture location on outcome; the Group II or lateral fractures have been further subdivided by Craig [11]:

Group II type:

1. Minimal displacement – An interligamentous fracture, not involving the acromioclavicular joint, occurring between the coracoclavicular and acromioclavicular ligaments
2. Injury medial to the coracoclavicular ligaments. Further subdivided as group II type 2 (a) conoid and trapezoid attached and group II type 2 (b) conoid torn trapezoid intact
3. Involvement of the AC joint articulation
4. Periosteal (paediatric) fractures
5. Comminuted fractures with neither ligament attached

A grasp of the relevant anatomy and its relationship to injury and widely used classification systems is a good baseline from which to develop your answer to clavicle fracture viva questions.

Having covered the basics, the question of management of clavicle fracture may arise and with particular reference to recent literature regarding middle third (Group I) fractures. Much debate arose on publication of the Canadian Orthopaedic Trauma Society multicentre, randomised trial of non-operative treatment compared with plate fixation of displaced midshaft clavicular fractures[12]. One hundred and thirty two patients with a displaced midshaft fracture of the clavicle were randomised and significant improvements in time to union, mal-union and function were reported in the operative group.

There is no question that operative intervention reduces non-union rate and this has been shown in other studies. What is in question is true improvement in overall function and this is at the cost of complications such as hardware removal and infection. Increasingly important, the cost of routine fixation of middle third clavicle fractures is considerable, and you need to be able to justify your management option.

More recently and perhaps to strengthen the argument for initial non-operative management, evidence has been shown to recommend delayed intervention when required for midshaft fractures. Potter et al.[13] report similar outcomes in patients undergoing operative intervention acutely and in those having a delayed fixation for symptomatic non-union.

It can be seen, therefore, that there are gains to be made from fixation although it would seem that accepting that the vast majority of midshaft fractures heal with little concern, a delayed intervention strategy may be a sound approach. With regard to the lateral clavicle, it is accepted that a higher non-union rates area is associated with these injuries and operative management for type 2 and type 5 fractures may be considered. Operative intervention, however, is associated with a risk of hardware failure and this must be balanced in the decision making.

Humeral diaphyseal fracture

There are three main aspects to questions involving the non-articular element of the humerus. Firstly, fracture management with regard to tolerances of non-operative intervention and in the context of special circumstances, such as multiple injuries, open fractures, pathological fractures, etc. Secondly, the applied anatomy of the humerus with regard to its behaviour once fractured. Lastly, and most likely to be enquired about with any case involving humeral shaft injury – The impact of radial nerve palsy either associated with the primary trauma or following manipulation or fixation.

A clear and reasoned approach to all elements of this fracture will result in a satisfactory and comfortable viva performance.

Overall tolerances of non-operative management may be simplified as <3 cm shortening or $<30°$ angulation in either the coronal or the sagittal plane. Mal-union of up to 30° is accepted due to the ability of the shoulder to compensate in placing the hand in space.

With regard to applied anatomy, part of the issue with regard to humeral fracture management is the deforming forces particularly around the proximal humeral diaphysis. With a fracture line that runs above the insertion of both pectoralis major and the deltoid, the proximal fragment is pulled into abduction and external rotation by the rotator cuff and the distal segment is pulled medially and anteriorly by the insertion of pectoralis major which is attached to the diaphyseal portion.

Slightly more distal, should the fracture line extend distal to the insertion of pectoralis major but remaining proximal to that of the deltoid tendon, the distal segment is abducted by the unopposed deltoid and the proximal fragment is pulled medially by the combined vector of the triple insertion of pectoralis major, latissimus dorsi and teres major.

With a distal diaphyseal fracture, the fracture line is distal to all the major muscle group insertions. The proximal fragment, therefore, is pulled into abduction, flexion and external rotation, mimicking the situation seen at the proximal femur.

When answering viva questions, as with the reality of fracture clinic, the fractured diaphyseal humerus is managed non-operatively in the majority of cases. Good results can be obtained within the described tolerances with splinting and functional bracing. There is good evidence for functional bracing following a two-week period of splinting with the vast majority of these fractures healing with a good functional result without an operation[14].

There are relative indications for operative intervention out with the accepted tolerances of simple fracture patterns as described. These are segmental fractures, the humeral fracture in the setting of multiple trauma, open fracture, dysvascular limb and loss of alignment following non-operative management.

It can be seen, therefore, that for the majority of humeral shaft fractures, non-operative management is appropriate. For the small numbers that require an operation the two main options are intramedullary nailing and plate fixation.

The evidence surrounding the use of either implant is poor but meta-analyses suggest that the overall outcomes in terms of fracture union, radial nerve injury and infection are similar. What is known is that intramedullary nailing is associated with an increased risk of shoulder dysfunction, implant failure and requirement for further surgery. Pragmatically, nailing is considered for pathological fractures and segmental injuries with plating chosen predominantly for the remainder.

The humeral shaft fracture with radial nerve compromise

It is the question of the radial nerve that opens the humeral diaphyseal fracture to further exploration at the viva. Examiners are looking for a safe and sensible approach and adding in a radial nerve injury to a closed diaphyseal fracture opens up potential pitfalls for the unwary candidate. A reasoned approach, supported by limited, basic evidence will, however, secure a solid viva performance, converting this from a potential problem question to a gift exam scenario.

Traditionally the Holstein–Lewis fracture[15]; a distal third extrarticular humeral fracture with proximal and medial displacement of the distal fragment; was associated with an increased incidence of radial nerve injury. As with a number of 'classic' orthopaedic references, this paper has little scientific rigour, being based on seven cases, four of which were lost to follow up. Whilst some still propose an increased incidence of radial nerve injury with this pattern, the bulk of literature suggests that it is with the middle and middle/distal third fractures together that put the nerve at greatest risk. Anatomically this reflects the close apposition to the bone in the spiral groove and the tethering it may undergo due to fracture angulation when it pierces the lateral intramuscular septum. In addition, the nerve is at greater risk with a transverse or spiral fracture than a comminuted one.

Whilst this pattern recognition is of value, it is not the main issue with this question. Candidates must have an answer to the question of how they would manage the patient with radial nerve palsy both at initial presentation with a fracture and that manifesting after manipulation or surgical intervention.

For the closed humeral shaft fracture with a radial nerve palsy identified at presentation, the literature is supportive of non-operative management as the majority of cases are of contusion and tractional injury. There has been raised the concept of early ultrasound as an adjunct to nerve assessment and this may have prognostic power in future care of these cases but the 'exam answer' is non-operative management as complete recovery occurs in over 70% of cases.

The next issue that may be raised is how to proceed if no radial nerve deficit is recorded on presentation but one occurs following manipulation, such as when applying a splint. Whilst opinion is divided, having documented that a nerve is functioning normally then finding it is not following a manipulation, the safe examination answer is to recommend nerve exploration. Having established that the bulk of injury is by contusion, this occurs at the time of injury. If contusion sufficient to cause palsy is not manifest at admission then when a palsy occurs following manipulation, it must be presumed that the nerve is no longer in continuity. Personally we would explore via a posterior approach, with the patient in the lateral or prone position with fixation, using a standard large fragment plate. Highlight the importance of documenting the position of the nerve in relation to the screw holes in the plate, should revision surgery ever be necessary.

The last scenario is that of the nerve palsy following fixation. The approach to this is straightforward. If a nerve stops working after a surgeon has been near it with an instrument or drill, then it must be assumed cut until proven otherwise unless integrity of the nerve was confirmed prior to closing the wound. Postoperative neuropathic pain similarly is an absolute indication for exploration. Delay in these situations is unacceptable.

By applying these principles, candidates should be able to navigate through the management of humeral shaft injury with ease.

Adult forearm fractures – Simple and complex

A radiograph of a deformed forearm may be used as a position from where to examine the candidate's grasp of anatomy, fracture healing, operative fracture management, compartment syndrome and multiple trauma management. Be aware and listen to what the examiner is asking. It is impossible to detail sensible responses to all of the possibilities here and so aspects relating primarily to the assessment and management of forearm fractures and complex injuries will be concentrated upon.

Applied anatomy of the forearm

A highly complex entity, packed with muscles, tendons, ligaments and neurovascular structures, the compartments of the forearm their contents and implications for injury and surgical approach must be immediately available to the candidate and is assumed knowledge. You may be shown a cross-sectional diagram for the forearm/elbow/upper arm and asked to identify structures.

The ulna acts as an axis around which the laterally bowed radius rotates in supination and pronation. This concept is key to understanding the requirement for fracture fixation. The 'radial bow' is fundamental for movement and function.

Management of 'simple' forearm fractures

An 'intra-articular' injury, all radial fractures, even minimally displaced should be considered for fixation to allow optimised forearm function and early rehabilitation.

The relationship between the radial and ulnar shafts and, therefore, the ability for the bones to achieve a satisfactory pronosupination arc is determined by the interosseous membrane, the tension of which may be altered by poor fracture fixation. Anatomical reduction and absolute stability of the radius is required although care must be taken to restore the radial bow as well. A huge torsional stress goes through the radius and, hence, short plates are discouraged and a minimal 3.5 mm small fragment LCDCP is required.

Ulnar fractures are treated subtly differently. Non-operative management may be considered in the distal third segment, if >50% cortical contact is present and <10° angulation seen. Proximal ulna fractures are generally treated operatively. Initial management in a cast/splint is acceptable for distal fractures although prolonged time to union and increased risk of non-union is encountered. For the young fit patient in order to minimize time off work and limit non-union, an operative approach is acceptable for these fractures although there is no robust evidence to support this.

Management of 'complex' forearm fractures

Monteggia fracture dislocations (Proximal ulna fracture with radial head dislocation)

This is a combination of a proximal ulna fracture and a radial head dislocation and it is the nature of the latter that dictates its subtype in classification.

As with any forearm injury, prompt assessment of circulation and documentation of the integrity of the anterior interosseous, posterior interosseous and ulnar nerves is mandatory followed by splintage and re-assessment. Radiographs in two planes of the forearm and focused two plane views of the elbow and wrist are required.

Classification – A favourite with examiners and trauma meeting discussion alike, the Bado classification is required knowledge:

I. Anterior dislocation of the radial head
II. Posterior dislocation of the radial head
III. Lateral dislocation of the radial head
IV. Type I, i.e. anterior, with an additional radial fracture

Type I fractures are the most common and represent a fall on an outstretched hand with the elbow failing in hyperextension and the radial head being pulled out by the biceps tendon.

Management

Operative management is mandatory in these fractures and it is important to note at the viva that screening the elbow at the beginning of the case under image intensification is mandatory to exclude other injuries such as radial head fracture and capitellar injury. If any doubt occurs preoperatively, a low threshold for early CT scanning is sensible to inform operative planning.

Open, anatomical reduction of the proximal ulna will often suffice as the radial head usually reduces following fixation of the ulna. If this is the case, stability of the elbow should be assessed by intraoperative on table screening. In comminuted fractures of the ulna, care should be taken to attempt to regain ulna length as a minimum to aid proximal radioulnar joint congruency. Should ulnar shaft reduction and fixation be satisfactory but the radial head still will not reduce, interposition of the annular ligament must be ruled out and this mandates an open exploration of the ligament laterally. One of the complications associated with this injury is a deficit of the posterior interosseous nerve. Hence, as per all limb injuries, precise documentation, serial evaluation and postoperative assessment of the named nerves of the limb is essential.

Galleazi fractures (radial shaft – most commonly distal third-fracture with disruption of the distal radioulnar joint)

With the focus on the broken radius, disruption of the distal radioulnar joint (DRUJ) may be missed and so it is important with all radial shaft injuries to both clinically and radiographically assess the joint above and below the fracture. Two plane radiographs of the forearm and focused views of the wrist and

elbow are again mandatory. The closer to the wrist the radial shaft fracture, the greater the risk of DRUJ disruption.

Management

The Galeazzi fracture is one of operative necessity – Unless the patient has significant co-morbidity, operative management should be chosen due to the considerable morbidity associated with DRUJ dysfunction.

The radial shaft fracture should be approached and reduced through a standard volar approach and the DRUJ screened once the radius is fixed. If the DRUJ is stable, it should be left and an above elbow cast in supination is applied. The patient should have a check radiograph in 1 week in the clinic. If the DRUJ is unstable but reducible, the joint should be stabilised with 2 percutaneous 1.6 mm wires with the forearm in neutral and rested in an above elbow cast for 4 weeks. The wires can be passed through and through the distal radius and ulna, so if the wires did break they can be removed from either side, leaving no metal work in situ. If the DRUJ is irreducible once the radius is fixed, then interposition of the extensor carpi ulnaris should be suspected and this warrants open assessment.

Complications of forearm fractures

Whilst a wide range of possible complications may occur following forearm fractures, there are a number that must be focused upon. These include:

Compartment syndrome

Discussed in greater detail elsewhere, compartment syndrome is essentially an increased pressure in an enclosed osseofascial compartment leading to ischaemia through diminished capillary inflow. With regard the forearm, there are three compartments, the mobile wad, the dorsal and the volar with the latter split into superficial volar and deep volar. The deep volar is most susceptible as it is compressed against rigid bone and the interosseous membrane. When discussing management, decompress the superficial volar first, then the deep volar and ensure that the lacertus fibrosis proximally and the carpal tunnel distally are included in the decompression. Approaching the hand, it is important to not extend the incision any more radial than the radial border of the ring finger in order to avoid injury to the superficial branch of the median nerve. The dorsal musculature is decompressed through a separate incision between the two compartments allowing both to be decompressed through one incision.

Posterior interosseous nerve (PIN) palsy

This leads to total loss of extension of the fingers and the thumb. Note, wrist drop does **not** occur although radial deviation with the wrist in extension may be seen as the long extensors of the wrist are innervated by the motor branch before PIN arises in supinator. Note that a PIN palsy may be distinguished from a pure extensor tendon injury due to maintenance in the former of the tenodesis effect of digit extension on passive wrist flexion.

Anterior interosseous nerve (AIN) palsy

This injury to the branch of the median nerve serving the long flexors of the index and middle finger, the long flexor of the thumb and the pronator quadratus may occur in forearm fracture either at injury or operation. It is distinguished by a median nerve proper lesion due to the maintenance in the AIN lesion of sensation and function of the thenar musculature. Classically, the AIN palsy manifests as an inability to perform the 'okay' sign due to an inability to flex either the thumb or the index finger at the interphalangeal joint (distal).

Infected non-union and loss of fracture reduction

Can you formulate a plan in order to find an infective organism, treat appropriately, and obtain bony stability while the fracture heals?

Refracture after plate removal

More applicable to the paediatric population although may still be seen in adults should plate removal be required. Refracture is associated with early (<1 year) plate removal, presence of comminution at original injury, mismatch between bone and plate stiffness, i.e. using a 4.5 mm plate, will dispose towards refracture on removal due to stress shielding effect. Finally, lack of compliance or repeat trauma is also associated with refracture.

It can be seen that forearm fractures are a straightforward exam topic if sound knowledge of key anatomy and surgical principles are understood and demonstrated efficiently. Remember that if a forearm radiograph is put up in front of you at the viva, any one of the above issues may be discussed so stay flexible and be thinking of the associated complications as well as the proposed management.

Examination corner

Distal radial fracture viva 1

EXAMINER: Talk me through how you would manage this from attendance in the ED onwards

CANDIDATE: History, exam, analgesia, haematoma block – Describe, check x-rays describe x-rays – ?acceptable – Discuss the parameters you are using.

EXAMINER: Describe factors that correlate to loss of reduction[16]?

CANDIDATE:

- Initial dorsal angulation>20°
- Dorsal comminution
- Radio carpal intra-articular involvement
- Associated ulnar fracture
- Age >60

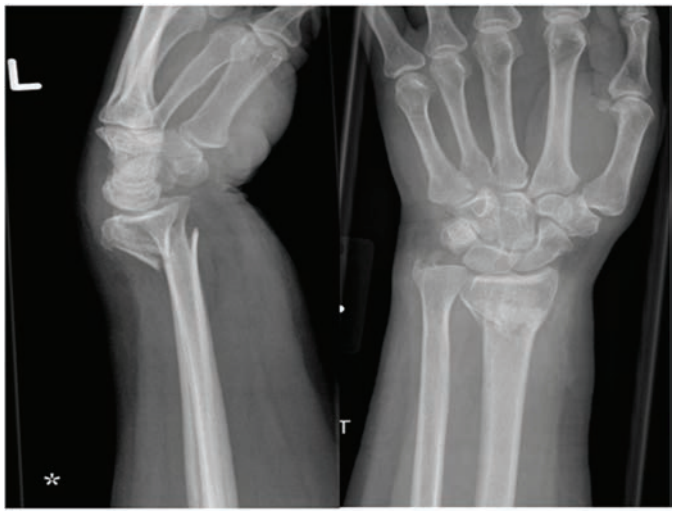

Figure 29.8 AP and lateral radiograph of distal radial fracture

If ≥3 of the above and cast treatment this results in loss of reduction.

The risk of loss of reduction increases with advancing patient age[17] (>58 years → 50% risk of slippage, >80 years → 77% risk of slippage).

EXAMINER: What next? What would you do and why? Explain the findings from DRAFFT study?

COMMENT: Please review the paper but in summary 'Contrary to the existing literature, and against the rapidly increasing use of locking plate fixation, this trial found no difference in functional outcome in patients with dorsally displaced fractures of the distal radius treated with Kirschner wires or volar locking plates. Kirschner wire fixation, however, is cheaper and quicker to perform.'

EXAMINER: Has this altered the way you manage wrist fractures?

EXAMINER: The patient presents with or develops carpal tunnel syndrome. What now? Discuss management, single incision approach and release[18]? Or separate fixation and release?

What are the benefits if any of polyaxial locking screws?

Outline your postoperative management plan.

Early mobilization/late mobilization? Benefits if any?

Describe complex regional pain syndrome – Diagnosis and management.

When would you consider dorsal plating and describe you surgical method

Explain the benefits of volar plating over dorsal plating?

- More space available – Pronator quad. Interposed between bone and tendons
- Volar cortex typically less comminuted – Reduction easier
- Volar scars better tolerated
- Less disruption of blood supply to radius

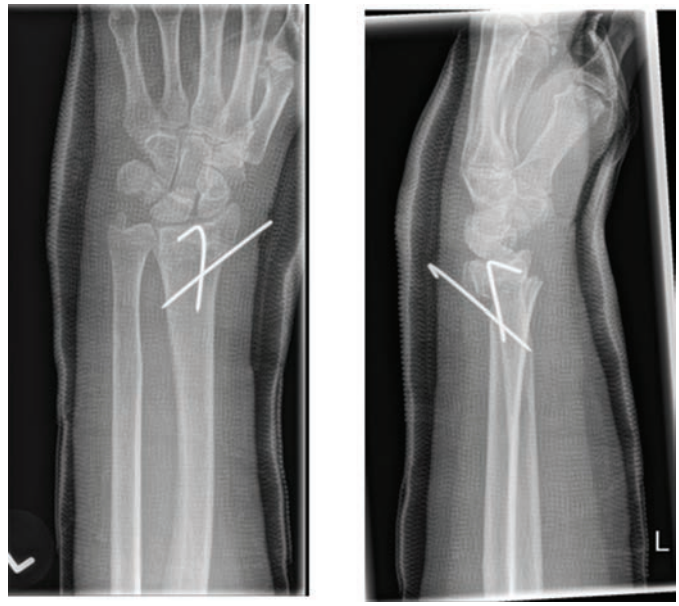

Figure 29.9 AP and lateral radiograph of distal radial fracture managed with closed reduction and K-wire fixation

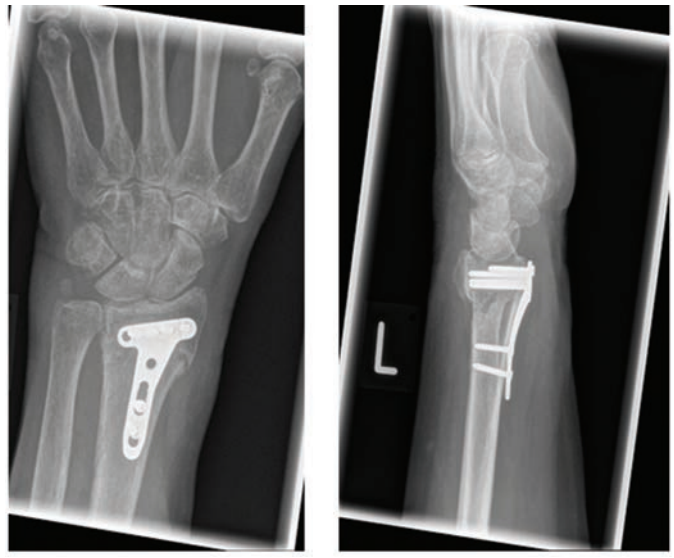

Figure 29.10 Removal of wires and volar plating performed

You review a patient (Figure 29.9) in fracture clinic 2 weeks after closed manipulation and K-wiring.

- Discuss the current position. Why has this happened?
- Outline the problems you may now encounter.
- What next? What are you going to tell the patient? (Figure 29.10)

Distal radial fracture viva 2

These are the radiographs of a 21-year-old male who has come off his motorbike at high speed sustaining this isolated closed injury. He is right hand dominant

COMMENT: Even though the history may suggest other potential injuries the clue that the ATLS® default statement is not needed is 'isolated'.

CANDIDATE: These are AP and lateral radiographs which demonstrate an intra-articular displaced highly communited fracture of the right distal radius. There is radial shortening and involvement of the distal radioulnar joint

EXAMINER: How would you classify this fracture?

CANDIDATE: Various classification systems are used including the AO, Frykman, Melone and Fernandez classifications. I would use the classification described by Frykman, which defines the fracture according to pattern of intra-articular involement and is useful for planning surgical strategy.

COMMENT: Candidates may not always get asked to classify a wrist fracture but should still should be able to discuss the various classification systems used and the one 'you yourself' prefer and why.

EXAMINER: How would you manage this fracture?

CANDIDATE: I would manage this fracture with open reduction internal fixation using a fixed angle locking plate to achieve anatomical articular reduction and stable fixation of the fragments

COMMENT: A candidate may then be asked to describe the volar distal Henry's approach to the wrist or the evidence for using a volar locking plate.

There is no Level I clinical evidence suggesting a superior mode for treatment of distal radial fractures. I would choose open reduction and internal fixation since this will allow accurate fracture reduction and restoration of radial length and inclination. I would be concerned with using k-wire fixation with this degree of fracture comminution if we could achieve fracture reduction closed, if we could maintain fracture reduction without redisplacement as it is a highly unstable injury. The patient is young and it is his dominant hand, so I think volar locking plate is a safer option.

Candidates may be asked about factors which may determine a poor outcome for a wrist fracture. Know the literature on this[k].

Several studies suggest that inaccurate reduction and articular incongruency of >2 mm, failure to restore radial length to within 5 mm and articular comminution correlate with a poor outcome.

Classic reference

Knirk JL, Jupiter JB. Intra-articular fractures of the distal end of the radius in young adults. *J Bone Joint Surg Am.* 1986;68:647–59.

In a retrospective review of 40 young adults at a mean follow-up of 6.7 years, Knirk and Jupiter showed that a step-off ≥ 2 mm was associated with 100% incidence of radiological arthritis of which 93% (26 of 28) were symptomatic.

Intra-articular fractures (AO types B and C) with a step of ≥ 2 mm in the radiocarpal joint inevitably result in osteoarthritis and functional impairment. Knirt and Jupiters article is one of the most important studies on the management of intra-articular fractures of the distal radius. Prior to its publication, the critical factors that determined successful long-term management of intra-articular distal radius fractures in young patients had not been determined. Fractures of the distal radius at the time were thought to be relatively benign injuries and were often all treated with the same methods and post-traumatic arthritis and associated disability not well appreciated.

Knirt and Jupiter's conclusions challenged the contemporary understanding of the injury. They found that the most important factor determining a successful outcome from this type of injury and preventing long-term arthritis was accurate articular restoration.

Haus and Jupiter[19] revisited the article in 2009 reviewing the methodological flaws contained in the original study. This paper is an excellent illustration of how to critically review a paper. It is a good educational article for a journal club to use.

Trauma oral 3

The viva can surprise some candidates in starting off with the complication of a distal radius fracture. These include median nerve dysfunction, non-union or mal-union, post-traumatic osteoarthritis, complex regional pain syndrome and tendon rupture. The viva would then concentrate in more detail on one or two of these in particular EPL rupture and its management or CRPS.

Other upper limb areas to review include the following:

- Proximal humeral fractures
- PROFHER study, etc

A multicentre RCT that aims to obtain evidence of the effectiveness and cost-effectiveness of surgical vs non-surgical treatment for the majority of displaced proximal humerus fractures in adults

There is great variation in the treatment of these fractures, both in basic (the use of surgery) and specific (type of implants and surgical technique; non-surgical management and rehabilitation packages) terms. There is a lack of evidence from RCTs to inform management decisions for proximal humerus fractures

The difficulty for candidates sitting the exam is that you can not default to the view that we don't know what to do with these fractures and are awaiting results form ongoing RCTs. Candidates need to be aware of the factors that will influence when best to operate and when to treat conservative

The default statement is 'Although I are not aware of any Level-I randomised controlled trials or high quality prospective studies that have evaluated any of the treatment options for proximal humeral fractures there is evidence from prospective comparative studies, case-control studies, and retrospective comparative studies. This data represent

[k] The Jupiter paper is a good paper to pull out of your pocket as most examiners are vaguely familiar with this.

fair evidence for or against recommending an intervention and I would consider papers presented in the peer-reviewed journals'[20]

- Reverse polarity replacement for trauma
- Multilock nail/locked plates for proximal humeral fractures
- Approaches/setup for images

Examination corner

Three part valgus impacted humeral fracture

Plain radiographs shown to candidate and correct identification of fracture pattern.

Discussion of the deforming forces on the fracture fragments.The supraspinatous and infraspinatous pull the greater trochanter posterosuperiorly and externally rotate the the fragment.The subscapularis pulls the lesser trochanter medially.The pectoralis major pulls the proximal humeral shaft anteromedially.

EXAMINER: What next?

CANDIDATE: I would order a CT scan of the shoulder.

EXAMINER: Here is the CT scan.

The candidate didn't understand how to interpret the pictures and gave a poor description of the anatomy being unable to identify the lesser and greater tuberosities[1]

EXAMINER: The candidate knows how to order a CT scan but not how to interpret the scans.

COMMENT: Practice looking at a CT scan of the shoulder, be able to identify the relevant anatomy and follow on from this with a fracture description.

A CT scan more clearly defines fracture configuration and is indicated for comminuted intra-articular fractures involving the humeral head. It gives an idea of fracture pattern, need for fixation and additional info for planning surgery.

Only a brief discussion of Neer's classification. Much greater part of the viva spent discussing which fracture pattern needs fixing and which could be managed conservatively. Little was discussion about age and co-morbidity factors influencing management options.

The impacted bifocal fracture pattern (AO11–B1.1) represents nearly 15% of all proximal humerus fractures. This type B1.1 fracture may be represented by a minimally displaced fracture, a displaced two-part surgical neck fracture, a displaced two-part greater tuberosity fracture, or a three-part variant with significant displacement of both the surgical neck and greater tuberosity with valgus impaction[21]. These fractures occur in elderly patients in whom reconstructive surgery is difficult.

Court-Brown et al.[22] reviewed 125 patients with B1.1 fractures with a mean age of 71 years who all had a valgus impacted B1.1 fracture treated non-operatively. All fractures united with 80% good to excellent results at one year using the Neer criteria.

COMMENT: Methods of fixation include percutaneous techniques with pins or screws and open reduction and internal fixation with contoured locking plates or intramedullary fixation. Fractures that are not amenable to operative fixation, including comminuted fractures, head-split injuries, and associated fractures.

EXAMINER: Which fractures are prone to AVN?

CANDIDATE: Hertel et al.[23]. devised a method of assessing the risk of osteonecrosis and concluded that the critical components include the integrity of the medial hinge and the length of the posteromedial metaphyseal head extension. Fractures resulting in disruption of the medial hinge and those with 8 mm of calcar bone attached to the articular segment were more likely to be associated with the development of osteonecrosis.

COMMENT: This is a difficult question which probes indepth understanding of this fracture.

Intramedullary nailing (IMN) vs plating of humeral shaft fractures

'I was shown a radiograph of a comminuted midshaft humeral fracture. It was a young patient and the inference from the examiners was that that was unsuitable for conservative treatment. Almost immediately we began discussing the pros and cons of IMN vs plating along with various radial nerve scenarios.'

'The examiners were keen to know about the literature evidence which I vaguely knew about but not to any great degree.'

'In retrospect I should have been more skillful with the question steering the discussion onto safer territory for myself.'

'For starters I should have begun by stating that the vast majority of humeral shaft fractures are treated conservatively. The indications for operative treatment included polytrauma, floating elbow, segmental fracture, pathological fracture, open fracture, non-union, mal-union, progressive vascular impairment and inability to maintain reduction with conservative treatment. This would have scored me some initial points.'

When comparing plating vs IMN the arguments should focus on biology and principles of fracture healing, biomechanics and complications.

Intramedullary nailing results in higher rates of re-operation and no differences between rates of non-union, infection or iatrogenic nerve injury when compared to compression plating. IMN is associated with more shoulder pain, reduced range of shoulder movement and possibly shoulder impingement. IMN may cause considerable shoulder morbidity and be poorly tolerated in a younger more active patient.

There are some types of humeral shaft fracture best suited for IMN. An extensively communited long fracture is better nailed as plating would involve widespread stripping of soft tissue that may result in delayed or non-union . If the fracture pattern is nearby the radial nerve it is best be reduced and fixed

[1] The candidate appears stupid to us – asking for a CT scan of the proximal humerus but not being able to even remotely identify any structure. The examiners were more objective and forgiving.

under direct vision protecting the nerve rather than blind closed reaming and nailing as this may injure the radial nerve.

Associated radial nerve injury

Radial nerve injury occurs in approximately 10% of humeral shaft fractures with an increased risk with middle third and junction of middle/lower one-third fractures. Around 90% are neuropraxia and recover fully, but the nerve may also be lacerated or caught between fracture ends. Transverse and spiral fracture patterns are significantly more likely to be associated with a radial nerve palsy than oblique or comminuted fractures.

There are essentially six scenarios of radial nerve dysfunction:

1. **Open fractures**

   Because irrigation and debridement is required for open fractures, it is reasonable to explore the nerve at this same operation
2. **Radial nerve palsy following closed reduction**

   Although there is some controversy in the literature the safe exam answer is that if the nerve was functioning before the manipulation then nerve exploration is required to ensure the nerve has not been trapped or lacerated
3. **Radial nerve palsy with an unstable fracture pattern**

   The fracture should be plated and nerve exploration undertaken at the same time
4. **Radial nerve palsy with a stable fracture pattern**

   A conservative approach may initially be adopted
5. **Radial nerve palsy following IMN**

   This depends on the level of the fracture and the placement of locking screws.Either avoid IMN if the fracture is at the level of the spiral groove, wide fracture gaps or significant comminution present or expose the nerve to ensure no injury during fracture reduction or reaming. Gentle fracture reduction and minimal reaming. If the nerve has been directly visualized and a palsy develops then this may be observed. However, if a nerve palsy develops following a blind closed IMN it is necessary to explore
6. **Radial nerve following plating**

   If the nerve was seen at surgery and protected, the plate applied to the humerus avoiding the nerve then the palsy can be observed. Failure to adequately identify the nerve would be an indication for exploration

Other upper limb trauma topics

Other upper limb trauma topics include:

- Principle of scapular fixation
- Floating shoulder SCC
- Brachial plexus management principles

  Diagram of clockwork
  MRI
  Stanmore peripheral nerve service or your local referral pathways
- Complex C3 elbow fractures

  Fix
  Total elbow replacement/distal humeral resurfacing
  Open elbow fracture dislocations and their management
  Articulating elbow X-fix application
- Distal biceps avulsion

  1 vs 2 incision reconstruction: Boyd–Anderson[24]
- Terrible triad. Management. It still dislocates, what next? Coronoid reconstruction. Articulating external fixator
- Radial head fracture: Anatomy approaches/classification/fixation vs spacer.
- Scaphoid fractures
- Complex carpal injuries
- Hand/tendon injuries and infections

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Chapter 30

Basic science oral topics

Kevin P. Sherman

Introduction

The basic science oral is often approached with trepidation by candidates; in many cases this trepidation is justified because 'revision' of the basic sciences has in fact been learning for the first time and often at the last minute.

Despite this a working knowledge of the basic science that underpins clinical practice is essential for an understanding of why we do what we do.

Basic science should not, therefore, be seen as a topic in isolation but should be seen as integral to the various clinical and technical aspects of Orthopaedics and it should be learnt throughout training; unfortunately, this is often not the case. When revising for the examination the basic science topics should be learnt in relation to their clinical context as the questions will usually be posed in a clinical scenario-based way.

When revising for the basic science oral it is essential to ensure that you really understand each topic and not just that you can reproduce various equations and diagrams from textbooks; all too often candidates become stuck when asked to draw a diagram that differs slightly from the ones in the textbooks (which are, of course, known to the examiners!), such as the free body diagram for the left hip rather than the right. Examiners can pick up very quickly when a candidate is just reproducing something rote fashion.

A good way of ensuring that you really understand a basic science topic is to revise in small groups and make sure that you can explain the 'how' and 'why' of the topic to another member of the group and, where appropriate, draw a diagram. The ability to draw a diagram during the examination is a skill that should be practised well beforehand and not done for the first time during the examination itself.

Some tips for drawing diagrams:

- Make the diagram big enough
- Make sure you can label the diagram
- Make sure you can explain what the diagram shows
- Where the diagram refers to a three-dimensional object, make sure that you can draw it from another angle or with the left and right sides reversed

An organized, structured answer will score much more highly than a disorganized one. Final revision for the basic science oral is more usefully devoted to ensuring that you can produce a logical list of headings under which you could discuss the various topics; write these headings on revision cards. This will enable you to structure your answers better and not just produce a series of random one-line answers.

When organizing your revision for the basic sciences, make sure that you refer to the curriculum, which will guide you on the levels of knowledge that are expected on the various topics.

The basic science section of the examination syllabus includes the following headings:

- Anatomy
- Tissues
- Physiology
- Biochemistry
- Genetics
- Biomechanics
- Bioengineering
- Bone and joint diseases
  - Osteoarthritis
  - Osteoporosis
  - Metabolic
  - Bone diseases
  - Rheumatoid and other inflammatory arthropathies
  - Haemophilia
  - Inherited musculoskeletal disorders
  - Neuromuscular disorders
  - Osteonecrosis
  - Osteochondritides
  - Heterotopic ossification
  - Bone and soft-tissue primary tumours
  - Metastases
- Investigations (radiological, etc)
- Operative topics
- Infection
- Thromboembolism
- Pain
- Prosthetics and orthotics
- Statistics
- Research and audit
- Medical ethics

This section of the book will take you through areas that are commonly tested from the above list. The content cannot be

comprehensive; you should check through the above list after reading this chapter and identify areas of weakness in your own knowledge. Preferably obtain objective evidence of areas of weakness from others in a revision group as it is easy to convince yourself that you know about a topic and then find that, when you are asked to draw a diagram or to explain the topic in a clinical setting, you do not in fact know it as well as you think you do. One of the commonest fallacies I have heard is 'I know it, I just can't describe it/draw it' – This phrase is, unfortunately, an exercise in self deception; if you cannot describe something to someone else you do not in fact understand it.

Anatomy is an important component of the basic science oral but it will not be covered in this chapter as it is dealt with comprehensively in other texts. It is important not to forget to revise surgical approaches as you are very likely to be given a question on this subject. You will be expected to be familiar with commonly used approaches, such as those to the bones of the forearm, or the surgical approach for decompressing a compartment syndrome. However, do not neglect the less commonly used approaches, as you will be expected to have an adequate knowledge about surgical approaches that are not necessarily in the day-to-day practice of the more generalist orthopaedic surgeon. The posterior approach to the shoulder, the anterior approach to the cervical spine, the brachial plexus, the posterior approach to the knee and approaches to the subtalar joint have all been asked on several occasions and are good vehicles for exploring a candidate's knowledge of the topographical anatomy of the area.

The principles of the management of bone tumours are frequently asked in the basic science oral, but this topic is discussed elsewhere in this book.

Candidates frequently ask which topics have been asked in recent years. The examination is constantly evolving, and new topics can arise in any diet, or old questions may be asked in a new way. The following are popular topics that it is essential to understand well, although there are, of course, no guarantees of what will come up in any particular examination – The only way to be confident is to have a good understanding *across the breadth* of the curriculum and *in sufficient depth:*

- The ultrastructure and mechanics of articular cartilage
- The structure and function of the meniscus
- Structure and function of the intervertebral disc
- Osteoporosis – Pathology and diagnosis
- Calcium and vitamin D metabolism
- The gait cycle
- Mechanical properties of metals
- Mechanical properties of viscoelastic materials
- Prosthetic design (e.g. hip and knee replacements)
- Tribology, including wear modes and mechanisms, and joint lubrication
- Working length of intramedullary nails and plates
- Genetics
- The science of radiological investigations

Finally, although this chapter is organized under discrete headings, your understanding should not be too compartmentalized; you should be able to discuss a topic that ranges across these various headings. An example would be a question that starts by asking about the ultrastructure of articular cartilage and then develops into a question about the kinematics of a joint. Another example would be a question on the kinematics of the subtalar joint that went on to explore the gait cycle. The most important thing to be able to demonstrate to the examiners is that you have a true *understanding* of the topic and that you can *apply* it in a clinical setting; this is much more important than memorizing some minute detail or figure and then reproducing it out of context.

Genetics

A number of orthopaedic conditions display genetic inheritance patterns and an understanding of these patterns is important[1]. You should be able to talk about one or two examples of each of the main inheritance patterns.

Genetic abnormalities may be grouped into chromosomal, single gene or multiple gene abnormalities.

Before discussing examples of these abnormalities it will be useful to revise the structure of DNA and the way in which it is structured functionally.

The structure of DNA

The whole structure of DNA can be described as a double helix consisting of two spiraling sugar-phosphate backbones with a chain of paired bases between them, like the steps on a spiral staircase.

- The building blocks are a sugar (deoxyribose), a phosphate group and a base
- The 5 carbon atoms of the deoxyribose are numbered 1' to 5' (1-prime to 5-prime) clockwise from the Oxygen atom to 4' with 5' being in the $HOCH_2$ side chain attached to the 4' carbon atom of the ring. Figure 30.1 illustrates the structure of deoxyribose
- A phosphate group becomes attached to the 5' carbon atom in place of the –OH group

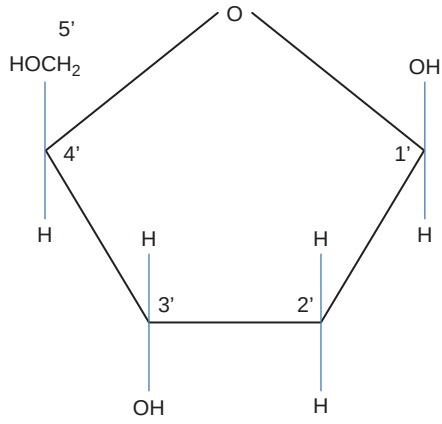

Figure 30.1 Deoxyribose

- The bases are four in kind – Cytosine, thymine, adenine and guanine. Adenine and guanine are both purines (single ring structures). Cytosine and thymine are both pyrimidines (double ring structures)
- One of the four bases attaches to the 1' carbon atom with the loss of OH group on the 1-prime carbon atom in a 'condensation reaction'
- The chain of the nucleotide is built up by the phosphate group attached to the 5' carbon linking with the 3' carbon atom on the next nucleotide with the loss of an –OH from the phosphate group and an –H from the 3' carbon (another condensation reaction leading to the loss of a molecule of water)
- The resulting chain will have a 'spare' –OH attached to the 5' carbon atom at one end (the '5" end) and a 'spare' –OH attached to the 3' carbon atom at the other (the '3'end'; the chain has direction. When the two chains are joined to form the DNA one sugar-phosphate backbone will have the spare –OH attached to the 5' carbon atom at the 'top' and the spare –OH attached to the 3' carbon atom at the 'bottom', and the other will be the other way up. The base pairs are always formed with one purine and one pyrimidine
- One of the two strands in the DNA is the *coding strand* and the other is the *template strand*
- A sequence of three bases in the coding strand of the DNA forms a Codon, which codes for amino-acid synthesis. The codons are read from the 5' end to the 3' end. Only two amino acids are coded by a single codon, the others require more than one codon. Other codons signal the start and stop of sequences; these are called *signaling codons*
- A section of DNA that controls the formation of an amino acid chain or polypeptide chain is a gene. To date only about 5% of the DNA has been identified as forming coding genes; the function of most of the rest of the DNA (about 95%) is unknown
- Each of a pair of genes occupying equivalent sites on the two matched chromosomes is called an *allele*; if they are the same the then the genes are *homozygous* and if they are different there are *heterozygous*

Some key terms

Despite the genes for certain conditions being present (i.e. the genotype is present) some people may not have the condition or they may have a condition to a greater or lesser extent. The terms variable penetrance and variable expression can cause confusion:

Penetrance: Despite the genes for the condition being present only a proportion of the population with these genes may exhibit the condition. Penetrance describes the percentage of the group with the genes for the condition who actually show the features of that condition – It is a statistic for a *group*. Examples include osteogenesis imperfecta and achondroplasia. The cause of incomplete penetrance is not completely understood and may include interference from other genes or the need for some environmental factors to be present for the genotype to be reflected in the phenotype. The importance of incomplete penetrance is that even if the condition is not apparent, due to lack of penetrance, the genotype is present and the condition can, therefore, be passed on to children

Expression: This term describes the degree to which the features are expressed in an individual. Only a proportion of the typical features may be manifested in the *individual*. An example is Marfan's syndrome

Incomplete dominance – Intermediate expression: There is incomplete dominance resulting in a range of the characteristic being expressed (example is human voice pitch – No obvious orthopaedic example)

Co-dominance: Both forms expressed – Example is blood grouping – No obvious orthopaedic examples

Multiple alleles: Characteristics controlled by multiple sets of genes – Many examples, including the HLA immune system

Spontaneous mutations – Not all genetic abnormalities are inherited from parents – In achondroplasia 83% of cases are spontaneous mutations

Mosaicism (Lyonisation) – Not all cell lines are affected. Lyonisation is also instrumental in X-linked conditions being found in males but females only being carriers – This depends upon the affected X-chromosome being suppressed

Regulator genes: These are responsible for turning other genes on and off. Examples include the Homeobox and Hox genes, which regulate the formation of body parts, such as limbs, etc. These also regulate maturation and ageing

Modifier genes: These genes modify the effects of other genes

Stuttering alleles: The defective gene segment doubles with each successive generation leading to progressively worsening symptoms in each successive generation – An example is myotonic muscular dystrophy

In the following section the differing types of inheritance patterns will be described, with some examples of orthopaedically relevant conditions. The clinical features of these conditions are beyond the scope of this chapter.

Chromosome abnormalities

- Whole chromosome abnormalities – Incorrect number of chromosomes **(aneuploidy)**
  - Loss of a chromosome – Only survivable if affecting X chromosome (XO – Turner's syndrome, 1 in 2000 live births, loss of paternal X, short-stature female, scoliosis, hip dislocation, cubitus valgus, short fourth metacarpal/metatarsal)
  - Extra chromosome – E.g. trisomy 21; 1 in 600 live births, increasing risk with increasing maternal age, usually failure of separation at meiosis (94%)
  - Structural abnormalities of chromosome

		Mother	
		A	a
Father	A	AA	Aa
	a	Ab	aa

Figure 30.2 Punnet square

- Point mutations, deletions, inversions, translocations
- may be autosomal or X-linked
- Often variable penetrance or expression

Mendelian inheritance (single gene)

Single gene traits are predictably inherited in fixed proportions. Many candidates become confused when asked to calculate the probability of a particular condition being inherited from a parent who has, or carries, a condition, and you should be well practised at drawing Punnett squares. Figure 30.2 illustrates a Punnett square, where capital letter 'A' indicates a dominant gene and 'a' indicates a recessive gene.

- **Autosomal dominant conditions**
  - Clinical cases usually heterozygous (homozygous usually fatal)
  - Males = females
  - Probabilities: If one parent has the condition 50% chance of inheritance of abnormal gene
  - Examples – Achondroplasia, multiple epiphyseal dysplasia and most cases of osteogenesis imperfecta (although some cases of OI are autosomal recessive)
- Most cases of achondroplasia have normal parents; this is due to a high spontaneous mutation rate. Most cases are due to a defect in the Fibroblast Growth Factor Receptor gene 3 (FGFR3), which is found on chromosome 4. The defect causes a deficiency of chondrocyte growth
- **Autosomal recessive**
  - Homozygous have the condition, heterozygous carry the condition
  - Males = females
  - Probabilities: If both parents carriers, 25% chance of child being affected, 50% chance of children being carriers
  - Examples – Mucopolysaccharidoses (except type II), sickle cell disease, the severe forms of hypophosphatasia (milder forms of hypophosphatasia may be autosomal dominant)
- **X-linked dominant**
  - Females more commonly affected than males
  - Inheritance pattern depends on whether the mother or father has the affected gene. If mother has the gene 50% of sons and daughters inherit the condition
  - Probabilities: If father has the gene 100% of daughters and none of sons inherit the condition
  - Example – Hypophosphataemic rickets (defective *PHEX* gene, causing failure to break down Fibroblast Growth Factor 23 (FGF23) resulting in lack of reabsorption of phosphate in the renal tubules)
- **X-linked recessive**
  - Homozygous affected, heterozygous carriers
  - Males more commonly express the condition than females
  - Probabilities: If the mother is a carrier, 50% of sons will have the condition and 50% of daughters are carriers. If the father has the condition and the mother a carrier, 50% of the daughters carry the gene, 50% of the daughters have the condition, 50% of sons have the condition
  - Examples – Duchenne's muscular dystrophy (fault in the dystrophin gene, which has an adverse effect on the muscle cell membrane), haemophilia

Non-Mendelian inheritance

For chromosomal and single gene conditions the inheritance pattern can be relatively accurately predicted, but many conditions, including orthopaedic conditions, have a less predictable pattern of inheritance. There are many causes of this but some of the factors are as follows:

- Polygenic inheritance
- Variable penetrance and expressivity
- The effect of modifier genes
- Environmental factors may cause the phenotype to become expressed

In conditions with non-Mendelian inheritance closer relatives of someone affected will have a higher risk of developing the condition than more distant relatives.

- Example – Talipes equinovarus; 1 in 1000 live births. Risk increased by 25 in first-degree relatives (1 meiosis away – parent, sibling, offspring); risk increased by 5 in second-degree relatives (2 meioses away – Grandparent, grandchild, aunt, uncle, nephew, niece)

Another relevant example is low back pain, where there is probably a significant genetic factor (including the *PARK2* gene and a gene regulating Collagen IX formation) but where other factors also influence the development of symptoms.

Embryology

Although a detailed knowledge of embryology is not required, some understanding of the formation of the spine and limbs helps to explain the anatomical arrangements of nerve roots and spinal nerves and limb development abnormalities.

Somite formation

The spine forms from *somites,* which develop as paired structures in the paraxial mesenchyme either side of the notochord and neural tube. Somitic condensation is regulated by the *paraxis* gene. Initially a spherical epithelial somite forms

around free somitocele cells. The somites then condense into three layers: The sclerotome, the myotome and the adjacent dermatome.

Sclerotome formation

The somite boundaries from the ventral and ventromedial walls of the somite develop into the *sclerotomes,* which will develop into the vertebral bodies and vertebral arches. Sclerotome condensation is regulated by the *Pax1* gene. The ventral and medial mesenchymal cells move around the notochord and neural tube. The dermomyotome forms from the epithelial plate of the somite.

Sclerotome division

Each sclerotome divides into a cranial and caudal part (the line of division being von Ebner's fissure). The division of the sclerotome is regulated by the *Mox1* gene. The caudal part of the sclerotome fuses with the cranial part of the next sclerotome to form a vertebra. The cranial portion of the first cervical sclerotome fuses with the occiput and the caudal portion of the eighth sclerotome fuses with the cranial part of the first thoracic sclerotome to form the first thoracic vertebra. The spinal nerve grows through the cranial half of the sclerotome (which becomes the caudal half of the vertebra). These arrangements explain the relationship of the nerve root to the vertebrae in the region of the foramina and also explain why there are eight cervical nerves and only seven cervical vertebrae.

Vertebra formation

The sclerotome consists of central, dorsal, ventral and lateral parts, with the dermomyotome covering the dorsal, central and lateral parts. The ventral part lies close to the notochord. The sclerotomal cells surrounding the notochord develop into chondroblasts and become the centrum of the vertebral body. The pedicles and ventral part of the neural arches also form from the central part of the sclerotome. The dorsal part of the vertebral arches form from the dorsal part of the sclerotome, which invades the space between the neural tube and the surface ectoderm.

The neural arch forms from coalescence of paired bilateral pedicles and laminae. Three projections arise from the junction of the pedicle and lamina: Cranial and caudal articular processes and lateral projections to form the transverse processes.

Intervertebral disc formation

The intervertebral discs form from both notochordal cells and somitocele that have remained mesenchymal. The notochordal cells in the region of the centrum of the vertebral body are replaced by sclerotomal mesenchymal cells, but those in the region of von Ebner's fissure expand to form the nucleus pulposus of the disc. The annulus fibrosus forms from the sclerotomal mesenchymal cells surrounding the notochord. By the second decade of life the notochordal-derived cells are replaced by cells from the inner layer of the annulus fibrosus. The intervertebral joints also form from somitocytic mesenchymal cells in the same region.

- The intervertebral discs and the spinal nerves are segmental
- The vertebrae are intersegmental
- The process of segmentation progresses from cranial to caudal

Limb formation

Limbs form between the fourth and eighth week of gestation.

At limb levels the ventrolateral border of the sclerotome proliferates to form the limb bud. The limb buds develop from a combination of mesenchymal cells (note that the dermomyotome gives rise only to the epaxial skin and muscle, i.e. dorsal to the spine, and not to the limb muscle and skin). Initial limb bud formation is controlled by fibroblast growth factor.

Limb bud axes

The limb can be divided into three axes:

- *Proximodistal axis*
- *Craniocaudal axis* – Any line running from the pre-axial to the post-axial border perpendicular to the proximodistal axis
- *Dorsoventral axis* – Any line perpendicular to both the proximodistal and craniocaudal axes

Growth along the proximodistal axis is controlled by the apical ectodermal ridge (AER), which maintains the adjacent rapidly dividing underlying mesenchymal cells (the progress zone) in an undifferentiated condition. Severe transverse phocomelic defects may be caused by damage to the AER.

Differentiation along the craniocaudal axis is controlled by a small population of somatopleuric mesenchymal cells on the post-axial border of the limb bud: The zone of polarizing activity (ZPA). The type of digit formed depends on the distance from the ZPA and the number of digits depends on the width of the AER. ZPA function is controlled by the *shh* gene (sonic hedgehog) via *HOX* genes.

Growth along the dorsoventral axis is controlled by the surface ectodermal covering of the limb bud. The dorsal surface grows faster than the ventral, leading to ventral curving of the limb.

Bone

As it is one of the main 'raw materials' in orthopaedics, an understanding of the structure and function of bone is essential for the examination. This section will deal initially with normal bone and its development and will then go on to cover diseases and disorders of bone and bone metabolism.

Function

- Biomechanical – Framework for support and propulsion
- Biomechanical – Protection of soft tissues

- Biochemical – Mineral homeostasis
- Haematological – Marrow cells

Development

Long and short bones develop in cartilaginous precursors – Enchondral ossification.

Flat bones develop in loose condensations of mesenchymal tissue – Intramembranous ossification.

Structure

Bone consists of cells (10%) within a matrix (90%) that has inorganic and organic components. The proportion of inorganic to organic matrix is approximately 60 : 40. The mechanical properties of the bone are greatly influenced by the organization of the structure (trabecular or cancellous).

Primary bone (in embryo, in growing metaphysis and following fracture) is usually formed as woven bone, which has a relatively random orientation of collagen fibres; this then becomes remodelled into lamellar bone, which has an organisation appropriate to its mechanical function

Woven (immature) bone

- Forms more quickly than lamellar bone
- Has less mineral content than lamellar bone, with smaller crystals
- Is more flexible than lamellar bone and has a higher turnover
- Contains more osteocytes than lamellar bone
- Is isotropic – Collagen fibres are aligned randomly

Lamellar (mature) bone

- Lamellar bone is organized into layers, or lamellae
- Collagen fibres arranged according to the stresses on the bone
- Is anisotropic
- Stronger than woven bone
- Has lower turnover than woven bone
- Primary lamellar bone forms at the periosteal surface of long bones

Trabecular bone

- Found in epiphyses and metaphyses of long bones, and the major part of short bones (short bones are mainly cortical in thin areas and have a trabecular layer in thicker areas)
- Made up of lattice of plates and/or rods (called struts) in a 'honeycomb' arrangement
- The arrangements of the plates and rods dictates the mechanical properties
- In lamellar trabecular bone the lamellae are approximately parallel to the strut surfaces and form packets
- Packets are separated from each other by cement lines
- In osteoporosis the thickness and number of struts decreases
- No Haversian system

Cortical bone (compact bone)

- Forms the cortex of long bones and also occurs in flat bones
- Osteoblasts deposit bone matrix in thin sheets, or lamellae
  - Some of the osteoblasts become left in small lacunae and become osteocytes (others become bone lining cells and others apoptose)
- The structure has a cylindrical arrangement with concentric lamellae osteons, or a Haversian canal system around a central Haversian canal containing neurovascular structures
- Osteons usually have about 5–7 lamellae
- The osteon is surrounded by a cement line, which can be relatively weak
- Volkmann's canals run perpendicular to the Haversian canals and connect them to each other
- Interstitial lamellae are found between osteons and are the remnants of remodelling of cortical bone; these do not contain Haversian canals
- The concentric arrangement of lamellae, and possibly also the cement lines, help to resist crack propagation through the bone

Constituents of bone

Bone consists of an extracellular matrix (ECM) and cells. These will be discussed below.

Extracellular matrix

The bone matrix forms provisionally as osteoid, which is formed by osteoblasts. The bone ECM consists of a mineral inorganic component (60–70% dry weight) and an organic component (30–40% dry weight).

Inorganic matrix

- The bone matrix differs from the extracellular matrix of osteoid or lacunar ECM in having a large inorganic component
- Bone surfaces have a thin layer of non-mineralized osteoid
- Mineral is closely associated with collagen fibres
- Calcium hydroxyapatite – $Ca_{10}(PO_4)_6(OH)_2$ crystals are laid down in the holes and pores of the helically arranged collagen fibrils
- The mineral content of the matrix gives it compressive strength and stiffness

Organic matrix

- Collagen
- Non-collagenous proteins
- Growth factors
- Proteoglycans

Collagen

- Approximately 90% of organic matrix

- Mainly type I collagen (the word *bone* contains *one* as its last three letters, making it easy to remember *type one* collagen
- Collagen is arranged in a triple helix, with two α1 chains and one α2 chain
- Gives the bone tensile strength
- The collagen (tropocollagen) helices form large complexes called fibrils. Fibrils combine to form collagen fibres
- Collagen fibrils have a staggered arrangement of helices, with gaps (holes) between the ends and pores running between the fibrils, into which the mineral is deposited

Non-collagenous ECM proteins

- Osteocalcin – Associated with mature osteoblasts, so a good marker of bone turnover (urine levels increase both when bone formed and also when released from matrix when bone resorbed)
- Adhesive proteins – E.g. fibronectin, vitronectin, etc interact with osteoblasts and osteoclasts to control their adhesion to bone surfaces
- Matricellular proteins – E.g. osteonectin are involved in cell-mediated functions and control of mineralization
- Phosphoproteins – May be involved in the initiation of mineralization
- Growth factors and cytokines – E.g. bone morphogenetic proteins (BMPs), insulin-like growth factor (IGF), basic fibroblast growth factor (bFGF), etc, involved in regulating bone turnover and bone cell differentiation

Proteoglycans

- Linear protein core with long chain glycosaminoglycan side chains
- Many different types
- Less abundant in bone than in cartilage
- Mainly regulatory function

Bone cells

Osteoblasts, osteocytes and bone lining cells all arise from mesenchymal stem cells (MSC) originating in the bone marrow. Osteoprogenitor cells are cells that are committed to osteoblastic differentiation.

Osteoblasts[2]

- Bone-forming cells found on surface of bone
- Regulate activity of osteoclasts
- Form bone matrix and may also facilitate mineralisation
- Deposit osteoid (type I collagen) on mineralized ossification front
- Eccentric nucleus. Detect hormones at apical surface, secrete matrix at basal surface
- Have a number of different receptors, including PTH receptor, 1,25-diydroxyvitamin D receptor, prostaglandin, oestrogen and glucocorticoid
- Secretory pathway – Rough endoplasmic reticulum → Golgi apparatus → secretory vesicles
- Important regulator of activity is LRP5 (low-density lipoprotein receptor-related protein 5)
- Osteoblasts differentiate from mesenchymal stem cells under the influence of a number of factors, including BMPs, cytokines and growth factors. The process is not completely understood but Runx 2 and Osterix transcription factors are required. Loss of Runx 2 causes cleidocranial dysostosis
- Once differentiated an osteoblast has a half-life of approximately 100 days, after which it will become either a bone-lining cell or an osteocyte or apoptose[3]

Bone-lining cells

- Flat cells lining the surface of bone
- Inactive cells whose function is not fully understood; may be capable of becoming osteoblasts or may prevent the ingress of osteoclasts

Osteocytes

- Form approximately 90% of bone cells
- Form from osteoblasts that become embedded in bone (approximately 30% of osteoblasts will do this)
- When osteoblasts becomes osteocytes they lose much of their secretory apparatus and become non-polarized
- Have high nuclear to cytoplasm ratio
- Produce small amounts of matrix proteins
- Have many narrow cytoplasmic cell processes that extend into bone through the canaliculi to connect with processes from other osteocytes[4]
- It is postulated that they act as mechanosensors

Osteoclasts

- Osteoclasts are of different lineage from osteoblasts, osteocytes and bone-lining cells. Osteoclasts differentiate from haematopoietic precursors
- RANK on the surface of the osteoclast precursor binds to RANKL (RANK Ligand) on the surface of osteoblasts to trigger differentiation – Physical contact between the osteoblast and osteoclast may be required
- In the presence of macrophage-colony stimulating factor (MCSF) the RANKL–RANK interaction stimulates transformation of the osteoclast precursor into an osteoclast
- *Osteoprotegerin* binds to RANKL and prevents it reacting with RANK. Formation of osteoprotegerin is increased by oestrogen and by strontium
- Osteoclasts are large (20–100 μm), multinucleated giant cells, usually with between 3 and 20 nuclei
- Osteoclasts resorb mineralized bone matrix (they cannot resorb unmineralized matrix) – This action is stimulated by interleukin-6 (IL-6) produced by osteoblasts

- Osteoclasts adhere to the bone surface at the sealing zone. Cell attachment anchoring proteins (integrins) are important in this process
- Carbon dioxide is used in the cell to form carbonic acid
- Matrix degrading enzymes (acid-resistant proteolytic lysosomal enzymes such as tartrate-resistant acid phosphatase and cysteine proteinases) are synthesized
- Carbonic acid and matrix degrading enzymes secreted through the ruffled border into the resorption space formed by the sealing zone
- Removal of bone from the bone surface leaves a pit or Howship's lacuna
- Degradation products are absorbed back through the ruffled border and are then either further broken down or secreted into the extracellular space
- There are surface receptors for calcitonon. Calcitonin causes dissolution of ruffle border
- Bisphosphonates cause loss of the ruffled border and, therefore, interfere with the action of osteoclasts. Bisphosphonates may also induce osteoclast apoptosis
- The half-life of osteoclasts is approximately 10 days

Summary of osteoclast activity regulation:

Osteoclast activity is increased by: RANKL, IL-6

Osteocast activity is degreased by IL-10, bisphosphonates, calcitonin

Bone growth

Bone growth, and in particular the growth plate, is a popular topic in the basic science section of the examination. You should be able to draw a growth plate and describe its components. You should also be able to recognise a photomicrograph of a growth plate and not confuse it with articular cartilage!

In the following section the zones of the growth plate will be desribed, followed by a list of some of the disease processes that can affect the different zones, and then finally the effects of various hormones on the growth plate will be considered.

Zones of the growth plate

The growth plate can be divided into a number of zones:

- Reserve zone
- Proliferative zone
- Hypertrophic zone
- Maturation zone
- Degeneration zone
- Zone of provisional calcification
- Vascular invasion zone (primary spongiosa)

The features of these individual zones are given below.

Reserve zone

- Cells relatively quiescent
- High proportion of ECM to cells
- Contains germinal cells
- Cells not organized into columns
- Low oxygen tension (epiphyseal arteries pass through but do not form terminal capillaries)

Proliferative zone

- Chondrocytes ordered into columns
- Cells have flattened appearance
- Cells dividing (cell at epiphyseal side of zone is mother cell)
- High oxygen tension
- Proliferation of cells is controlled in a local feedback loop by three substances synthesized by growth plate chondrocytes: Parathyroid hormone-related peptide (PTHrP), transforming growth factor-beta (TGF-β) and Indian hedgehog (Ihh)[5]

Hypertrophic zone

- Production of ECM separates cells from each other
- Matrix is mainly type II collagen and proteoglycans. The main proteoglycan is aggrecan. Proteogylcans inhibit mineralization
- Cell division ceases
- Chondrocytes increase in size with proliferation of mitochondria and endoplasmic reticulum
- Increase in cell height responsible for about half of growth in length of bone
- Type X collagen formed (uniquely in this zone although its function is unknown)
- Alkaline phosphatase activity high
- Oxygen levels low
- Calcium accumulated in mitochondria
- Matrix vesicles deposited in ECM
- Cell death by apoptosis (no inflammatory response, unlike necrosis), with release of calcium in the zone of provisional calcification

Zone of vascular invasion

- Capillary loops break through the mineralized transverse septum and invade the lacunae left by the apoptosed chondrocytes
- Calcified cartilage bars replaced with woven bone

Secondary spongiosa

Finally, deep to the growth plate, the woven bone remodels in the metaphysis to form lamellar bone.

Hueter–Volkmann law

Increased compression at the growth plate slows longitudinal growth.

Delpech's law states that increasing tension on the growth plate speeds growth.

The underlying mechanisms for this phenomenon remain unexplained.

Physeal–metaphyseal junction

The physeal–metaphyseal junction is the 'weakest' part of the growth plate.

Shearing forces are reduced by:

- Microscopic irregularities – Mammillary processes
- Macroscopic contouring – Undulations

Periphery of the physis

- *Groove of Ranvier* – A peripheral wedge-shaped area of chondrocyte progenitor cells that supplies reserve zone cells to the periphery of the growth plate for lateral growth
- *Perichondral ring of Lacroix* – Dense fibrous band at the periphery of the growth plate, which anchors and supports the physis

Growth in width of long bones

Long bones increase in width by intramembranous ossification (appositional growth) from the osteogenic cells in the inner layer of the periosteum.

Premature growth plate arrest (physeal injuries)

Physeal injuries can result in a bridge (bar) of bone forming across the physeal cartilage. The bars can be central, peripheral or linear. A central bar may lead to limb length discrepancy and a more peripheral bar may cause angular deformity.

Diseases affecting the growth plate

Reserve zone

- Diastrophic dwarfism
- Pseudoachondroplasia
- Gaucher's disease

Proliferative zone

- Achondroplasia
- Gigantism
- Malnutrition
- Irradiation

Hypertrophic zone (zones of maturation, degeneration)

- Mucopolysaccharidoses

Hypertrophic zone (zone of provisional calcification)

- Rickets
- Osteomalacia
- Slipped upper femoral epiphysis (SUFE)

Primary spongiosa

- Metaphyseal chondroplasia
- Acute oteomyelitis

Secondary spongiosa

- Osteopetrosis (abnormality of osteoclasts, internal remodelling)
- Osteogenesis imperfecta
- Scurvy

Effect of hormones and growth factors on the growth plate

The growth plate is affected by:

- Hormones (growth hormone, thyroxine, insulin, parathyroid hormone (PTH), calcitonin)[7,8]
- Growth factors (transforming growth factors, bone-derived growth factor (BDGF), epidermal growth factor (EGF) and fibroblast growth factor (FGF))
- Vitamins (vitamins A, C and D)

These factors influence chondrocyte proliferation and maturation and matrix synthesis and mineralization. Some factors have a specific effect on a particular zone whilst others affect the entire growth plate.

Reserve zone

- Parathyroid hormone
- Interleukin-1 (IL-1)

Proliferative zone

- Thyroxine
- Growth hormone
- Insulin
- Transforming growth factor-beta (TGFβ)

Hypertrophic zone

- TGFβ
- BDGF
- Vitamin D
- Calcitonin

Specific effects of hormones and vitamins

Thyroxine

Increases DNA synthesis in cells in the proliferative zone.

Parathyroid hormone

Direct mitogenic effect on epiphyseal chondrocytes and stimulates proteoglycan synthesis.

Calcitonin

Accelerates growth plate calcification and cellular maturation.

Glucocorticoids

Decrease proliferation of chondroprogenitor cells.

Growth hormone

Affects cellular proliferation.

Vitamin D

Vitamin D deficiency results in an elongation of the cell columns in the growth plate.

Bone remodelling

Bone remodelling is essential to bone function. Bone is a dynamic material and remodelling is occurring continuously. You should be well practised in drawing a cutting cone, or in recognising its features on a photomicrograph.

- Bone adjusts to match its functions by the process of remodelling; woven bone remodels to lamellar bone, damaged bone is replaced and bone adjusts to the forces placed through it by this process
- In *coupled bone remodelling* there is no net change in bone mass, but if uncoupling occurs between resorption and formation there will be a change in bone mass
- Bone turnover in adults amounts on average to approximately 5% per year
- The sequence of events consists of resorption of bone from a surface (possibly being preceded by retraction of bone lining cells), leading to the formation of a Howship's lacuna, followed by the appearance of osteoblasts that lay down new bone. Finally, the osteoblasts become osteocytes, bone-lining cells or apoptose
- The group of cells involved in the process is termed a basic multicellular unit (BMU)
- A remodelling unit (BRU) is an area of bone that has been remodelled by a BMU
- The change from resorption to bone formation is termed a *reversal*
- The junction between the original bone and the site where resorption ceased and new bone formation commenced forms a cement line or reversal line
- The resorption phase is much shorter than the formation phase, in line with the half lives of osteoclasts and osteoblasts, respectively
- In trabecular bone the result of a remodelling cycle at a particular site is the formation of a packet
- Remodelling can only take place at a bone surface, so in cortical bone a surface must be created; this is done by the formation of a cutting cone
- A cutting cone consists of a group of osteoclasts at the tip of the cone, which resorb the bone to create a tunnel. Behind the cutting cone a blood vessel forms and osteoblasts differentiate. The osteoblasts lay down bone matrix, which then becomes mineralized at a calcification front. Some of the osteoblasts become entombed in the new bone to form osteoclasts. The result is a new osteon with a Haversian canal
- The new bone may cut across previous osteons to leave partial osteons or interstitial lamellae

Control of bone remodelling and modelling[6,7]

Hox and *Pax* genes are involved via systemic hormones and local cytokines, growth factors, matrix metalloproteins and other factors. Mechanical loads on bone also affect bone remodelling, probably in response to piezoelectric phenomena.

Wolff's law

Bone remodels according to the stresses applied to it: More stress results in more bone formation and vice versa.

Molecular mechanisms

- Control of remodelling is not completely understood
- There is a 'coupling' between resorption and formation that is regulated through a complex mechanism
- PTH can cause bone-lining cells to retract, thus, exposing the bone surface to osteoclasts. Osteocytes may also be involved in this regulation
- PTH acts on osteoblasts but not on osteoclasts. For PTH to stimulate osteoclasts it must first act on osteoblasts, which form M-CSF (see osteoclast section bullet point 2) and RANKL, which in turn binds to RANK on the osteoclast precursor surface to cause differentiation and stimulation of the resulting osteoclasts
- Osteoblasts produce IL-6, which stimulates the osteoclasts to resorb bone
- Osteoblasts also respond to RANKL to increase bone formation
- OPG (osteoprotegerin) binds to RANKL and inactivates it, thus, blocking the differentiation of osteoclasts. OPG production is stimulated by oestrogen
- Bone resorption may also release BMP, TGFβ and IGF-1 from the matrix, which then stimulate osteoblast differentiation
- When coupling is balanced the bone mass will not change

In summary, the control of bone adaptation to stresses placed through it, and the general process of bone remodelling, is not completely understood. There is known to be a complex mechanism of homeostasis with interaction between the osteoblasts and osteoclasts, which probably also involves osteocytes and bone-lining cells. Control is mediated by a number of factors.

Biomechanics of bone

Bone, like all biological materials, is viscoelastic and anisotropic; this means that its mechanical properties are time-dependent and that they vary with the direction of loading.

- Cortical bone tends to be stiffer and stronger in the axis of the osteons
- Cortical bone demonstrates transverse anisotropy – The mechanical properties within a given plane are similar but differ from those through a plane perpendicular to the given plane
- Cortical bone stiffness and strength are higher at higher strain rates

- Cortical bone become less ductile and more brittle at very high strain rates
- Trabecular bone has lower stiffness and strength than cortical bone
- Trabecular bone is less anisotropic than cortical bone
- Trabecular bone compressive strength is highly dependent on its density
- Trabecular bone is relatively tough (it absorbs a high amount of energy before failing completely)
- Trabecular bone yield point is independent of its mass or the applied load but depends almost entirely on the strain - the strain before yield will, therefore, be the same for osteoporotic and normal bone; the applied load to achieve that strain will, however, differ between the two as the normal bone is stiffer than the osteoporotic bone

Fracture healing

Fracture healing can occur by primary or secondary bone healing.

Primary bone healing

- Requires close anatomical reduction with minimal movement at the fracture site (<2% strain)
- In the initial stages, osteoblasts differentiate from mesenchymal cells and lay down woven bone in any gaps. Lamellar bone may be laid down directly if there are no gaps
- Remodelling then occurs across the fracture site, with cutting cones passing across the fracture site
- Healing is slow

Secondary healing (by callus)

- Requires some motion at the fracture site (>2% but <10%)[8]
- Hard callus forms under periosteum at periphery
- Endochondral callus – Fibrocartilage forms, becomes calcified and is then replaced with bone
- In secondary healing by callus, the callus undergoes a process of progressive stiffening. In the earlier, less stiff, stages it is more resilient to movement at the fracture site but less good at taking loads or resisting deformation. The strength of the healing fracture does not necessarily correlate with its stiffness

Stages of secondary fracture healing by callus

- Stage 1: First week. Haematoma formation with invasion of macrophages, leukocytes and lymphocytes. Proinflammatory cytokines (including IL-1 and IL-6 and tumour necrosing factor α), and peptide signal molecules (including BMPs, TGFβ and PDGF) are present. Progenitor cells invade. Granulation tissue forms
- Stage 2: 1 week to 1 month. Soft callus forms. In this stage, fibrous tissue, cartilage and woven bone form. Chondroblasts and fibroblasts differentiate and form collagen (mainly type II) and fibrous tissue. Proteoglycans are produced, which suppress mineralisation. The chondrocytes then release calcium into the ECM and also protein-degrading enzymes that break down the proteoglycans, thus, allowing mineralization to take place
- Stage 3: 1–4 months. Hard callus forms. The soft callus is invaded by new blood vessels and chondroclasts break down the calcified callus, which is replaced by osteoid (type I collagen) formed by osteoblasts. The osteoid calcifies to form woven bone. The osteoid callus is stiffer than the soft chondroid callus
- Stage 4: Remodelling – Several years. The woven bone is remodelled to lamellar bone. The medullary canal reforms as the bone remodels in response to the stresses placed upon it

Bone graft

Function

- Mechanical (structural support)
- Biological (bone healing)

Graft properties

Osteoconductive

- Acts as three-dimensional scaffold or matrix on which new bone forms
- Supports ingrowth of capillaries, perivascular tissues and osteogenic precursor cells
- Example – Coral scaffolds

Osteoinductive

- Provides a biological stimulus that has the capacity to activate and recruit from the surrounding mesenchymal-type cells, which then differentiate into cartilage-forming and bone-forming cells
- Mediated and regulated by graft-derived factors, including TGF, BMPs (bone morphogenetic proteins), IGF-1 and IGF-2 (insulin-like growth factors), interleukins, etc
- Example – Fresh frozen allograft

Osteogenic

- Graft contains living cells that are capable of differentiation into bone
- Graft has inherent biological activity
- Example – Fresh allograft

Genetics

- *Autograft* (same individual) – Including vascularized and free grafts
  - No immunogenicity
  - No risk of disease transmission

  - Cheap
  - Donor site morbidity
- *Allograft* (another individual, same species)
  - No donor site morbidity
  - Slow incorporation
  - Risk of disease transmission
  - Immunogenic
- *Xenograft* (different species)
  - As allograft but greater potential for rejection
- *Isograft* (genetically identical – Twins or clones!)

Tissue composition

- Cortical
- Cancellous
- Corticocancellous
- Osteochondral
- Bone marrow aspirate

Preservation methods

- **Fresh graft:** Highest antigenicity. Viable cell population with associated cytokine growth factors
- **Fresh frozen:** Less immunogenic than fresh. Preserves BMPs
- **Freeze dried:** Least immunogenic. Loss of structural integrity. Depleted BMPs
- In **bone matrix gelatin (BMG)**

Processing

To remove superfluous proteins, cells and tissues to:

- Reduce disease transmission
- Reduce immune sensitization
- Allow better graft preservation

Methods:

- Physical debridement of unwanted tissue
- Ultrasonic processing with or without pulsatile washing to remove remaining cells and blood
- Ethanol to denature cell proteins and reduce bacterial and viral loads
- Antibiotic soak to kill bacteria
- Freezing or freeze drying
- Sterilization (aseptic vs irradiation if contaminated)

Graft incorporation

The process by which invasion of the graft by host bone occurs, such that the graft is replaced partially or completely by host bone.

Five stages of graft healing (Urist)

1. **Inflammation:** Chemotaxis stimulated by necrotic debris
2. **Osteoblast differentiation:** From precursors
3. **Osteoinduction:** Osteoblast and osteoclast function
4. **Osteoconduction:** New bone forms over scaffold
5. **Remodelling:** Process continues for years

Following bone grafting, a haematoma rich in nutrients forms around the bone graft. Platelet-derived growth factor (PDGF) attracts lymphocytes, plasma cells, osteoblasts and polynuclear cells to the bone graft. Necrosis of the graft occurs and an inflammatory response is established, with which granulation tissue forms, with an ingrowth of capillary buds bringing macrophages and mesenchymal cells. Fibrovascular stroma develops with an influx of osteogenic precursors and blood vessels. IL-1, IL-6, BMP and IDGF are secreted, stimulating osteoblast and osteoclast activity. The graft is penetrated by osteoclasts, which initiate the resorptive phase and incorporation.

These earlier stages are similar for both cortical and cancellous bone, but the osteoconduction and remodelling stages differ between the two types of bone, as indicated in Table 30.1.

In cancellous bone graft the graft is eventually replaced during the remodelling phase by a process of creeping substitution; osteoblasts laying down new bone on the scaffold of dead trabeculae with simultaneous osteoclastic resorption.

In cortical bone graft the initial inflammatory response is slower and osteoclastic resorption then occurs by cutting cones entering the graft. Mechanical strength is lost in the first 3–6 months and returns over 1–2 years.

Table 30.1 Autograft incorporation in cancellous and cortical bone

Autograft incorporation	Cancellous	Cortical
Revascularization rate	Rapid	Slow, used for structural defects
Mechanism and order of repair	Osteoid laid down on dead bone, donor bone later reabsorbed	Donor bone reabsorbed before laying down of appositional new bone
Radiographs	Radiodense	Loss of mechanical strength and reduced radiodensity
Completeness of repair	All donor bone eventually removed. Creeping substitution	Some necrotic bone remains. Cutting cones

Bone banking

Contraindications to allograft donation:

- Any evidence of current symptomatic infection
- History, or suspicion, of past infections: TB, hepatitis B and C, sexually transmitted diseases

- HIV and high-risk activities for HIV
- Malignancy
- Dementia
- Long-term steroid use
- Metabolic bone disease
- Any condition of uncertain aetiology where altered immune competence or viral involvement is suspected or implicated: Rheumatoid arthritis, CJD, multiple sclerosis, etc

Detailed past medical history and social history is obtained. Serological testing is carried out for:

- Hepatitis B and C
- Syphilis
- HIV
- Rhesus status

Bone graft substitutes

Calcium sulphate

- For use as bone substitute the crystals need to be regular shaped and uniform in size
- Osteoblasts can attach to calcium sulphate and osteoclasts can resorb it

Calcium phosphate ceramics

Calcium phosphate ceramics are made from mineral salts by sintering. They form an osteoconductive material.

Hydroxyapatite

- Based on corals. The calcium carbonate in natural coral can be substituted with hydroxyapatite
- Form a good scaffold for bone formation
- Brittle and poor strength

Tricalcium phosphate

- Partially converted to hydroxyapatite once implanted

Calcium phosphate-collagen composites

- Collagen (types I and III) combined with hydroxyapatite or tricalcium phosphate
- No structural strength
- Osteoconductive properties

Calcium phosphate cement

- Paste made from calcium and phosphate
- When mixed forms dahllite
- Has osteoconductive properties but is mainly used as an aid to fixation
- Examples include Norian SRS and α-BSM

Polymers

- Polyglycolic acid and polylactic acid have osteoconductive properties
- Structural scaffold

Bioactive glass

- Silica-based, containing calcium oxide and phosphate
- Form surface layer of hydroxyapatite when implanted
- Current versions have no structural strength

Bone morphogenetic proteins

- BMPs are cytokines
- Over 20 discovered
- Most useful appear to be BMP-2, BMP-7 and BMP-14, which are all members of TGFβ supergene family
- BMP-2 and BMP-7 shown to have osteogenic properties
- Current versions not yet proven but may work best in conjunction with matrix

Bone circulation

Bone receives 5–10% of the cardiac output.

Anatomy

The blood supply is from three sources:

- High pressure nutrient artery system
- Metaphyseal–epiphyseal system
- Low pressure periosteal circulation

Nutrient artery system

- High pressure system
- The nutrient artery originates as a branch from the major artery of the systemic circulation
- The nutrient artery enters the mid-diaphyseal cortex (outer and inner tables) through the nutrient foramen to enter the medullary canal. The foramen passes at an angle to the cortex with respect to epiphyseal growth centres in long bones; hence, 'from the knee I flee, to the elbow I go'
- The nutrient artery branches into ascending and descending arteries, which divide into arteriole branches supplying the inner two-thirds of the diaphyseal cortex from within (endosteal supply)

Metaphyseal–epiphyseal system

- The periarticular vascular complex penetrates the thin cortex and supplies the metaphysis, physis and epiphysis
- In epiphyses with large articular surfaces, such as the femoral and radial heads, the vessels enter in the region between the articular cartilage and the physis and, hence, the blood supply can be tenuous

Periosteal system

- Low pressure system
- The periosteal system forms an extensive network of capillaries covering the entire length of the bone shaft
- Supplies the outer one-third of the cortex
- Very important in children, for circumferential bone growth (appositional)

Physiology: Direction of flow

- In mature bone the flow is centrifugal (inside to outside)
- The direction is reversed in a displaced fracture when there is disruption of the endosteal supply
- Arterial flow in immature developing bone is centripetal (outside to inside) because the periosteum is highly vascular and is the predominant component of bone blood flow
- Venous flow in mature bone is centripetal, with cortical capillaries draining into venous sinusoids to the emissary venous system
- Remember Batson's valveless venous plexus – Accounting for the spread of infection/tumour between the retroperitoneum and the spine

Regulation

- Blood flow to bone is under the regulation of metabolic, humoral and autonomic inputs
- The vessels within bone possess a variety of vasoactive receptors
- The arterial system of bone has greater potential for vasoconstriction than for dilatation

Blood flow to bone after fractures

- Bone blood flow is the major determinant of fracture healing
- Bone blood flow delivers nutrients to the site of bony injury
- The initial response after fracture is decreased bone flow after vascular disruption at the fracture site, with reversal of flow to become centripetal if the endosteal flow is disrupted
- Within a few hours to days bone blood flow increases (a regionally accelerated phenomenon) and peaks at 2 weeks, returning to normal at between 3 and 5 months

Bone metabolism

Questions on calcium and vitamin D metabolism are common in the basic science section of the exam and yet many candidates come completely unprepared on this topic.

- 99% of body calcium is stored in the bone
- The extraosseous fraction, although constituting only 1% of the total, is vital for functioning of nerves and muscles, and also in the clotting cascade
- Many disorders of bone metabolism are 'side effects' of problems with calcium and phosphate control systems
- Calcium circulates in the plasma in two forms
  - Bound to albumin, amounting to just under half the total. The calcium bound to albumin is physiologically inactive
  - Free ionized calcium, which is physiologically active
- The normal plasma concentration of phosphate is between 2.2 and 2.6 mmol/l. When interpreting the plasma level of calcium, the level of free ionized calcium should be assessed by noting the albumin concentration in the specimen
- Calcium and phosphate levels are primarily regulated by two hormones
  - PTH
  - Vitamin D and its metabolites

The interaction between PTH and Vitamin D is complex and regulated by a series of feedback loops.

Parathyroid hormone

Parathyroid hormone (PTH) is the primary immediate regulator of calcium levels in the blood.

PTH is a peptide containing 84 amino acids, secreted by the chief cells of the parathyroid glands. PTH has effects on bone metabolism both as a result of direct effects and also via its effect on the vitamin D pathway, in stimulating production of the active form of vitamin D (1,25-dihydroxycholecalciferol) rather than the inactive form (24,25-dihydroxycholecalciferol).

- Active PTH is formed from a 115 aminoacid polypeptide 'Pre-pro-PTH', the 115 precursor is initially cleaved to a 90 amino acid chain and then to the 85 amino acid chain
- If serum calcium levels fall there is an increase in secretion of the active 85 amino acid form within seconds to minutes
- The half life of the 84 amino acid form is 2 to 4 minutes
- If the calcium levels remain low degradation of the active PTH in the parathyroid cells is decreased within about an hour
- If levels of calcium remain low increased gene expression of PTH occurs within hours to days
- If levels of calcium still remain low the number of parathyroid cells increases within days to weeks

Effect on intestine

- No direct effect
- Indirectly increases calcium absorption via effect on vitamin D pathway

Effect on kidney

- Increases reabsorption of filtered calcium in the kidney
- Increases phosphate urinary excretion (decreases re-absorption)
- Stimulates hydroxylation of 25-hydroxycholecalciferol in the proximal tubular cells

Effect on bone

- Stimulates osteoclastic resorption of bone (this requires a 'permissive' level of active Vitamin D)
- Mobilizes calcium and phosphate from bone

Net effect

- Increases serum calcium
- Effect on phosphate levels may be neutral due to the opposing effects on bone and kidney
- If continuous effect is to increase bone resorption, mainly through effect on RANKL and osteoprogeterin
- If intermittent facilitates bone formation

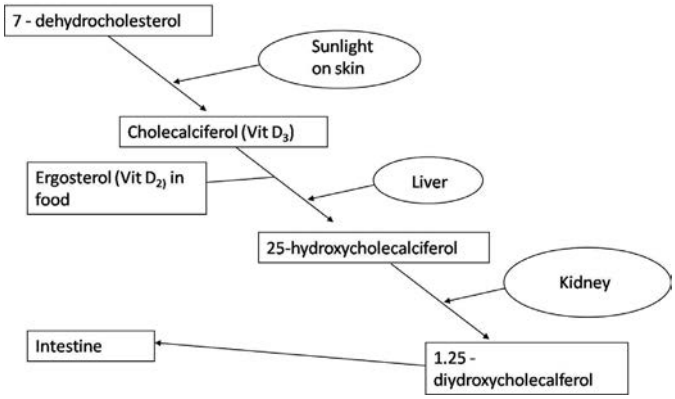

Figure 30.3 Vitamin D metabolism

Factors stimulating production

- Decreased serum calcium

Factors inhibiting production

- Raised serum calcium
- Raised serum 1,25-dihydroxycholecalciferol

Vitamin D

Figure 30.3 shows the metabolic pathway for vitamin D metabolism. 1,25-vitamin D is broken down into at least 25 different metabolites, which have different metabolic actions. The overall action of vitamin D is, therefore, complex.

- Vitamin D is both manufactured in the skin and ingested in the diet
- Vitamin D is fat soluble
- In the skin UV radiation converts the precursor 7-dehydrocholesterol into vitamin D_3. Between 10 and 15 minutes' exposure of the face and hands to sunlight is sufficient to produce the minimum daily requirement of vitamin D
- Vitamin D (vit D_2) is ingested as ergocholesterol in the diet; this is found in fish oils and some plants
  - Vitamin D undergoes two hydroxylations to form the active form – 1,25-dihydroxycholecalciferol
  - The first hydroxylation takes place in the liver
  - The second hydroxylation takes place in the kidney
  - The vitamin D_2 from the gut is incorporated into micelles and transported to the liver in chylomicrons
  - The vitamin D_3 from the skin is transported to the liver bound to Vitamin D binding protein

Effects of vitamin D

In intestine

- Causes increased calcium absorption
- Causes increased phosphate absorption

In the kidney

- Causes increased calcium retention
- Causes increased phosphate excretion (via effect on FGF23)

In bone

- Regulates osteoblast function
- Facilitates PTH induced osteoclast activation

Net effect of vitamin D

- Increases calcium levels in the blood/serum
- Usually increases phosphate level in blood (effect on gut is partially balanced by effect on kidney via FGF23)
- Facilitates bone formation
- If blood levels of calcium and phosphate are low can cause bone resorption

Calcitonin

Calcitonin is a peptide containing 32 amino acids, secreted by the parafollicular C cells of the thyroid gland. It is of lesser importance in the regulation of calcium than PTH and vitamin D.

Effect of calcitonin

- Kidney – Decreases calcium reabsorption
- Gut – Decreases calcium absorption
- Bone – Decreases osteoclast resorption of bone

Factors stimulating production

- Elevated serum calcium

Factors inhibiting production

- Decreased serum calcium

Feedback loops

There are a number of feedback loops in the control of calcium and phosphate levels. These are shown in Figure 30.4.

- Increased serum calcium causes a decrease in PTH and a decrease in production of the active form of vitamin D
- Decreased serum calcium causes an increase in PTH and an increase in production of 1,25 vitamin D
- Increased serum phosphate causes a decrease in production of 1,25 vitamin D
- Increased levels of 1,25 vitamin D cause a decrease in PTH
- Increased levels of 1,25 Vitamin D cause an increase in production of FGF23, which suppresses re-absorption of phosphate

It is of note that in autosomal dominant hypophosphataemic rickets degradation of FGF23 is impaired, leading to excessive loss of phosphate.

Clinical manifestation of abnormal calcium levels

Hypercalcaemia

Clinical features

- May be asymptomatic
- 'Bones' – Excessive bone resorption
- 'Stones' – Renal calculi, polyuria, polydipsia

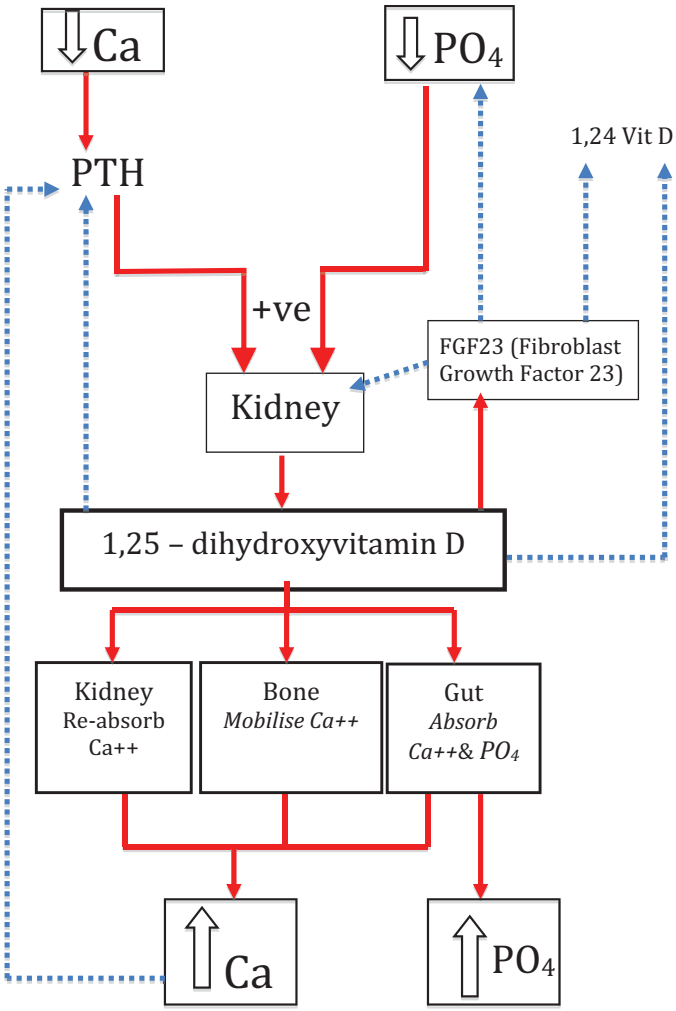

Figure 30.4 Feedback loops in calcium regulation

- 'Groans' – (gastrointestinal) nausea, vomiting, constipation, abdominal pain, anorexia
- 'Moans' – (CNS) lethargy, disorientation, hyperreflexia
- Other side effects – *Sudden* cardiac arrest, hypotension

Causes

- Malignancy
  - Bone destruction from bone metastases or myeloma
  - PTH-like secretion of malignant tumours, e.g. squamous cell carcinoma
  - 1,25-dihydrocholecalciferol synthesis in lymphoma
- Endocrine
  - Pituitary
  - Thyroid
  - Adrenal
- Genetic
  - Familial
  - Multiple endocrine
  - Familial hypocalciuric hypercalcaemia
- Sarcoidosis
- Steroids
- Vitamin D intoxication

Hypocalcaemia

Clinical features – Acute

- Neuromuscular irritability (tetany, seizures, Chvostek's sign, Trousseau's sign)
- Depression
- Perioral paraesthesia
- ECG shows prolonged QT interval

Clinical features – Chronic

- Cataracts
- Fungal nail infections

Causes

- Thyroid surgery or hypothyroidism
- Hypoparathyroidism

Hyperparathyroidism

Hyperparathyroidism may be either primary or secondary. Secondary parathyroidism occurs in response to low serum calcium levels.

Primary hyperparathyroidism

- Parathyroid adenoma (up to 90% of cases) – Usually solitary, occasionally multiple
- Parathyroid chief cell hyperplasia
- Parathyroid carcinoma (rare – 1%)

Secondary (elevated PTH secretion in response to low calcium levels)

- Chronic renal failure (see section on chronic renal failure)
- Vitamin D deficiency or calcium deficiency in diet
- Malabsorption

Effects of hyperparathyroidism phosphate and calcium levels in blood

- Decreased phosphate levels
  - Increased excretion in urine
- Increased calcium levels
  - Increased absorption from intestine
  - Increased reabsorption from kidney
  - Increased mobilization from bone

Effects of hyperparathyroidism on bone

Calcium from bone is predominantly mobilized from the cortical bone, leading to loss of the lamina dura in the teeth, subperiosteal resorption and osteitis fibrosa cystica.

Radiological features of hyperparathyroidism

- Diffuse demineralization (osteopenia)
- Subperiosteal resorption (radial borders of proximal phalanges and tufts of terminal phalanges, skull, medial end of clavicle)
- Osteitis fibrosa cystica ('brown tumours') – Increased giant cells, extravasation of RBCs, haemosiderin staining, fibrous marrow replacement
- Chondrocalcinosis and metastatic calcification of soft tissues
- Shaggy trabeculae
- Deformed osteopenic bones
- Rugger jersey spine
- Bilateral sacroiliac joint widening and erosion

The appearances are caused by increased osteoclastic resorption of bone. Attempts at bone repair fail because of poor mineralisation caused by low phosphate.

Laboratory findings

- Elevated PTH levels
- Elevated plasma calcium
- Depressed plasma phosphate

Osteomalacia and rickets

Osteomalacia is a defect of skeletal mineralization caused by a deficiency of the active metabolites of vitamin D or a deficiency of phosphate. The result is an accumulation of increased amounts of unmineralized matrix (osteoid) and a decreased rate of bone formation.

Rickets is the juvenile form of osteomalacia with impaired mineralisation of cartilage matrix (chondroid) affecting the physis in the zone of provisional calcification. There is a failure to form primary spongiosa.

Clinical features of rickets

The clinical features depend on the severity of the deficiency and the age of onset.

General features

- Retarded bone growth causing short stature
- Symptoms of hypocalcaemia
- Under the age of 18 months may present with failure to thrive, restlessness, muscular hypotonia, convulsions or tetany but only minimal bone changes

Localized features – 'skull to toe'

- Delayed fontanelle closure and frontal and parietal bossing
- Dental disease
- Rachitic rosary (enlargement/hypertrophy of costochondral junction)
- Harrison's sulcus (groove/sulcus/depression in the sternum where the diaphragmatic attachments pull on the softened ribs)
- Pigeon chest
- Protuberant abdomen, hepatomegaly
- Kyphosis
- Genu valgum or varum, anterolateral bowing of distal tibia and secondary adaptive changes in the foot
- Coxa vara, anterolateral bowing of the femur
- Waddling gait

Clinical features of osteomalacia

- Much more insidious onset that rickets
- Bone pain initially vague and non-specific but gradually becoming more severe and sometimes localized
- Proximal muscle weakness

Radiology

Both rickets and osteomalacia

- Looser's zones (stress fractures on concave side of bones)
- Milkman 'pseudofractures' on concave side of bones (fractures that have united but not mineralized)
- Biconcave 'cod fish' vertebrae – Can lead to kyphosis
- Generalized osteopenia

Rickets

- Growth plate enlarged, thickened and widened, disorientated
- Metaphysis cupped, flared and jagged
- Trefoil pelvis

Causes of osteomalacia

Candidates frequently have difficulty remembering the long list of potential causes of osteomalacia and rickets. It is helpful to group the causes in a systematic way. A useful way of doing this is to think of the causes as falling into three main categories: Intake problems, processing problems and output problems.

Intake problems – Affecting the supply of 'raw materials'

- *Inadequate exposure to sunlight*
- *Nutrional deficiency (usually vitamin D)*
  - Vitamin D deficiency
  - Calcium chelators – E.g. oxalates
  - Phosphorus – E.g. aluminium antacids
- *Failure to absorb in the gut*
  - Biliary disease – Interferes with fat-soluble vitamin D
  - Short bowel syndrome
  - Rapid transit syndrome
  - Crohn's and coeliac disease

Processing problems – Affecting the processing of the raw materials. Problems with synthesis or activity of 1,25-dihydrocholecalciferol.

- Vitamin D-resistant rickets

  - Type I – Genetic or acquired deficiency of the enzyme converting 25-hydrocholecalciferol to 1,25-dihydrocholecalciferol
  - Type II – Organ insensivity to 1,25-dihydrocholecalciferol
- Anticonvulsant medication
  - Enzyme that increases vitamin D metabolites induced in liver (phenytoin, phenobarbitone, etc)
- Autosomal dominant hypophosphataemic rickets – Deficient enzyme that degrades FGF23
- Hypophosphatasia – Autosomal recessive; defective phosphate synthesis; increased urinary phosphoethanolamine

Output problems – Affecting the preservation/recycling of essential ingredients

- X-linked hypophosphataemic vitamin D-resistant rickets/osteomalacia
- Albright's syndrome
- Fanconi syndrome – Several types
- Phosphaturia and glycosuria ± aminoaciduria
- Renal tubular acidosis
  - Acquired (systemic disease)
  - Genetic
    - Debré-de Toni–Fanconi syndrome
    - Lignac–Fanconi syndrome
    - Lowe's syndrome
- Renal osteodystrophy

Most common causes of rickets/osteomalacia

To make sense of this list it is also useful to bear in mind the most common causes, which are:

- Chronic renal failure
- Vitamin D deficiency
- Vitamin D pathway abnormalities
- Hypophosphataemic syndromes

Rarer causes include:

- Renal tubular acidosis
- Aluminium toxicity
- Hypophosphatasia
- Mesenchymal tumours causing hypophosphataemia

Diagnosis of osteomalacia

- Clinical
  - Proximal muscle weakness
  - Bone pain and tenderness
  - Fracture (incomplete or bilateral)
- Blood tests depend on cause
- Tetracycline-labelled bone biopsy best (also needed to detect aluminium deposition)

Renal osteodystrophy

Renal osteodystrophy is a topic that many candidates find difficult. The subject can be simplified by thinking of the two main routes by which renal disease affects bone:

- As a result of damage to the renal tubules, the synthesis of the active form of vitamin D is impaired, resulting in impaired calcium absorption in the gut and loss of calcium from the kidneys
- As a result of glomerular damage, uraemia and phosphate retention occur. The elevated phosphate levels further suppress activation of vitamin D in the kidney
- The low serum calcium levels can then lead to excessive parathyroid production, which leads to the features of hyperparathyroidism, which can be further exacerbated by the elevated phosphate levels, resulting in ectopic calcification
- Renal osteodystrophy can, therefore, be divided into two main types
  - High turnover where there is a chronic elevation of the PTH levels
  - Low turnover where the PTH levels are normal or reduced
- The resulting clinical and radiological picture is complex

The changes seen in renal osteodystrophy are summarized in Figure 30.5 and Table 30.2.

Osteoporosis

Predicted demographic changes indicate that fragility fractures will probably become increasingly common over the next few decades. Questions on osteoporosis are, therefore, very likely to arise in the exam as they have great relevance to clinical practice.

The WHO consensus definition states that osteoporosis is a systemic skeletal disease characterized by low bone mass and microarchitectural deterioration of bone tissue, leading to enhanced bone fragility and a consequent increase in fracture risk.

The usually accepted diagnostic criterion is a bone mineral density lower than 2.5 standard deviations below the mean for a race- and sex-matched young adult (i.e. below peak bone mass for a group of the same race and sex).

The biochemistry in osteoporosis is normal.

Risk factors

Primary osteoporosis

- Genetic: Positive family history, white or Asian, thin
- Hormonal: Loss of oestrogen protection
- Environmental/lifestyle: Smoking, excessive alcohol, inactivity
- Diet: Deficiency of calcium or vitamin D

Secondary osteoporosis

- Chronic medical conditions: Endocrine, GI, chronic liver disease, chronic renal failure
- Drugs

Type I and type II osteoporosis

Type I: Affects mainly cancellous bone at the time of menopause, so vertebral and distal radial fractures are common: Related to loss of oestrogen at the menopause; high turnover osteoporosis

Type II: Age-related and affects cortical and cancellous bone; occurs 10–15 years later than type I; poor calcium absorption; low turnover osteoporosis

Assessment

Pitfall: Avoid commenting on osteoporosis on a single plain radiograph; the most you can say is that the bones appear osteopenic.

Table 30.2 Renalosteodystrophy

High bone turnover	Low bone turnover
• Phosphate retention	• Aluminium deposition
– Reduced 1,25-dihydrocholecalciferol	– PTH release inhibited
	• Low PTH
	• Osteomalacia
• High PTH (secondary hyperparathyroidism)	
– Osteitis fibrosa cystica	
– Osteosclerosis	
– Soft-tissue calcification	
But pattern of calcium and bone changes is complex	

Radiographic absorptiometry

DEXA scan

Dual energy x-ray absorptiometry (DEXA): This is currently the 'gold standard' method for detecting and assessing osteoporosis. It is quick and accurate and involves a low radiation dose. The technique involves simultaneous measurement of the passage through the body of x-rays with two different energies. By using two different energy beams it is possible to minimize the effect of soft tissues, particularly fat, on the result.

- Causes of false-negatives in the spine
  - Osteoarthritis with resulting osteophytes and sclerosis
- Cause of false-positive in the spine
  - Previous laminectomy

Quantitative CT

Accurate for vertebral cancellous bone, but expensive and involves a high radiation dose.

Quantitative ultrasound

This is inexpensive and the machine is portable. It involves no ionizing radiation. It is not at present as accurate as DEXA scanning but gives information about the architecture and elasticity of bone.

T- and Z-scores

T-scores present the result as the number of standard deviations above or below the mean peak bone mass for a population matched for *sex and race*.

Z-scores present the result as the number of standard deviations above or below the mean bone mass for population matched for *age, race and sex*.

As the Z-score is measured against an age-matched group it cannot detect age-related osteoporosis. The T-score is used for diagnosis of osteoporosis. A low Z-score, however, indicates that the osteoporosis is *not* age-related and, therefore, a cause for the condition should be sought as there may be some treatable condition.

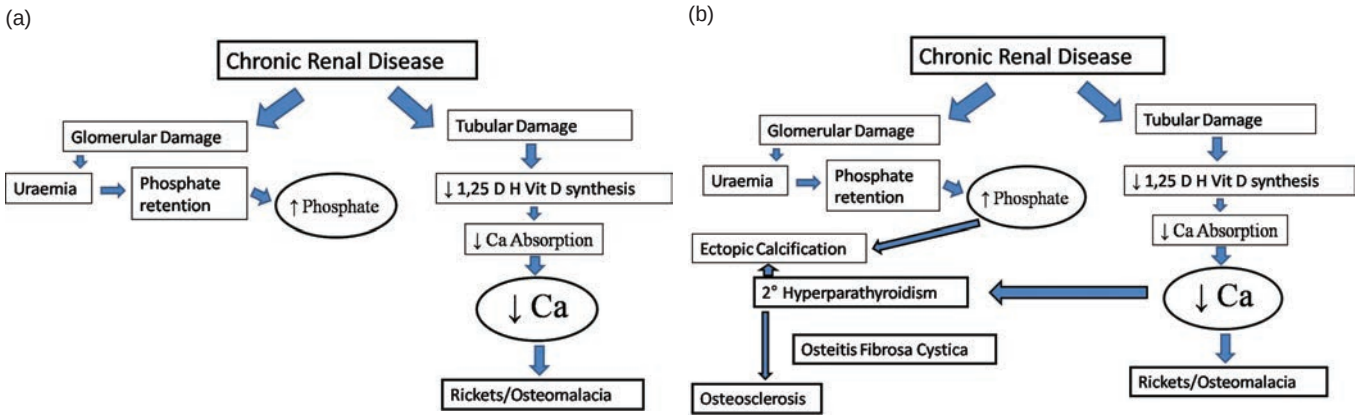

Figure 30.5 (a, b) Renal osteodystrophy

Treatment and management

Evaluation of vertebral fractures in the osteoporotic patient (acronym TOMEO)

Screen for:

- T – Tumour (radiographs, bone scan, MRI and CT)
- O – Osteopenia (DEXA scan)
- M – Marrow (full blood count and serum electrophoresis)
- E – Endocrine (parathyroid, Cushing's disease, thyroid function, diabetes)
- O – Osteomalacia (screening for Vitamin D deficiency)

Simple measures

- Stop smoking
- Reduce excessive alcohol intake
- Exercise and healthy diet

Calcium and vitamin D

- Decreases bone resorption but does not increase bone mass or density
- Evidence to suggest modest protective effect – More effective for type II osteoporosis

Bisphosphonates

- Inhibit/kill osteoclasts
- Preferentially bind to mineral component of bone, which is exposed by osteoclasts
- Non-nitrogen containing Bisphosphonates cause loss of function/apoptosis of osteoclasts by causing accumulation of ATP metabolites within the cell
- Nitrogen containing bisphosphonates inhibit production of cholesterol, which in turn interferes with the cell membrane
- Consider in cases where steroid intake implicated
- Be aware of the risk of osteonecrosis of the jaw in patients undergoing bisphosphonate treatment
- For post-menopausal women and men alendronate is currently the most commonly used, with risedronate and etidronate as alternatives

Oestrogen therapy (HRT)

- Increases risk of breast cancer and uterine cancer (if progesterone not included)
- Helps to decrease bone resorption and slows progression of osteoporosis but does not increase bone mass
- Best within 6 years of menopause
- Doubles the risk of DVT/PE

Selective oestrogen receptor modulators (e.g. raloxifene)

- Works like oestrogen to prevent bone loss but may increase menopausal symptoms
- Good evidence for protection against vertebral but not hip fractures

Teriparatide (recombinant PTH)

- Increases bone formation and improves microarchitecture
- Activates bone-lining cells and osteoblasts
- Reduces the likelihood of both vertebral and non-vertebral fractures

Paget's disease

Epidemiology

- Prevalence is 3–4% in the over 40-year-old age group, 10% in >90 years
- Most common in North America, England, Northern Europe and Australia
- Very rare in Scandinavia, Asia and Africa
- Family history in 15–25% of cases
- Polyostotic 83%, monostotic 17%

Pathology

- Increased osteoclast size and number leading to increased bone resorption, followed by compensatory increase in disorganized osteoblastic bone formation
- Accelerated but disorganized bone remodelling; a chaotic over-activity in bone
- Bone is rapidly laid down and also rapidly resorbed
- Bone is both enlarged and biomechanically weak

Bone is:

- Poor quality
- Very vascular
- Thickened and bent
- Weak
- Mosaic pattern with irregular areas of lamellar bone
- Erratic cement lines
- Marrow tends to become fibrous

Pathological phases (acronym: LAB)

- L – *Lytic* (osteolytic): A front of osteoclastic resorption is seen, usually near the metaphyseal region of a long bone or osteoporosis circumscription in the skull
- A – *Active* (mixed): Both osteoblastic resorption and osteoblastic bone formation occur in the same area of bone
- B – *Burnt-out* (sclerotic): A dense mosaic pattern of bone is seen

Laboratory

- Serum calcium usually normal
- Raised alkaline phosphatase (bone)
- Raised serum acid phosphatase
- Raised urinary hyroxyproline and collagen-derived cross-linked peptides (markers of collagen turnover)

Aetiology

- Precise cause unknown
- Probably a viral origin as Pagetic osteoclasts have been shown to contain mRNA from paramyxoviruses and canine distemper virus

Clinical features

- A: Arthritis
- B: Blood flow complications (high output cardiac failure)
- C: Cranial nerve compression
- D: Deformities (long bones, spine)
- P^3: Pain, pathological fracture, pseudarthrosis
- M^2: Metabolic abnormalities (hypercalcaemia), malignant change

Lesions detected on bone scan are usually painful, but many of those seen on x-ray are not. Bone pain is unrelated to activity and is worse at night. Acute pain is related to fractures. Severe pain should arouse suspicion of sarcomatous change.

Radiological features

Long bones

- Trabeculae of cancellous bone thickened, coarse, irregular and wide
- Cortex thickened, irregular and sclerotic
- Bones thick, bent and widened (lateral bowing of femur, anterior bowing of tibia)
- Candle flame-shaped lesions (arrow or flame sign) and V-shaped lytic defects in the diaphysis
- Loss of corticomedullary differentiation
- Involvement from one end of bone (proximal) along the shaft
- Stress fractures (convex side)

Skull

- Osteoporosis circumscription: Discrete areas of osteolysis (well-defined lytic lesions)
- Cotton-wool appearance: Mixed lytic and blastic pattern of thickened calvarium
- Diploic widening with inner and outer table involvement

Pelvis

- Acetabular protrusio

Spine

- Picture frame vertebral body: Enlarged, square vertebral body with thickened peripheral trabeculae and radiolucent inner portion
- Ivory vertebra (increased density)

Differential diagnosis

Other causes of increased and disorganized bone turnover with fibrosis, including:

- Osteitis fibrosa cystica (hyperparathyroidism)
- Fibrous dysplasia
- Osteoblastic secondaries
- Osteopetrosis
- Lymphoma

Management

Most patients require no active treatment.

Calcitonin

Lowers bone turnover by decreasing activity and number of osteoclasts

Must be given parenterally. When discontinued, bone activity levels quickly return to pre-treatment levels but relief of pain may persist for months

Bisphosphonates

- Slow down both the formation and dissolution of calcium hydroxyapatite
- Narrow therapeutic window between resorption inhibition and mineralization defect
- Early complication – Hypocalcaemia
- Delayed complications – Musculoskeletal pain, osteonecrosis of jaw, bisphosphonate fractures
- Nitrogen containing bisphosphonates usually used
- IV bisphosphonates, such as pamidronate or zoledronic acid more effective and last longer
- Oral bisphosphonates, such as alendronate or risedronate less well absorbed

Paget's in total hip arthroplasty

- Problems with hypercalcaemia and metabolic acidosis
- Problems with excessive intraoperative bleeding
- Problems with deformity of bone, including bowing of femur and acetabular protrusion
- Increased incidence of heterotopic calcification postoperatively
- Bisphosphonates given for 3 months preoperatively

Osteopetrosis – Marble bone disease or Albers–Schonberg disease

- A group of rare congenital diseases characterized by a marked increase in bone sclerosis
- Many types described – nine or more
- Impaired osteoclast function. Osteoclasts lack normal ruffled border and clear zone required for effective resorption
- Increased sclerosis and obliteration of medullary canal
- Marrow spaces filled with necrotic calcified cartilage
- Empty lacunae and plugging of Haversian canals

Genetics

Autosomal recessive

- Infantile malignant form
- Bone encroachment on marrow results in pancytopenia, anaemia, haemolysis and hepatomegaly
- Repeated infection or haemorrhage usually leads to death
- More benign and rarer condition – Carbonic anhydrase II deficiency – Associated with renal tubular deficiency. In this condition CO_2 is not converted to carbonic acid in osteoclasts, so osteoclasts cannot acidify the lacuna

Autosomal dominant

- Tarda – 'benign' form
- Patients survive to adult life
- Often asymptomatic
- Lifelong risk of fractures that heal poorly

Radiological features

- Increased bone density
- Cortices widened
- Narrow medullary canals
- Sandwich vertebrae: End plates densely sclerotic giving appearance of sandwich. Demarcation between cancellous and cortical bone is lost
- Do not confuse with ill-defined bands of sclerosis seen in the rugger jersey spine of HPT
- Skull thickened and base of skull densely sclerotic

Articular cartilage

This is a very popular topic. The usual format is to be asked to draw the ultrastructure of articular cartilage and explain the appearances in relation to function.

Function

- Shock absorption
- Provides low friction surface for joints (coefficient of friction 0.002 – 30 times better than the best artificial joint!)

Contents

The contents are shown in Table 30.3. The main components of the ECM are water, collagen and proteoglycans. Articular cartilage has:

- Few cells
- No blood supply (nutrition supplied via synovial fluid)
- No nerve supply
- No blood supply
- No lymphatics

Chondrocytes

- Comprise 1% of articular cartilage

Table 30.3 The make up of cartilage

Cells (chondrocytes) (5%)			
Extracellular matrix	Fibres	Collagen(10–20%)	Type II, IX, XI
			Type VI, X
		Elastin	
	Ground substance	Water	
		Proteoglycans and glycosaminoglycans (10–15%)	
		Glycoproteins	
		Degradative enzymes (matrix metalloproteinases)	

- Responsible for maintaining the ECM
- Ability to synthesize some proteoglycans decreases with age

Water

- Up to 80% of the ECM
- Permits deformation of the articular cartilage by the movement of water in and out of the cartilage and also within the cartilage
- Increased water content leads to increased permeability, decreased strength and decreased elasticity
- Water content decreases through the deeper layers
- Responsible for lubrication and nutrition

Collagen

- About 60% of dry weight
- Gives the articular cartilage its tensile stiffness
- Main type is type II (90%)
- Type VI helps chondrocytes adhere to the matrix
- Type XI constrains proteoglycan matrix
- Type X only found near calcified zone

Proteoglycans

Give the articular cartilage its compressive strength and elasticity

- Consist of a protein core and glycosaminoglycan chains with negatively charged carboxyl or sulphate group, resulting in long strings of negative charges that repel each other
- Negative charges hold water[9]
- Glycosaminoglycans in articular cartilage include hyaluronic acid, chondroitin sulphate, keratan sulphate
- The large aggregating proteoglycan molecules are called **aggrecans**, and have large numbers of chondroitin sulphate

and keratin sulphate charges attached to the core protein. This appearance looks rather like a 'test tube brush'
- Aggrecans are associated with hyaluronic acid (HA) by link proteins to form **proteoglycan aggregates**
- The compressive stiffness is largely directly proportional to the aggregate (proteoglycan) content
- Loss of cartilage integrity in arthritis is associated with impaired aggrecan function due either to proteolytic cleavage of the aggrecan core protein, which decreases aggrecan charge, or to cleavage of the HA, which decreases aggregate size

Structure

The histological structure can be divided into zones.

1. **Superficial (tangential) zone:** 10–20% of thickness
   - Thinnest articular cartilage zone
   - High concentration of collagen fibres arranged parallel to surface, forming a dense mat
   - Most superficial part of this layer contains no cells (lamina splendens)
   - Deep to the lamina splendens is a cellular layer with chondrocytes parallel to surface, flat shaped, high density, many cells 1–3 cells thick
   - Good resistance to shear forces
   - Proteoglycan at low concentration
   - Water at high concentration, can be squeezed out to help create lubrication
2. **Middle (transitional) zone:** 40–60% of thickness
   - Collagen fibres oblique
   - High concentration of proteoglycan
   - Cells round shape, random, oblique arrangement, progressively lower density, fewer cells
3. **Deep (radial) zone:** 30% of thickness
   - Collagen fibres vertically arranged (perpendicular to tidemark)
   - High concentration of proteoglycans
   - Cells spherical, in vertical columns
4. **Tidemark**
   - Resistant to shear
5. **Calcified zone**
   - Hydroxyapatite crystals anchor articular cartilage to subchondral bone
   - Forms a barrier to blood vessels supplying subchondral bone

The superficial, transitional and radial zones are only poorly differentiated on cross-section. The appearance is due to the cartilage being cross-sectioned in one plane. The three-dimensional structure shows arcades (*Arcades of Beninghoff*) of collagen that arch through the articular cartilage, giving rise to the appearance of the three zones when cross-sectioned.

Nutrition

Articular cartilage obtains its nutrition from the synovial fluid. Nutrients diffuse through the matrix. Intermittent loading and motion are essential to produce the flux of water required for this nutrition.

Biomechanics of articular cartilage (Table 30.4)

- Articular cartilage is a viscoelastic material. When loaded, water moves through the matrix. The speed of water movement depends on the internal friction caused by aggrecans in the matrix. For a short duration the loading strain is relatively low, but it increases substantially if the load is maintained (increasing by up to a factor of 10 if the load is maintained for up to 30 min)
- Under very high compressive load the internal friction is increased in the fluid due to compression of the macromolecules; this results in cartilage being stiffer under very high loads
- Deformation speed under maintained load is relatively high initially as water diffuses through the matrix (the fluid phase), but as the matrix becomes compressed the load is eventually taken by the solid matrix and the rate decreases (solid phase). The mechanical behaviour of the cartilage is, therefore, biphasic[10].

Table 30.4 Biochemical changes of articular cartilage: Ageing vs osteoarthritis

	Ageing	Osteoarthritis
Water content	Decreases	Increases then decreases
Synthetic activity	Decreases	Increases
Collagen	Unchanged	Breakdown of cartilage collagen network
PG content	Decreases	Decreases
PG synthesis	Decreases	Increases
PG degradation	Decreases	Increases
Keratan sulphate	Increases	Decreases
Chondroitin sulphate	Decreases	Increases
Hydroxyapatite	Increases	Decreases
Enzymes		Increased activity MMPs
Matrix subunit molecules		Increases
Chondrocyte size	Increases	
Chondrocyte number	Decreases	
Modulus of elasticity	Increases	Decreases

MMPs = metalloproteinases; PG = proteoglycan.

Cartilage repair and healing

Classification of cartilage degeneration (Jackson)

1. Softening
2. Fibrillation and fissuring
3. Partial-thickness loss, clefts and chondral flaps
4. Full-thickness loss with exposed bone

Osteoarthritis vs ageing

Table 30.4 above illustrates the changes that take place in articular cartilage with ageing and contrasts these changes with those that occur in osteoarthritis.

Acute trauma to articular cartilage[11]

Superficial laceration, not reaching tidemark

- Chondrocytes die, matrix disrupted
- Chondrocytes do not migrate to the site of injury
- Defect does not fill
- No adequate cellular response – Repair does not take place

Deep laceration, crossing the tidemark

- Haemorrhage and fibrin clot formation
- Growth factors released, attracting inflammatory cells and fibroblasts
- Fibrocartilaginous scar formation
- No organisation into 'zones'
- Poor loadbearing properties
- V-shaped defects more likely to form some hyaline-like cartilage
- May progress to osteoarthritis

Blunt trauma

- Chondrocyte death, matrix damage, fissuring of surface, injury to underlying bone
- Loss of proteoglycans and chondrocyte clumping
- Increase in subchondral bone stiffness
- Cartilage fibrillation, causing an increase in water content and softening

Treatment of cartilage defects

- Abrasion arthroplasty
- Microfracture
- Mosaicplasty
- Autologous chondrocyte implantation

Comparative results of these treatments have been inconclusive.

Tendons

Tendons are dense, regularly arranged collagenous structures that transmit loads generated by muscle to bone. Tendons enable muscles to act at a distance through confined spaces and they also enable muscles to work at varying angles. Tendons fall into two main groups: Those with a synovial covering running in tendon sheaths and those covered by paratenon. Some tendons arise from deep within the muscle, allowing a multipennate arrangement of muscle fibres; this increases the relative power of the muscle but at the expense of range of movement.

Composition and structure

- The main component of tendon is type I collagen, contributing up to 80% of dry weight
- Where tendons change direction they become subject to forces other than pure tensile ones and in these areas the composition may change, with some type II collagen and an increase in glycosaminoglycan content and formation of aggrecan proteoglycan. The arrangement of collagen fibres also becomes less parallel and more 'woven'
- Proteoglycan contributes up to 5% of the dry weight
- Proteoglycan molecules play a large part in maintaining the water content of tendons through their highly negatively charged glycosaminoglycan side chains
- The main constituent of tendon, when not considered as dry weight, is water, which contributes up 60% of the wet weight
- The collagen molecules generally form into a triple helix pattern, made up of three α chains
- The collagen fibrils combine together to form fibre bundles and collections of fibre bundles form fascicles. The fibrils are arranged in a closely packed parallel formation. Groups of fascicles form the tendon
- The collagen fibres also demonstrate *crimping*, a wavy appearance, which influences the mechanical behaviour of tendon material

Insertion into bone

Tendons may insert into bone by a fibrous insertion (typically found when the tendon inserts into the diaphyseal or metaphyseal region) or by a fibrocartilaginous insertion (typically where the tendon inserts into an apophysis or epiphyseal region).

In fibrocartilaginous insertions there are four transitional tissues/zones:

- **Zone 1:** Parallel collagen fibres at the end of the tendon
- **Zone 2:** Collagen fibres intermeshed with unmineralized fibrocartilage
- **Zone 3:** Mineralized fibrocartilage
- **Zone 4:** Cortical bone

These zones allow a gradual increase in the stiffness of the tissue, so there is less of a stress-concentrating effect at the insertion into bone, minimizing the risk of insertion site failure at these insertions, where the applied loads tend to be greater than in those of tendons inserting further away from the epiphysis.

Surrounding connective tissue

The fascicles within a tendon are surrounded by loose areolar tissue – The *endotenon,* which permits longitudinal movement between collagen fascicles. The endotenon is surrounded by the *epitenon*.

The tendon is surrounded either by a paratenon or by a synovial sheath.

Neurovascular supply

- The blood supply to tendons is derived primarily through the musculotendinous junction, with some further communication with the periosteal vessels at the insertion
- In those tendons with a paratenon, blood vessels penetrate the tendon throughout its length
- In those tendons with a synovial sheath, the outer and inner sheaths (parietal and visceral, respectively) are linked by a mesotenon, which transmits the vessels. The mesotenon may be continuous, or it may be confined to vinculae, as in the long flexors of the digits
- Further nutrition is derived from the synovial fluid, and this may be the major source of nutrition for some long tendons, such as the long flexors of the fingers
- The blood vessels form a network in the epitenon and then pass between fascicles in the endotenon
- The nerve supply is derived from the corresponding muscle, and tendons contain both fast and slow adapting sensory organs (Golgi organs, Pacinian corpuscles and Ruffini endings)

Mechanical behaviour

Tendons are viscoelastic structures and, like all viscoelastic structures, they display creep, hysteresis and stress relaxation. As a result of their viscoelastic behaviour, tendons not only transmit forces but are also capable of storing energy, which improves the efficiency of the muscle–tendon unit during repeated high impact activity.

The load–elongation curve for tendons is non-linear and can be divided into regions, as illustrated in Figure 30.6.

I **Non-linear region:** The tendon starts off relatively non-stiff and becomes progressively stiffer with increasing elongation. This 'toe' region probably reflects straightening out of the crimping of the collagen fibres

II **Linear region:** There is a linear relationship between increase in load and increase in length

III **Early sequential failure:** There can be small dips in the curve as failure of some stretched collagen fibres occurs

IV **Ultimate stress/strength:** The maximum load/stress before the ligament fails completely

Tendon healing[12]

Tendon healing generally follows three overlapping phases.

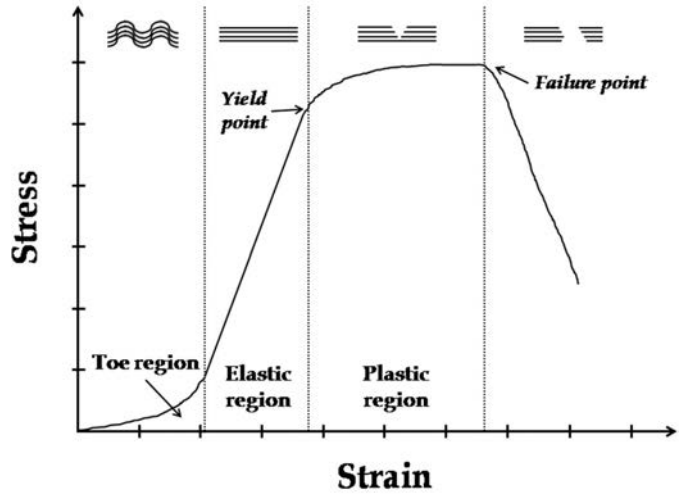

Figure 30.6 Load–elongation curve for tendon

Haemorrhagic/inflammatory phase

- Formation of haematoma
- Invasion by polymorphonuclear cells and monocytes/macrophages with release of cytokines and growth factors
- Debris removed and replaced with fibroblasts and capillary buds

Proliferative/fibroblastic phase

- Fibroblasts produce dense disorganized collagen laid down, bridging the gap between tendon ends to form tendon callus
- Early collagen is mainly type III
- This phase may commence within 3–5days of injury and continues for several weeks, usually peaking at about 4 weeks

Remodelling phase

- Collagen fibres reorganize to become orientated along the long axis of the tendon
- Type III collagen is replaced by type I
- Fibroblasts become tenocytes
- This phase may last many months or even years

The above sequence is found in tendons with a paratenon. In tendons with a synovial sheath there is controversy over the relative contributions from two healing processes:

- **Intrinsic:** Cell invasion occurs from the tendon ends and from the epitenon
- **Extrinsic:** Healing occurs via granulation tissue that invades via the tendon sheath. Extrinsic healing is more likely to produce adhesions and lead to a less satisfactory clinical outcome

Ligaments

Ligament structure is generally similar to that of tendons although there are some differences. Ligaments:

- Connect bone to bone
- Tend to be shorter and wider than tendons
- Most ligament-bone insertions are direct, with the collagen fibres running directly into bone, where they become anchored by bone that grows around them (Sharpey's fibres)
- Are mainly composed of type I collagen
- Have a higher elastin content than bone

Ligaments tend to be strongest when forces are applied parallel to their fibres and weakest when shear forces are applied at their insertions into bone.

Ligaments are viscoelastic[15] and display the viscoelastic properties of creep, stress relaxation and hysteresis. The load–elongation graph has a toe region, as for tendons. Beyond the ultimate stress there may be low resistance to elongation, resulting in a ligament that is intact but very lax.

Ligaments do not function homogeneously; they are composed of functional bands, which come under tension at different joint positions.

Sprains can be divided into three grades:

Grade I sprain – Partial tear disrupting at least one functional band. No clinically detectable instability

Grade II sprain – Sufficient disruption to cause clinically detectable instability

Grade III sprain – Complete rupture

Ligament healing follows a similar pattern to that for tendons.

Muscle

Structure

- The basic muscle cell, or fibre, which is surrounded by the basal lamina. Within the muscle cells there is a hierarchical arrangement of contractile elements
- Fundamental units are actin and myosin molecules, which are arranged linearly. The myosin 'ratchets' along the actin to achieve shortening, using energy from adenosine triphosphate (ATP)
- Troponin blocks the binding sites on the actin to limit contraction; calcium unblocks these sites
- The actin and myosin filaments form sarcomeres, which have a characteristic pattern on light microscopy
  - I band = actin (thin) filaments (lightest band on electron microscopy) where there is no overlap with myosin filaments
  - A band = myosin (thick) filaments
  - H band = myosin filament segments where there are no interdigitating actin filaments
  - M line in the middle of the A band where myosin filaments are joined together
  - Z line in the middle of the I band where actin filaments are joined together
- The arrangement of actin and myosin filaments is that of a hexagonal lattice in the centre of a sarcomere, i.e. each myosin filament is bounded by six actin filaments
- The sarcomeres are arranged end to end to form myofibrils
- Groups of myofibrils in parallel form the muscle fibres. The myofibrils are mechanically connected to each other by proteins, mainly desmin; the muscle fibres are surrounded by epimysium
- The muscle fibres, or cells, in turn are grouped into fascicles surrounded by a perimysium
- Fascicles are grouped into a muscle surrounded by an epimysium

Muscle contraction

- Muscle contraction is initiated by release of acetylcholine at the neuromuscular junction
- The acetylcholine diffuses into the synaptic clefts
- Muscle contraction is controlled by calcium, which is stored in, and controlled by, the sarcoplasmic reticulum
- Calcium is transmitted into the muscle via the transverse tubular system (T system)
- Calcium binds to troponin on the actin filaments, thus, releasing the actin filament and enabling it to interact with the myosin, resulting in contraction
- Contraction velocity of muscle is proportional to fibre length (i.e. number of sarcomeres)
- Maximal muscle power is proportional to the physiological cross-sectional area, which is proportional to the muscle mass and the surface pennation angle
- The relationship between length and muscle tension follows an approximate inverted U pattern
- The excursion of a muscle needs to be considered when choosing muscles for transfer

Muscle spindle

- Sensory structure within a muscle that regulates tension and acts as a proprioceptive organ
  - Primary afferent endings (annulospiral fibres), which respond mainly to the rate of change of length
  - Secondary afferent endings (flower spray fibres), sensitive to steady level tension

Fibre types

Histochemical classification

- **Slow oxidative:** Slow to fatigue, require oxygen for sustained activity, large concentration of myoglobin (red in colour), many mitochondria – For *endurance*
- **Fast oxidative and glycolytic:** Resist fatigue (white in colour), rich in mitochondria
- **Fast glycolytic:** High levels of ATPase, few mitochondria, anaerobic and quick to fatigue – For *sprinting*

ATPase stability classification

- **Type 1, 2A and 2B** fibres have been identified according to their ATPase response to varying pH. These three types are

often equated to slow oxidative, fast oxidative and glycolytic, and fast glycolytic, respectively, although this is probably not justified

Fibre types are not immutable and fibres can change their type in response to their mechanical environment.

Hill model – Biomechanically, muscle can be considered to have a force because of both its elasticity and its contractility. The passive stiffness of muscle is probably largely related to the protein titin and is not related to the length-tension curve for active contraction.

Types of muscle contraction

Isotonic (dynamic)

- Muscle tension is constant through the range of motion
- Muscle length changes (e.g. biceps curls)

Isometric (static)

- Muscle tension is generated but the length of the muscle remains unchanged, e.g. pushing against a wall

Isokinetic (dynamic)

- Muscle tension is generated as the muscle contracts at a constant velocity over a full range of motion

Concentric contraction

- Muscle shortens during the contraction

Eccentric contraction

- Muscle lengthens whilst contracting against an opposing force

Muscle-tendon junction

- Muscle and tendon fibres are almost parallel, which generates high shear forces
- A high degree of membrane folding generates a large surface area, reducing stress at the junction and reducing the angle of force vector. The net result is that the junction is very strong
- This area has a specific morphology, which is adapted to its function. Specific features include: Shorter sarcomere lengths, greater synthetic ability, greater number of organelles per cell, interdigitation of the cell membrane and intracellular connective tissue

Nerves

Anatomy

- **Cell body** – Site of metabolic activity, must be in continuity for regeneration
- **Axon** – Always carries impulse away from cell body; **dendrite** carries impulse towards it. Thus, sensory fibres are always dendrites and motor fibres are always axons!
- **Myelinated nerve fibre** – Axon/dendrite with associated **Schwann cell** and surrounding **endoneurium** (basement membrane)
- **Unmyelinated nerve fibre** – Single Schwann cell with several axons/dendrites embedded in it, called a Remak bundle
- **Perineurium** – Cellular layer round groups of fibres, creating fascicles
- **Epineurium** – Everything outside the perineurium that is not blood vessel or nerve; mostly collagen

Physiology

- Action potential results from Na^+ ions entering the cell and depolarizing the cell membrane
- Potassium ions leave cell
- After impulse the resting potential restored by the Na^+/K^+ exchange pump
- Myelinated conduction velocity is proportional to diameter
- Unmyelinated conduction velocity is proportional to the square root of the diameter
- Type A fibres: >2 mm in diameter, fast, motor, touch, pain
- Type B fibres: 3–15 mm in diameter, autonomic preganglion
- Type C fibres: 0.5–2.0 mm in diameter, chemonociceptors

Nerve injury (Seddon)

Neurapraxia

- Nerve contusion involving reversible conduction block without Wallerian degeneration
- Selective demyelination of the axon sheath

Axonotmesis

- Conduction block with axonal degeneration
- Axon and myelin sheath degenerate but endoneurial tubes remain intact

Neurotmesis

- All layers of nerve disrupted and there is Wallerian degeneration
- No recovery without repair
- 1 mm/day in adults after repair, 3–5 mm in children

Sunderland

- First degree – Same as neurapraxia
- Second degree – Same as axonotmesis
- Third degree – Axonal injury associated with damaged basal lamina and endoneurial damage (perineurium is intact); most variable degree of ultimate recovery
- Fourth degree – In continuity but, at the level of injury, is complete, scarring across the nerve preventing regeneration. Both perineurium and endoneurium are disrupted, continuing of nerve maintained by epineurium
- Fifth degree – Same as neurotmesis

Wallerian degeneration

- Axon and myelin degraded and removed by phagocytosis
- Existing Schwann cells proliferate
- Nerve cell body swells up and enlarges
- Rate of structural protein synthesis increases
- Wallerian degeneration will not occur in a sensory nerve if the nerve is still in continuity with the cell body, as is seen in pre-ganglionic injuries

Factors affecting nerve recovery

- Age – Noticeable change after age 30
- Level of injury – Distal repairs have more favourable prognosis than proximal ones
- Nature of injury – Sharp lacerations do better than crush or avulsion injuries
- Type of nerve – Pure motor or pure sensory do better than mixed nerves
- Delay before repair – 1% of neural function permanently lost for each week of delay beyond third week from injury
- Gap between nerve ends

Repair

- Epineural
- Fascicular – Repairs the perineural sheaths
- Group fascicular

Neuropathy

- **Acute**
  - Autoimmune – Guillain–Barré
- **Chronic**
  - Genetic
  - Metabolic (including vitamin B_{12} deficiency, diabetes)
  - Nutrition (alcoholism)
  - Amyloidosis
  - Neoplasia
  - Iatrogenic (phenytoin, bleomycin)

Neurophysiological tests

It is well worth visiting a neurophysiologist and observing how neurophysiological studies are performed.

Nerve conduction studies[14]

These use stimulating and recording electrodes and a ground electrode. Stimulation of a peripheral nerve generates:

- A nerve action potential (NAP) of 5–30 μV
- A compound muscle action potential (CMAP) of 5–10 μV (in response to supramaximal stimulus)
- The CMAP is measured by stimulating the nerve at one point with increasing current or voltage until no further increase in amplitude occurs. The process is the repeated at a more proximal point on the nerve. The difference in latency (the time from stimuation to first response) will give the conduction velocity. The amplitude is the voltage difference between the baseline and the peak upward deflection (negative is upward)
- The sensory nerve action potential (SNAP) can be measured *orthodromically* (stimulate distally and measure more proximally – I.e. in the direction the nerve normally conducts), or *antidromically* (stimulate proximally and measured more distally)
- A preserved SNAP implies that the dorsal root ganglion is in continuity; if motor function is reduced, pathology at the root or more proximally is implied
- After motor fibre transection the CMAP measured on stimulation distal to the injury will remain normal for several days as degeneration of the nerve takes time
- When some motor axons are lost but some intact the conduction velocity may be normal but the CMAP amplitude will fall (after a few days)
- Chronic axonal loss, as in neuropathies, leads to CMAP becoming more dispersed due to immature regenerating fibres, which conduct more slowly
- In standard conduction velocity tests only the fastest 20% of fibres are measured
- Note that because the SNAP is measured using the fastest 20% of fibres it is possible to have a normal SNAP even though the patient has a small fibres sensory neuropathy
- The following properties can be investigated
  - **F response** – When a motor nerve is stimulated there will be a distal impulse that causes the CMAP and a proximal impulse that reaches the anterior horn cells and causes depolarisation followed by a 'backfire' impulse that causes a second small muscle depolarisation – The F wave. This may be abnormal immediately after nerve root injury. Detects proximal nerve lesions early
  - **H reflex** – Equivalent to tendon reflex; monosynaptic reflex; absent in radiculopathies and polyneuropathies
  - **Latency** – Time between onset of stimulus and response
  - **Amplitude** – Size of response or 'evoked potential' (from baseline to peak upward deflection). Usually an average recording to reduce background 'noise'. Normal for median and ulnar nerve sensory >5 μV, and for motor >5 mV
  - **Nerve conduction velocity** – Distance between stimulating and recording electrodes divided by time; may be slowed by demyelination or focal entrapment. 'Normal' motor and sensory velocities for median and ulnar nerve >50 m/s. Values differ with age; the 'normal' motor conduction slows by 0.4–1.7 m/s per decade and 'normal' sensory velocity slows by 2–4 m/s per decade. Nerve conduction study is also influenced by the temperature of the limb

Electromyography

Two needle electrodes are placed in the muscle to be studied:

- Electrical activity in response to voluntary contraction
- Characteristic recruitment of motor units with increased force in the muscle
- Normal: No muscle activity at rest
- Immediately after section EMG in supplied muscle is normal
- Between 5 and 14 days: Positive sharp waves consistent with denervation
- Between 15 and 30 days: Denervation, fibrillation potentials present
- Evidence of re-innervation: Highly polyphasic motor unit potentials

Somatosensory evoked potentials

- Stimulation of a peripheral nerve (median or posterior tibial is standard)
- Electrical recordings at scalp electrodes are very small, therefore averaged over 100 or 200 stimulations
- Used for intraoperative monitoring of cord function
- Not absolute

Symptoms

- **Allodynia** – Painful response to a normally painless stimulus
- **Causalgia** – Burning pain extending beyond a nerve territory (sympathetic involvement)
- **Dysaesthesia** – Spontaneous unpleasant sensation
- **Hyperalgesia** – Increased level of pain to a normally painful stimulus
- **Paraesthesia** – Spontaneous abnormal sensation
- **Post-traumatic neuralgia** – Pain within a nerve territory

The meniscus of the knee

This is another favourite topic. You should be able to relate the anatomical structure of the meniscus to its function.

Structure and composition

- The meniscus is a fibrocartilaginous structure consisting of cells and ECM
- There are three types of cells: Fibrochondrocytes (mainly in middle and inner parts), fibroblast-like cells (mainly outer half) and superficial zone cells (at the surface)
- The ECM consists of water (approximately 70% of total weight), collagen, proteoglycans and non-collagen proteins
- The collagen within the meniscus is mainly type I (90%)
- Proteoglycans have glycosaminoglycans attached (chondroitin sulphate, keratan sulphate and dermatan sulphate), which bind to water. The concentration of proteoglycans in the meniscus is less than in articular cartilage
- Non-collagenous proteins – E.g. link protein and fibronectin
- Collagen fibres are mainly arranged circumferentially, with other fibres arranged radially

Vascular supply

- Mainly from the medial and lateral genicular arteries – In the adult blood vessels penetrate the outer 10–30% of the meniscus (slightly less penetration in the medial meniscus)

Function[15,16]

- **Loadbearing**
  - The meniscus is *anisotropic*
  - Compressive forces are resisted by hoop stresses in the circumferential collagen fibres
  - For hoop stresses to be generated the 'hoop' must be complete, i.e. the attachments to the bony structures and the circumference of the meniscus must be intact – The anterior and posterior horn attachments are particularly important
  - 50% of compressive loads through the medial compartment pass through the medial meniscus
  - 70% of compressive loads through the lateral compartment pass through the lateral meniscus
  - A greater proportion of the compressive load is taken by the meniscus during flexion of the knee
  - Shear forces within the meniscus are resisted by the radial collagen fibres ('*ties*')
  - The collagen and glycosaminoglycan network resists movement of water through the 'solid phase' of the meniscal tissue
  - Meniscal tissue is less stiff than articular cartilage; this is because of the lower concentration of proteoglycans
  - Meniscal tissue is more resistant to the internal movement of water through its tissue than articular cartilage
- **Shock absorption**
  - The presence of intact menisci reduces the peak forces on the articular cartilage and underlying bone from impacts by approximately 20%
- **Stabilisation**
  - The shape of the meniscus contributes to the stability of the knee
  - Loss of the meniscus leads to an increase in AP movement between the articular surfaces
- **Lubrication and nutrition**
  - The increased conformity of the surfaces contributes to nutrition and probably also to the lubrication of the joint

The intervertebral disc

Structure

- **Outer annulus fibrosus** – Type I collagen fibres arranged obliquely in lamellae that insert into the adjacent vertebral bodies via Sharpey's fibres. The collagen fibres in adjacent lamellae lie perpendicular to each other
- **Inner annulus fibrosus** – Type II collagen, less structured than outer annulus
- **Nucleus pulposus** – Relatively high proportion of proteoglycans and low proportion of collagen compared with annulus fibrosis (particularly outer annulus). In young patients it is a hydrated gel
- Collagen in disc has high proportion of cross-links
- There is evidence that in some people there is a genetic predisposition to back pain due to a fault in the synthesis of Collagen 9 caused by a defective *COL9A3* gene on chromosome 20

Function

- The intervertebral disc forms part of the functional spinal unit: Disc, facet joints, vertebral end plates and ligaments
- The intervertebral disc and the adjacent endplates should be considered together – On loading there is deformation of the endplates
- The disc must be able to resist compression, bending, shear and torsional (rotational shear) forces
- Compressive forces on the disc are greater when sitting than when standing
- Proteoglycans resist compressive forces (resulting from body weight above disc and action of paraspinal muscles)
- Collagen fibres resist tensile forces
- Under compression the nucleus pulposus resists the force by converting it into radial forces, which are resisted by circumferential hoop stresses in the annulus fibrosus
- Twisting and tensile forces are resisted by the oblique arrangement of the collagen fibres in the lamellae of the annulus fibrosus
- Without the pressure within the nucleus pulposus the annulus fibrosus can buckle, impairing its mechanical properties
- Disc is less stiff at low loads than at high loads
- Hysteresis decreases with repeated loading, thus, reducing ability to withstand further load cycles

Nutrition[17]

- Nutrition is by diffusion through the avascular disc material from the vascular plexus around the annulus fibrosus and cartilaginous end plates
- Nutrition may be affected by factors that interfere with the vascular plexus, e.g. smoking

Changes with ageing

- Proteoglycan content decreases (reducing resistance to compression)
- Water content decreases
- Uniformity of nucleus pulposus decreases – Fibrous areas and softer areas
- Load distribution becomes uneven
- Composition of nucleus pulposus approaches that of the inner annulus and the junction between the two becomes indistinct
- Collagen content increases
- Ability to withstand loads diminishes

Disc injury

- Under direct compression the normal disc is stronger than the end plates
- The normal disc is not damaged by pure compression
- Disc protrusion occurs when the degenerative young disc is subjected to bending with compression

Osteoarthritis/osteoarthrosis

Arthrosis is the preferred term since there is no inflammation at the onset of the disease.

Classification

- Primary – No cause identified
- Secondary – Rheumatoid arthritis and other inflammatory arthritides, trauma, etc

Aetiology

- Genetic[18] – Heberden's nodes are strongly heritable
- Developmental – DDH, Perthe's, SUFE, etc
- Mechanical factors – Microtrauma and macrotrauma
- Metabolic – Raised uric acid, diabetes, etc
- Hormonal (acromegaly)
- Occupation
- Obesity
- Age – Early; 45 years is the peak for IP joint and CMC joint of thumb, later for hip and knee
- Polyarthrosis (primary) is more common in females

Pathology[19,20]

- Degenerative process in hyaline cartilage starts at the surface and eventually results in exposure of bone, the bone eventually becoming polished (eburnated)
- There is controversy regarding the primary event; theories include altered proteoglycans within articular cartilage, impaired subchondral venous drainage and altered synovial biochemistry

- Osteoarthrosis is thought to be a failed attempt by chondrocytes to repair damaged articular cartilage; an imbalance of wear and repair
- Chondrocytes attempt to compensate by increasing their rate of synthesis
- The earliest features are fibrillation of articular cartilage in superficial and transitional zones, penetration of tidemark by blood vessels from subchondral bone and subchondral bone remodelling
- Subchondral bone cysts and peripheral osteophytes form

Changes in articular cartilage

Early changes

- Alterations in proteoglycans
- Decreased aggrecan concentration
- Increased water content
- The increased permeability to water within the matrix and decreased stiffness

Cellular repair response

- Chondrocyte proliferation
- Anabolic and catabolic activity
- Increased proteoglycan synthesis
- Simultaneous increase in degradation of matrix
- Degradation of type IX and type XI collagen
- Weakening of type II collagen network
- Increased levels of metalloproteinases (collagenase, gelatinase, stromelysin)
- Increased levels of IL-1 and IL-2
- Proteoglycan content decreases

Progressive loss of tissue

- Decreased anabolic response of chondrocytes
- Modulus of elasticity and strength decreased owing to increased water content
- Articular cartilage progressively lost

Subchondral bone changes include thickening of the subchondral bone by the laying down of new bone on existing trabeculae and the formation of bone cysts.

Rheumatoid arthritis[21]

Symmetrical, erosive, deforming, inflammatory polyarthropathy involving both small and large joints

Incidence

- Approximately 1% of population

Diagnostic criteria

Rheumatoid arthritis is defined by the presence of four of the seven diagnostic criteria established by the American College of Rheumatology:

Table 30.5 Radiographic features of osteoarthritis vs rheumatoid arthritis

Osteoarthritis	Rheumatoid arthritis
Loss of joint space	Loss of joint space
Osteophytes	No osteophytes
Subchondral cysts	Marginal erosions
Bony sclerosis	Osteoporosis
Deformity and mal-alignment	Deformity and mal-alignment
Loose bodies	Loose bodies uncommon
Asymmetrical	Symmetrical
Normal soft tissue	Soft-tissue swelling

- Morning stiffness >1 hour for >6 weeks
- Swelling of at least 3 joints for >6 weeks
- Involvement/swelling of wrist or hands for >6 weeks
- Bilateral symmetrical polyarthritis for >6 weeks
- Rheumatoid nodules
- Positive serum rheumatoid factor
- Radiographic changes typical of rheumatoid arthritis (periarticular erosions, osteopenia, etc). Table 30.5 demonstrates the key differences between osteoarthritis and rheumatoid arthritis

Rheumatoid factor

Positive in 80%. Rheumatoid factor has significant false-positive and false-negative rates and is not diagnostic for the disease, but a positive rheumatoid factor is associated with a more severe disease course.

Aetiology

- The aetiology is still unclear; it is thought to be a disordered immune response that causes an inflammatory response against soft tissues, cartilage and bone involving antigen-presenting cells, T-helper cells[22], natural killer cells and plasma cells
- Autoimmune mediators of tissue destruction: Macrophages, lymphocytes and plasma cells
- Environmental trigger is superimposed on a genetic predisposition (HLA DW4, HLA DR4)

Staging of rheumatoid disease

Early

Acute or subacute synovitis without destruction of soft tissues or articular cartilage.

Intermediate

Involvement of synovial-lined tendon sheaths impairs tendon excursion and may lead to rupture. Erosions appear in articular surfaces.

Late

Destruction and deformity of joints, etc.

Radiological classifications

I. Osteopenia and soft-tissue swelling
II. Marginal erosions and very slight narrowing of joint space
III. Marked narrowing of joint space
IV. Punched-out erosions through subchondral plate
V. Normal anatomical contours of the articular surface are destroyed

General characteristics

Insidious onset of morning stiffness, joint pain (polyarthritis), symmetrical swelling of the peripheral joints, hands and feet involved early.

Extra-articular systemic manifestations

- Rheumatoid nodules
- Vasculitis
- Ocular inflammation
- Amyloidosis
- Nephropathy and renal failure
- Cardiac (pericarditis, myocarditis, conduction defects, aortitis)
- Respiratory (pneumonitis, pleurisy, interstitial fibrosis)
- Myositis and muscle atrophy
- Neuropathy
- Anaemia (normochromic and microcytic)
- GIT (salivary problems and peptic ulceration)
- Cerebral complications
- Felty's syndrome (splenomegaly, leukopenia, lymphadenopathy, anaemia, skin pigmentation, weight loss)
- Sjögren's syndrome (conjunctival dryness or Sicca syndrome)

Atypical presentations

- Explosive arthritis
- Monarticular arthropathy (chronic pain and swelling)
- Isolated second MTP joint swelling

Differential diagnosis

- Seronegative arthropathy (psoriatic arthritis, ankylosing spondylitis, Reiter's disease)
- SLE
- Polyarticular gout
- Calcium pyrophosphate deposition disease
- Sarcoidosis
- Polymyalgia rheumatica

Principles of management

- Control synovitis and pain
  - Rest, splintage, non-specific drugs, specific disease-altering drugs and synovectomy
- Modify the disease progression
  - Drugs
- Maintain joint function
  - Drugs, physiotherapy and sometimes surgery
- Prevent deformity
  - Physiotherapy and splintage, tendon reconstruction and soft-tissue stabilisation surgery
- Reconstruction
  - Excision arthroplasty and joint replacement

Ankylosing spondylitis

Background

One of the seronegative spondyloarthropathies. A generalized chronic inflammatory condition with a predilection for the sacroiliac joints and spine. Strong familial tendency. Cause unknown but 90% of patients are HLA B27-positive, as are half of their first-degree relatives (but HLA B27 is *not* diagnostic and there is a high false-positive rate). More common in males than females with estimates of ratios ranging from 2 : 1 to 10 : 1.

Clinical features

- Insidious onset low back pain and stiffness in an adolescent or young adult reoccurring at intervals
- Progressive spinal flexion deformities
- Early – Little to find on clinical examination apart from slight loss of lumbar lordosis, limitation of spinal extension and sacroiliac joint tenderness
- Late – Characteristic posture with loss of the normal lumbar lordosis, thoracic kyphosis, chin on chest deformity, flexed hips and knees
- Inability to perform the wall test
- Entire spine is ankylosed
- Limited chest expansion
- Peripheral joint involvement; usually the hips
- Pronounced morning stiffness
- Protrusion acetabuli
- Heterotopic bone formation
- Whiskering enthesis

Differs from rheumatoid arthritis in that the disease is asymmetrical and affects large joints more than small joints.

Atypical presentation in 10% of cases. The disease can start with an asymmetrical inflammatory arthritis, usually of the hip, knee or ankle.

Extraskeletal manifestations (eyes, heart, lungs, gastrointestinal, etc)

- Heart disease (carditis, aortic valve disease)
- Pulmonary fibrosis, osteoporosis, uveitis, colitis, arachnoiditis, amyloidosis
- Poor outcome if there is pulmonary involvement, hip involvement or young age at onset of the disease

Radiographic features in the spine

- Earliest vertebral change is flattening of the normal anterior concavity of the vertebral body (squaring due to ossification of the anterior longitudinal ligament)
- Erosion and fuzziness of the sacroiliac joints occur and then later sclerosis, especially on the iliac side of the joint, and finally bony ankylosis and obliteration of the sacroiliac joint
- Ankylosis of sacroiliac joints is followed by ossification of the interspinous and interlaminar ligaments, ankylosis of the facet joints, ossification of the annulus fibrosus and syndesmophyte formation. The features proceed in a cranial direction and may produce a characteristic appearance – Bamboo spine

Radiographic differential diagnosis of the sacroiliac joint lesions

- Reiter's disease
- Psoriatic arthritis
- Ulcerative colitis
- Crohn's disease

Differential diagnosis

- Mechanical disorders
- Ankylosing hyperostosis (Forestier's disease) – A common disorder in older men with widespread ossification of ligaments and tendons. Superficial resemblance to ankylosing spondylitis but not an inflammatory condition, the spinal pain and stiffness are rarely severe and blood tests are normal
- Other seronegative spondyloarthritides

Pathology

Preferential involvement of tendon and ligament insertions. Inflammatory and erosive destruction of:

1. Diarthrodial joints
   - Sacroiliac joints, vertebral facet joints, costovertebral joints (chest pains aggravated by breathing indicate involvement of costovertebral joints)
2. Fibro-osseous junctions, syndesmotic joints and tendons
   - Affecting intervertebral discs, symphysis pubis, sacroiliac ligament, manubriosternal joint and bony insertions of large tendons

Pathological changes proceed in three stages:

1. Inflammatory reaction with round cell infiltration, granulation tissue and destruction of bone
2. Replacement of the granulation tissue with fibrous tissue
3. Ossification of the fibrous tissue leading to ankylosis of joints

Psoriatic arthritis

Definition

Seronegative polysynovitis with an erosive, destructive arthritis and a significant incidence of sacroiliitis and spondylitis.

Clinical features

Mild asymmetrical polyarthritis affecting some of the IP joints of the fingers or toes. Sacroiliitis and spondylitis are seen in about one-third of patients and are similar to those in ankylosing spondylitis. Affects up to 10% of patients with psoriasis. HLA B27-positive in 50% of cases (other loci also involved).

Diagnosis

The main differential is from psoriasis with seronegative rheumatoid arthritis.

Important characteristic features:

- Asymmetrical joint distribution
- Involvement of distal finger joints
- Presence of sacroiliitis and spondylitis
- Absence of rheumatoid nodules
- Nail pitting, fragmentation
- Sausage digits
- 'Pencil-in-cup' deformity (the distal end of the middle phalanx is the pencil in the cup of the distal phalanx)
- Rheumatoid factor usually negative

Systemic lupus erythematosus (SLE)

A chronic inflammatory disease of unknown aetiology associated with multisystem involvement.

Pathogenesis

Distension of soft tissues rather than direct destruction or fibrosis of supporting elements.

Clinical features

SLE arthritis affects >75% of patients with SLE although this is often overshadowed by systemic symptoms. Typically the arthritis is not as destructive as rheumatoid arthritis. Mainly occurs in young females.

- Fever
- Butterfly malar rash across cheeks and bridge of nose
- Pancytopenia
- Pericarditis
- Nephritis

- Raynaud's phenomenon
- Peripheral vasculitis
- Splenomegaly
- Polyarthritis

Laboratory tests

- Anaemia, leukopenia, elevated ESR, autoantibodies positive

Gout

Definition

A disorder of nucleic acid metabolism causing hyperuricaemia, which leads to monosodium urate crystal deposition in joints and recurrent attacks of synovitis.

Pathology

- Humans lack the enzyme uricase, which is involved in the elimination of excess nucleic acid purines and nitrogenous waste products through the production and excretion of allantoic acid; hence, in humans uric acid is the end-product of purine degradation
- Characterized by the presence of crystals in and around joints, tendons and bursae
- Crystals activate macrophages, platelets, phagocytosis and the complement system
- Release of inflammatory mediators into the joint
- Cartilage erosion and periarticular cyst formation secondary to deposition of monosodium urate
- Recurrent attacks of arthritis, usually in men aged 40–60 years, often in great toe
- Crystals deposited as tophi (ear, eyelid, olecranon, Achilles tendon)

Clinical

Two types described:

1. Primary (95%) – Inherited. Overproduction or underexcretion of uric acid
2. Secondary (5%) – Resulting from acquired conditions that cause either overproduction or underexcretion of uric acid (renal disease, multiple myeloma and polycythaemia)

The distinction may be somewhat arbitrary as people with a susceptibility to gout may develop the condition only after secondary precipitating factors are introduced, such as diuretic treatment, excessive alcohol intake, aspirin or localized trauma. Only a small proportion of people with hyperuricaemia develop gout.

Clinical presentation

Acute attack – Sudden onset of severe joint pain lasting for a week or two. Commonest sites great toe, elbow, finger joints and ankle. The joint is swollen and the overlying skin is shiny and red. Large joints not frequently involved. Spine very rarely affected

Chronic gout – Recurrent attacks merge into polyarticular gout. Joint erosion causes chronic pain, stiffness and deformity

Differential diagnosis

- Infection
- Reiter's disease
- Pseudogout
- Rheumatoid arthritis

Radiographs

Radiographic changes are a late feature and are usually associated with the chronic tophaceous stage:

- Well-circumscribed, punched-out periarticular cystic erosions with sclerotic overhanging borders. The size of the cysts is the differentiating feature from other arthritides; cysts larger than 5 mm are suggestive of gout
- Degenerative arthritis with joint-space narrowing, osteophyte formation and sclerosis

Diagnosis

- Elevated serum uric acid levels *not* diagnostic
- Diagnosis made by the demonstration of thin, tapered intracellular and extracellular needle-like crystals that are strongly negatively birefringent under polarized light microscopy

Osteonecrosis/avascular necrosis

Death of cells within bone as a result of transient or permanent ischaemia of the bone; either traumatic or non-traumatic.

Aetiology

Primary/idiopathic

- One-third of cases of AVN
- Young adults
- Usually bilateral
- Males > females

Secondary

- Trauma (e.g. subcapital femoral neck fracture or hip dislocation, etc)
- Sickle cell disease – Causes rapidly progressive femoral head disease
- Alcohol
- Steroids
- SLE
- HIV
- Caisson's disease and rapid decompression in divers
- Chronic liver disease
- Radiotherapy/radiation
- Chemotherapy

- Hyperlipidaemia
- Renal transplantation
- Gaucher's disease
- Haemophilia
- Pregnancy
- Smoking
- Endotoxins from bacteria (Shwartzman reaction leads to hypercoagulability)

Pathology

- Early stage – Within 2 weeks of precipitating event -necrosis of cells in marrow and bone – Empty lacunae
- Increased water content in marrow (visible on MRI scanning)
- Reactive hyperaemia and ingrowth of vascular tissue (reparative stage)
- Creeping substitution of cortical bone by cutting cones
- Osteoid laid down on dead trabeculae in cancellous bone

Ficat and Arlet radiographic staging for femoral head AVN

0. (Preclinical) – Normal radiographs and MRI scan
I. (Preradiographic) – Normal radiographs, early changes on MRI scan (increased T2 signal and decreased T1 signal, indicating increased marrow water content)
II. Radiographs show osteopenia/sclerosis, femoral head spherical, Tc and MRI scans positive
III. Radiographs show flattening of femoral head, crescent sign (necrosis of subchondral bone); Tc and MRI scans positive
IV. Radiographs show secondary degenerative changes; Tc and MRI scans positive

Symptom of pain in the groin starts in stage I. Technetium scan may be cold initially and then hot at about 2 months.

Core decompression, with or without vascular bone graft, may be useful in stage I.

Sickle cell disease

Inherited substitution of normal HbA with HbS (mutated chromosome 11). Homozygotes have disease, heterozygotes have trait. Common in malarial endemic areas owing to protective quality.

Pathology

Low O_2 tension causes polymerisation of HbS into longitudinal fibres, with deformity of erythrocytes, which then clump. Only manifests when HbF is lost, after 1 year of age. Reduced RBC lifespan from normal of 120 days to 20 days.

Clinical

Remember HBSS PAIN CRISIS:

H = Haemolysis

B = Bone marrow hyperplasia

S = Stroke

S = Skin ulcers

P = Pain

A = Anaemia

I = Infections

N = Nocturia

C = Congestive heart failure

R = Renal failure

I = Infarction of bone

S = Sequestration in spleen

I = Increased spontaneous abortion

S = sepsis.

- AVN occurs in up to 10%
- Osteomyelitis – Usually with *Staphylococcus aureus*

Vitamin C deficiency – Scurvy

- Characterized by haemorrhage secondary to capillary fragility
- Haemorrhage occurs in skin, gums, muscle attachments and, in children, subperiosteally
- Vitamin C deficiency leads to failure of collagen synthesis and repair and decreased osteoid formation
- Decreased chondroitin sulphate synthesis

Clinical features

- Fatigue
- Anaemia
- Bleeding gums
- Ecchymosis
- Intra-articular haemorrhages
- Poor wound healing

Radiology

- Generalized bone rarefaction, most marked in long bone metaphyses
- Thin cortices and trabeculae
- Metaphyses may be deformed or fractured
- Subperiosteal haematomas in children

Bleeding disorders

Haemophilia A

- 1 per 100 000 male births
- Lack of factor VIII

- X-linked recessive
- One-third are new mutations

Haemophilia B (Christmas disease)

- Lack of factor IX
- X-linked inheritance

Von Willebrand's disease

- Lack of factor VIII and cofactor
- Affects mucosae more than joints

Pathology

- Atraumatic joint haemorrhages cause synovial hypertrophy, synovitis and more bleeding
- Haemosiderin deposition in synovial villi
- Release of enzymes causes cartilage destruction
- Disuse osteoporosis
- Asymmetric physeal arrest in juvenile skeleton

Clinical

- Family history
- Haemarthroses in walking children
- Joint pathology during childhood, especially in weight-bearing joints
- Investigate with clotting screen and specific factor tests

Radiology

- Synovitis, distended capsule
- Thin cartilage
- Widened intercondylar notch on knee AP
- Enlarged ossification centres and widened epiphyses
- Flat femoral condyles
- Osteopenia

Biomaterials

The main groups of biomaterials encountered in orthopaedics are metals, ceramics, polymers and composites of these. Some knowledge of the different properties of these materials is important for their correct use, and questions on material properties are common in the exam.

Biomechanics of materials

It is important to have a clear understanding of the terms used to describe the mechanical properties of materials. The stress–strain graph for metal has been a long-term favourite examination topic although you should be prepared to demonstrate your understanding of what the graph means, not just be able to reproduce it rote fashion.

Definitions

- **Anisotropy** – The mechanical properties differ when loading occurs along different axes
- **Brittle** – A brittle material exhibits elastic behaviour up to the point of failure. The material undergoes little or no plastic deformation prior to failure. The yield stress equates to the failure stress
- **Compressive force** – A pushing force
- **Ductile** – A ductile material undergoes a large amount of permanent (plastic) deformation before complete failure
- **Elastic deformation** – Deformation of a material or object that reverses when the deforming force is removed
- **Endurance limit** – The maximum stress below which a material will not fail regardless of how many loading cycles are applied (by convention a figure of 10 million cycles is used for orthopaedic applications)
- **Fatigue failure** – Occurs as a result of repetitive applications of load at stresses below the ultimate stress and above the endurange limit
- **Hardness** – A surface property that describes a material's ability to resist scratching or indentation of the surface
- **Isotropic** – The mechanical properties are independent of the direction of loading
- **Plastic deformation** – Permanent deformation of a material or object that does not reverse when the deforming force is removed
- **Shear force** – A force that is applied parallel to the surface
- **Strain** – Change in length per unit length (no units or%), or angulation for shear strain
- **Stress** – Force per unit area (N/m^2 or MPa)
- **Stress riser** – A change in contour that increases the concentration of stresses, thus, increasing the risk of fatigue failure
- **Tensile force** – A pulling force applied to an object
- **Toughness** – Describes the amount of energy per unit volume absorbed by a material before breakage. There are several types of toughness – Impact toughness, notch toughness and **fracture toughness** – The latter two refer to the ability of a material that contains a flaw to resist propagation of a crack leading to a 'brittle fracture'. Fracture toughness is probably the most relevant to Orthopaedics as cracks tend to develop in materials over time
- **Fatigue toughness** – Describes the *work done* to failure under fatigue conditions

Deformation and the stress–strain curve

When a force is applied to an object made from a material it will deform. The stress–strain curve describes the pattern of deformation of a standardized sample of material.

An idealized stress–strain curve for metal is shown in Figure 30.7. You should be able to 'talk through' the various parts of this curve. In the first part of the curve there is a straight line, which represents the elastic phase of the material. For every incremental increase in stress there is a proportional increase in strain. The slope of the linear part of the plot is Young's modulus and it indicates the *stiffness* of the material

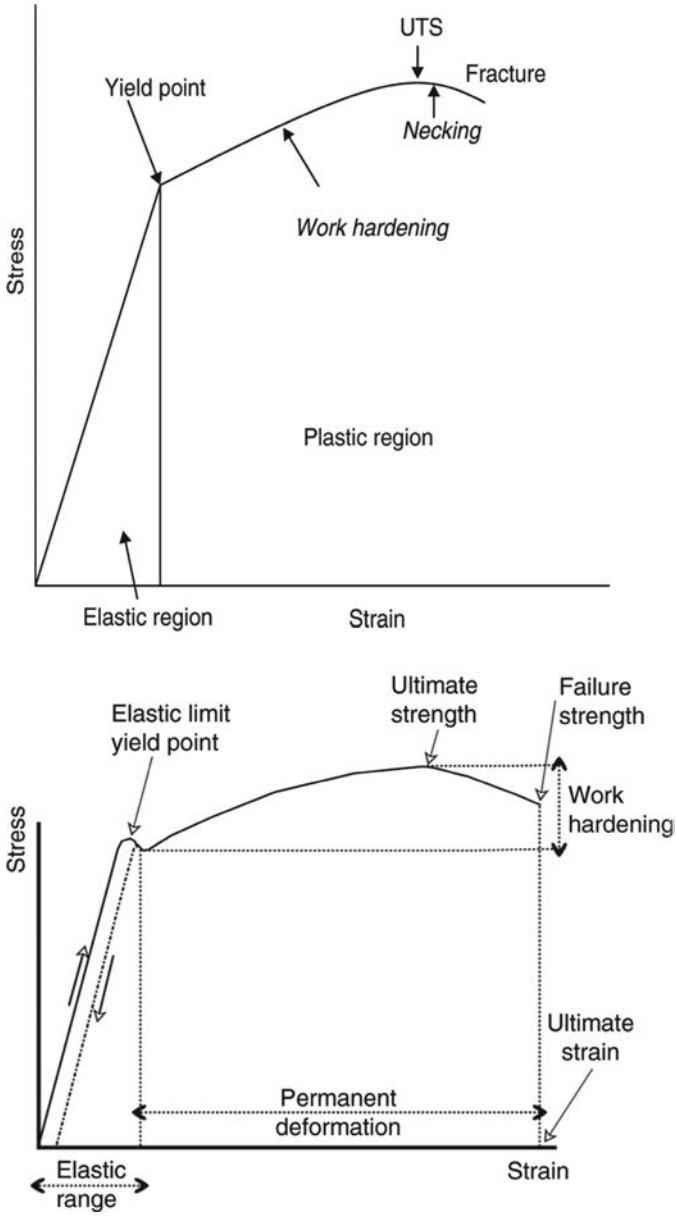

Figure 30.7 Idealized stress–strain curve for a metal. UTS = ultimate strength

(E = stress/strain). In the elastic phase the material will return to its original dimensions if the deforming force is removed. The *yield point* or elastic limit is that point at which the material starts to undergo permanent deformation and beyond this point the deformity will not completely recover if the force is removed. Unloading beyond the yield point will result in a linear plot parallel to the initial linear component of the plot but not returning to the intersection of the x and y axes. In the stress-strain curve for metal, the curve continues on an upward plot beyond the yield point and this represents *work hardening* of the material. The final downward slope before failure is a result of *necking* of the sample (the cross-section becomes smaller) prior to final failure. Fracture toughness is calculated from the area under the stress-strain curve and represents the energy to fracture.

The following are key terms that relate to the stress-strain curve:

Young's modulus – The slope of the linear portion of the graph. This indicates *stiffness*

Yield point – That point at which elastic behaviour changes to plastic, resulting in permanent deformation

Proportional limit – The limit of the linear relationship between stress and strain. Although often found at the same point as the yield point it does not have to be; some materials have a non-linear elastic part to the plot

Elastic limit – The same as the yield point

Ultimate strength – Indicated by the ultimate stress

Ultimate stress – The highest stress on the stress–strain curve

Breaking point – The point where the material fractures

Strain energy – The area beneath the elastic portion of the curve

Toughness – The area under the whole plot

Ultimate strain – The strain reached at the point where the material breaks

It is very important to understand that stiffness, strength, toughness, brittleness and hardness describe different and largely independent properties of materials, and that it is not possible to deduce one from the other.

Most stress–strain curves are drawn for tensile forces, but similar curves can also be drawn for compressive and shear forces so a material will have elastic moduli for tension, compression and shear. NB. Do not confuse this with anisotropy, which refers to the direction of loading along the x, y and z axes and not to the type of loading.

A brittle material will not have a plastic phase on the stress–strain curve. When brittle materials break, the opposing broken surfaces will still match if they are reassembled.

It should be noted that stress–strain plots are drawn for standardized samples of a material. For objects made from a material the plot should be termed a *load deformation plot*.

Viscoelastic materials

Viscoelastic materials display time and/or rate-dependent physical properties.

The mechanical properties of viscoelastic materials can be modelled as a spring (representing the elastic component) and a 'dashpot' or syringe, representing the viscous component.

All biological materials and most polymers encountered in orthopaedics are viscoelastic.

Many viscoelastic materials (including bone) become stiffer and stronger when they are loaded more rapidly.

Viscoelastic materials display some characteristic properties that are not seen in non-viscoelastic materials:

Creep – Deformation over time when under constant load (Figure 30.8). Creep has a more rapid initial phase, followed by a slower phase of deformation. At sufficient load levels, creep can eventually lead to a *creep fracture*

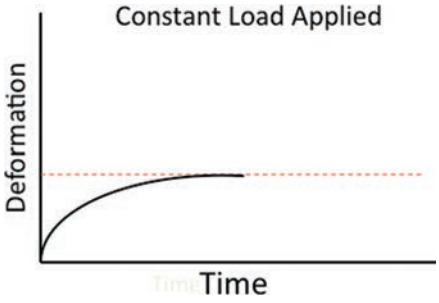

Figure 30.8 Creep

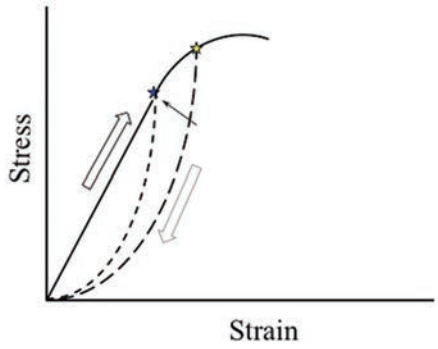

Figure 30.9 Hysteresis

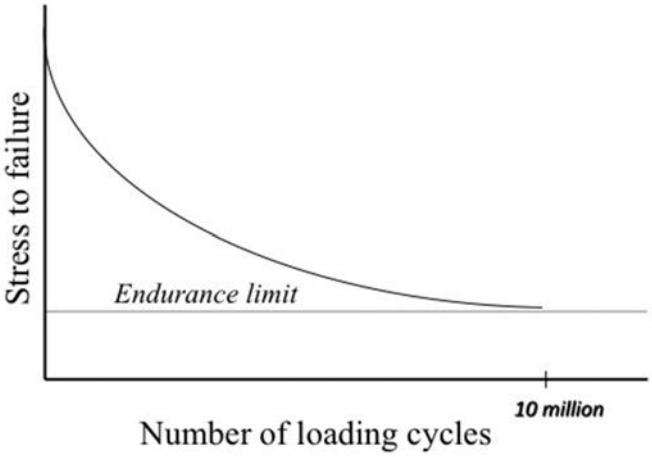

Figure 30.10 SN curve

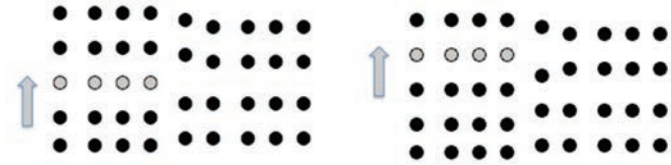

Figure 30.11 Dislocations in metal

Stress relaxation – Decreasing stress when held under constant strain

Hysteresis – When the stress-strain curve of the material to which a stress is applied and then removed from follows a different downward plot when in the elastic phase (Figure 30.9). The area between the loading and unloading curve represents energy lost, usually in the form of heat

The properties of a viscoelastic material cannot be described by a single stress-strain curve as there would be an infinite number of potential curves depending upon the rate and duration of loading.

Viscoelastic fluids (such as synovial fluid) are called non-Newtonian fluids.

Fatigue failure

The stress-strain curve shows the stress required to break the material on a single loading. If a material is put through repeated loading cycles, the stress required to cause failure becomes progressively smaller with increasing numbers of load cycles, and the relationship between stress to failure and load cycles is plotted on an *S–N curve*.

In many materials there is a stress below which the material could theoretically be loaded an infinite number of cycles without failure, and this is called the endurance limit (Figure 30.10).

Metals

Solid metals have a crystalline structure. The positive metal ions can be packed in different arrangements: Hexagonal close packed, face-centred cubic or body-centred cubic. The number of close contacts each metal has with neighbouring positive ions is the coordination number. Hexagonal close packed and face-centred cubic arrays have coordination numbers of 12, and body-centred cubic has a coordination number of 8. As molten metal cools, crystals start to grow. The crystals form grain boundaries where they meet other crystals. Dislocations are irregularities in the crystalline arrays. There may be millions of dislocations within a cubic millimetre of metal. The physical properties of the metal are greatly influenced by the grain size and the number of dislocations.

Metals are ductile; a large amount of plastic deformation occurs before failure. Ductility is the result both of slipping of the positive ions over each other to form new bonds with delocalized electrons, and also of movement of the dislocations through the lattice structure (Figure 30.11). Grain boundaries hinder the movement of dislocations.

The three most commonly used metals in orthopaedics are stainless steel, cobalt chrome alloy and titanium alloy.

Metal processing

Casting

Liquid metal is poured into a mould. Cooling does not occur completely uniformly and this can result in internal cracks and shrinkage voids.

Wrought

The cast material is modified by rolling and extending.

Cold working

The metal is forced into new shapes at room temperature by cold rolling, drawing or pressing (forcing onto a die or mould). During cold working, new dislocations are formed. Work hardening describes the increasing stiffness of metal when it is shaped and it is the result of increasing numbers of dislocations, which crowd up against the grain boundary, interfering with each other's movement through the material.

Annealing

The metal is heated to about half its melting point. The grain structure re-forms at the recrystallisation temperature as the metal cools and this reduces the number of dislocations, making the material more ductile again.

Hot working

The metal is heated above its recrystallisation temperature (usually to about 60% of melting temperature) and is then shaped whilst still hot. As annealing is occurring during the working, the metal does not become work hardened. Hot working is performed by rolling or by forging (the metal is hit by hammers or squeezed between a pair of dies).

Alloying

Small amounts of other elements are added to the pure metal to alter the physical properties. The addition of larger ions disrupts the regular metal lattice arrangement, making it more difficult for layers of the lattice to slip over each other; the material becomes less ductile. Smaller ions such as carbon and nitrogen fit into the holes in the lattice structure and also decrease the ability of the lattice layers to slip. The carbon content of steel greatly affects the stiffness and wear resistance. Adding small quantities of nickel and chromium increases corrosion resistance, and addition of other metals (such as molybdenum, cobalt, etc) affect strength and other properties. Steels with <4% chromium are called alloy steels. Stainless steel contains more than 4% chromium.

Quenching

The heated metal is suddenly immersed in cold water or oil. When alloyed metals are quenched, the alloying elements become trapped within the crystals, rather than being precipitated out, making the metal harder. The brittleness of a quenched metal can be reduced by tempering; the metal is heated to its tempering temperature (less than the recrystallizing temperature) and then re-quenched.

Passivation

An oxide layer is formed on the surface of the material to improve the mechanical properties and increase resistance to corrosion. If the passivating layer has the same volume as the underlying metal it will passivate. If the volume of the oxide is greater or less than that of the underying metal then the oxide layer will buckle or split respectively and passivation will not occur.

Types of metals

Stainless steel 316L

- The number 316 refers to 3% molybdenum and 16% nickel that is added to the alloy of iron, chromium and carbon
- The letter 'L' denotes low carbon (<0.03%)
- Strong and cheap
- Relatively easy to manufacture components
- Relatively ductile so easy to alter shape; useful in contouring of plates, etc during operative procedures
- Relatively biocompatible
- High Young's modulus of elasticity; can lead to stress shielding of lower modulus bone
- Usually cold worked by 30% to improve its yield and ultimate stress
- Good fatigue resistance
- Reasonably resistant to corrosion although susceptible to pitting, stress and crevice corrosion – *Stress corrosion cracking*

Cobalt chromium

- Chromium, cobalt and trace amounts of molybdenum, carbon and nickel
- Similar Young's modulus to stainless steel
- Very high ultimate strength and fatigue strength
- Good biocompatibility
- Better wear properties than stainless steel or titanium when used as an articular surface in joint replacement surgery
- Excellent resistance to corrosion, especially crevice corrosion

Titanium alloys

- Titanium 64 most commonly used (6% aluminium, 4% vanadium)
- Lower Young's modulus than stainless steel and cobalt chrome (approximately half), so less stress shielding of bone when used as implant, although Young's modulus still higher than cortical bone
- Forms a passivating oxide layer on surface so excellent resistance to corrosion
- Excellent resistance to pitting and intergranular corrosion
- Less interference with CT/MRI than stainless steel or cobalt chrome
- Poor hardness when used as articulating surface
- High coefficient of friction
- Notch-sensitive – Surface flaws or scratches predispose to fatigue failure
- Relatively expensive
- Low tensile strength

Ceramics

There does not appear to be a satisfactory comprehensive definition of what a ceramic is (the word ceramic comes from

the Greek for 'clay' or 'pottery'). Most ceramics are compounds of metallic and non-metallic elements.

Orthopaedic ceramics may be bioinert (alumina, zirconia) or bioactive (hydroxyapatite, glass ceramic).

Orthopaedic ceramics can be manufactured into implants by the process of sintering, in which the material in powder form is heated to a temperature below its melting point, and often subjected to high pressure.

Properties of bioinert ceramics

- Very hard materials with good wear resistance (wear particles very small)
- Surfaces can be made very smooth to give low coefficient of friction
- Excellent wettability
- Strong under compression
- Relatively weak under tension
- Stiff (high Young's modulus)
- Brittle (little or no plastic phase before failure)
- Low moisture absorption
- Biocompatible

Implants manufactured from ceramics have in the past been associated with susceptibility to fracture, resulting in many very sharp, hard and abrasive fragments. Although zirconia is tougher than alumina it can have poorer wear properties.

- The process of **transformational toughening,** with the introduction of small quantities of zirconia into alumina, produces a material that is much tougher and, therefore, less susceptible to fracture. Figures of 0.04% breakage for hip implants are now being reported
- **Hot isostatic pressing** has also led to improved mechanical properties
- Other methods of improving the Fracture Toughness of ceramics are being explored, often using composite materials

Bioactive ceramics

There is increasing interest in bioactive ceramics. These are of less interest as mechanical devices but are used for their ability to interact with the biological tissues. Hydroxyapatite and tricalcium sulphate are examples.

Bio glasses have the ability to allow ions to leach out of the material over time.

Polymers

Polymer = poly + mer (unit)

The most commonly used polymers in orthopaedics are ultra-high-molecular-weight polyethylene (UHMWPE) and polymethylmethacrylate.

UHMWPE

- Polyethylene is a long chain polymer formed of ethylene monomer molecules
- Made by low-pressure oxygen-catalysed addition polymerisation of ethylene (C_2H_4)
- Each molecule of UHMWPE can contain more than 200 000 units of ethylene
- UHMWPE has a molecular weight of 2×10^6–5×10^6
- When used as a bearing surface the material can undergo work hardening in the direction of movement, an advantage for bearings where the movement is predominantly in one direction, such as the knee

Factors affecting wear properties

- Manufacturing technique for the UHMWPE material
  - Molecular weight
  - Presence of calcium stearate (used as a stabiliser) can result in crystals that can cause weakness
  - Fusion defects can cause stress concentrations, leading to fatigue failure
- Production methods for the component
  - Machining produces sharp edges that can act as stress risers
  - Direct compression moulding and isostatic moulding produce components with more uniform UHMWPE
- The sterilisation method[23]
  - Oxidation during sterilisation can lead to chain scission and make the material susceptible to subsequent fatigue failure (delamination, typically commencing a few millimetres below the surface where shear forces are high)

Production methods

Ram extrusion – Powdered resin is forced through a die at high pressure with heat applied to form a block. Components are made from the block by machining. A disadvantage is the product's susceptibility to non-uniformity

Sheet compression moulding – The resin is heated and then cooled under pressure between two metal sheets. A disadvantage is the potential for pressure differences to affect consistency

Direct compression moulding – The material is directly moulded onto a metal backing or into a shaped mould. This method has been associated with good wear properties

Isostatic moulding – The resin is packed cold into a mould under vacuum. Heat and isostatic compression is applied. A uniform polymer is produced with reduced oxidative degradation

Sterilisation of polyethylene – Gamma irradiation of polyethylene causes some of the carbon-hydrogen bonds to break, producing free radicals. The free radicals can cause chain scission, breaking the long UHMWPE molecule chains

After chain scission the polyethylene molecules may undergo recombination to form the original long polymer molecules,

they may remain as shorter molecules with reduced wear properties, or cross-linking may occur, in which the free carbon atoms from one polyethylene molecule reattach to the carbon atoms of adjacent polyethylene molecules, resulting in side-to-side links between the molecules.

In the presence of oxygen the free radicals cause rapid oxidation of the material with resulting chain scission and impaired mechanical properties.

Gamma irradiation in the presence of oxygen has been found to lead to implants that are susceptible to fatigue failure (delamination). For this reason implant sterilisation by gamma irradiation is now carried out in an inert gas. Despite this, some free radicals may remain in the material, allowing subsequent oxidation to occur; implants should be used within a relatively short time interval to prevent degradation of the material (once implanted, the oxidation risk is reduced as synovial joint fluid has a relatively low oxygen content).

- Incorporation of antioxidants, such as vitamin E, may also reduce the effects of oxidation
- Post-production heat treatment (annealing) reduces the free radicals
- Sterilisation in ethylene oxide does not produce free radicals and cross-linking does not, therefore, occur; this will result in a polyethylene with different mechanical properties when compared with polyethylene sterilized by irradiation

Cross-linking caused by gamma irradiation in an oxygen-free environment improves the hardness of the material, thus, improving its performance as a bearing material. The amount of cross-linking increases with increased radiation dose. Highly cross-linking polyethylene results in:

- Increased surface hardness
- Improved wear characteristics
- A more brittle material
- A stiffer material
- Reduced ultimate tensile strength
- Reduced ability to undergo work hardening

The optimal amount of cross-linking has not yet been established, and may differ for different joints (the reduced work hardening may affect the knee more than the hip, which has multiaxial movement at the joint surfaces).

Composite materials

Oxidized zirconium (Oxinium®) is produced by diffusing thermally driven oxygen into the surface layers of metallic zirconium alloy to transform the surface layers into zirconium oxide, producing a surface with ceramic properties of hardness and smoothness.

Bone cement

Polymethylmethacrylate (PMMA) has the same chemical formula as Perspex® and Plexiglas®, but the formulation differs.

Mechanical properties

- Poor tensile strength (25 MPa)
- Moderate shear strength (40 MPa)
- Strong in compression (90 MPa)
- Brittle
- Notch-sensitive
- Young's modulus between that of cortical and cancellous bone (E = 2400 MPa)
- Young's modulus much less than orthopaedic metals
- Viscoelastic
  - Undergoes creep
  - Undergoes stress relaxation

Composition

Depending on the brand the liquid is added to the powder or vice versa.

- **Liquid monomer** – Supplied in glass vial, containing methylmethacrylate monomer; an **inhibitor/stabiliser** (usually hydroquinone) is usually added to prevent spontaneous polymerisation during storage and an **activator** (N, N-dimethyl-*p*-toluidine) is also added to promote the cold curing process and to offset the effect of hydroquinone once the reaction has begun
- **Powder polymer** – Contains polymer granules of PMMA, a polymerisation **initiator** (1% benzoyl peroxide) and a radio-opaque material (zirconium oxide or barium sulphate)

Polymerisation process

Carbon-to-carbon double bonds are broken down and new carbon single bonds are formed to give long-chain polymers that are largely linear and relatively free of cross-linking. The reaction is exothermic. The curing process has the following time periods, which are affected by humidity, temperature and rate of mixing:

- **Dough time** – Starts from the beginning of mixing and ends when the cement will not stick to an unpowdered surgical glove
- **Setting time** – The time from the beginning of mixing until the surface temperature is half maximum
- **Working time** – The difference between the dough time and the setting time

Factors affecting bone cement strength

- Storage temperature
- Moisture content
- Incorporation of antibiotics
- Incorporation of radio-opaque material
- Inclusion of air pockets
- Inclusion of blood or tissue
- Age after implantation
- Presence of stress risers (e.g. sharp edges on implant, increasing risk of fatigue failure)

Mode of action in arthroplasty

Bone cement acts as a *grout* and not as glue; there is no chemical bond between the PMMA and the bone or implant surface. In PMMA/bone interface strength depends upon the mechanical interface between the cement and the cancellous bone interstices.

In hip arthroplasty there are two main femoral component design concepts that rely on different implant/PMMA interface mechanisms:

- **Shape closed** prostheses (**complex cantilever** designs) rely on frictional shear forces between the femoral stem component surface and the bone cement
- **Force closed** design (the **polished double taper** design) relies on subsidence of the femoral stem component to cause radial compression of the cement, which is resisted by hoop stresses generated in the femoral cortex. In this design concept it is intended that the femoral component should be able to slide against the bone cement surface

Cementing techniques in hip replacements

The aim of cementing techniques is to consistently produce a homogeneous cement mantle of uniform mechanical properties with good interdigitation into the bone interstices.

First-generation

- Hand mixed cement
- Finger packing of cement
- No cement restrictors

Second-generation

- Femoral canal plug
- Pulsatile lavage to remove debris/fat, etc
- Cement gun to allow retrograde filling of femoral canal

Third-generation

- Cement porosity reduced during mixing by vacuum or centrifugation
- Femoral canal plug
- Pulsatile lavage
- Cement gun to allow retrograde filling
- Use of pressurisation both before and during implant insertion
- Spacers to ensure centralisation of implant

Tribology

- Tribology is the study of interacting surfaces in relative motion
- Derivation: *Tribos* (Greek), meaning rubbing

Friction

- Defined as the resistance to sliding motion between two bodies in contact

Figure 30.12 Asperities at the surface of materials

- The frictional force (F) is directly proportional to the applied load (W) across a bearing surface (applied normal to that surface)
- $F = \mu W$, where μ is the coefficient of friction of the combination of surface materials
- The force needed to start movement is greater than that required to maintain it, so there is both a dynamic coefficient (μ_d) and a static coefficient (μ_s); the dynamic coefficient is typically approximately 70% of the static coefficient
- In most situations the frictional force is independent of the apparent contact area
- Frictional wear is proportional to the sliding distance

Contact area

- The true contact area is between the asperities (bumps and peaks) on the surfaces, and will typically be <1% of the apparent surface area (Figure 30.12)
- As the force normal to the surface increases, the number of asperities in contact increases, explaining the relationship between force and friction
- The asperities deform in proportion to load and are inversely proportional to the surface hardness
- Bonds form at contact points and must be broken to initiate movement (hence $\mu_s > \mu_d$)

Lubrication

Lubrication mechanisms can be divided into two main groups:

1. **Boundary lubrication**
   - Occurs when the bearing surfaces are separated only by a boundary lubricant of molecular thickness
   - Involves the adsorption of a single monolayer of lubricant on each surface of the joint
   - In synovial joints the glycoprotein lubricin, found in synovial fluid, is believed to be the adsorbed molecule
2. **Fluid film lubrication**
   - The two bearing surfaces are separated by a fluid film whose minimum thickness exceeds the surface roughness (i.e. the heights of the asperities), thus, preventing asperity contact
   - As there is no asperity contact there is no wear
   - **Lambda value** λ = fluid thickness/composite surface roughness of the two bearing surfaces

In practice, in many situations the lubrication is mixed, consisting of a combination of boundary and fluid film.

Fluid film lubrication in artificial joints

Squeeze film

- Occurs when two bearing surfaces approach each other without a significant relative sliding motion
- Pressure builds up in the viscous fluid because it cannot instantaneously be squeezed out from the gap between the two surfaces
- As the fluid is forced out, the layer of lubricant becomes thinner and the joint surfaces eventually come into contact
- This mechanism is capable of carrying high loads for short lengths of time
- This mechanism of lubrication is probably active at initial contact during the gait cycle or during high-impact activities

Hydrodynamic

- Rigid bearing surfaces that are not parallel slide tangentially in relation to each other (note that a ball and socket joint requires a *clearance* between the two surfaces -the radii should not be identical)
- A converging wedge of fluid forms, and viscosity within the wedge produces a pressure within it that separates the two surfaces
- The fluid is *entrained* between the two surfaces
- Entrainment requires a relatively high sliding speed
- A rougher surface with higher asperities will require a thicker film to achieve fluid film lubrication
- The ideal lambda value is about 3. If λ exceeds 3, friction starts to increase again due to viscosity within the fluid film itself
- λ values between 1 and 3 generally lead to mixed lubrication
- May occur during relatively rapid movement such as during the swing phase of the gait cycle

Additional lubrication mechanisms that may act in the synovial joint

The precise mechanisms of lubrication in the synovial joint are not known. Synovial joints have a very low coefficient of friction (about 0.02), suggesting that they are at least partly lubricated by fluid film lubrication. There are several additional mechanisms that may be present in the synovial joint that are not found in artificial joints.

Elastohydrodynamic

Non-rigid bearing surfaces, such as those formed from articular cartilage, are able to deform under load and this can trap pressurized fluid and increase the surface area. Elastohydrodynamic lubrication can enhance both squeeze film and hydrodynamic lubrication.

Weeping lubrication

Because articular cartilage is fluid-filled, porous and permeable lubricant fluid can be squeezed from the surface of the articular cartilage when relative motion occurs.

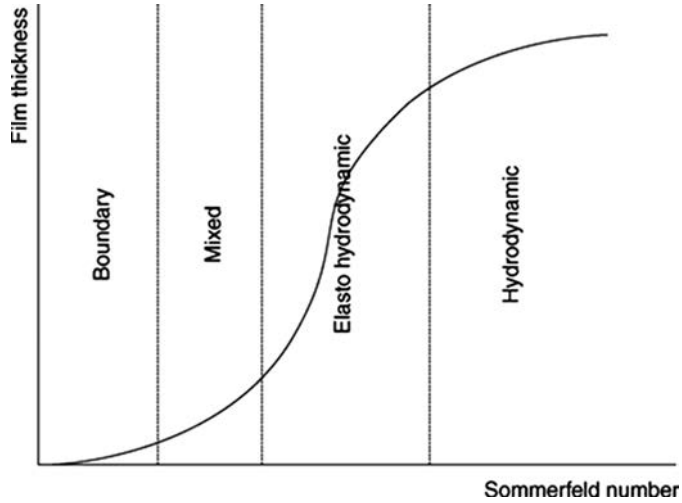

Figure 30.13 Lubrication mechanisms

Boosted lubrication

Under squeeze film conditions, water and synovial fluid may be pressurized into the articular cartilage, leaving behind a concentrated pool of hyaluronic acid protein complexes to lubricate the surface.

Figure 30.13 illustrates the differing mechanisms of lubrication that may occur between two biological materials.

Synovial fluid

- Produced by type B fibroblast-like cells of the synovium (type A cells are involved in phagocytosing debris)
- Made up of proteinase, collagenase, hyaluronic acid, lubricin and prostaglandins
- Is a dialysate of blood plasma without clotting factors or erythrocytes
- Has unique fluid properties conferred by the hyaluronic acid (it is a non-Newtonian fluid)
  - When the shear rate is varied, the shear stress does not vary in the same proportion (or even necessarily in the same direction)
  - Exhibits pseudoplasticity; a decrease in viscosity when the shear rate increases
  - Exhibits thixotropy; a time-dependent decrease in viscosity under constant shearing
- In conditions where hyaluronic acid is reduced (such as in rheumatoid arthritis or after joint replacement), the lubricating properties are impaired
- The **Sommerfeld number** is a property of a given lubricant = viscosity × velocity/stress and describes the relationship between the lubricant, the fluid film thickness and the potential types of lubrication

Wear

Lubrication and wear are closely related topics and questions on both frequently arise in the basic science oral.

Wear is the progressive loss of material from the surface of a body due to relative motion at that surface.

- Wear generates further 'third body' wear particles
- The softest material is worn

When thinking about wear it is important to distinguish between Modes, Mechanisms and Measurement.

Modes of wear in artificial implants

Mode describes between which parts of a component wear is taking place:

- **Mode 1** wear occurs between the two surfaces that are articulating together in the manner intended by the designer
- **Mode 2** wear occurs between a bearing surface and a non-bearing surface
- **Mode 3** wear occurs as a result of third body particles coming between the bearing surfaces
- **Mode 4** wear occurs between two non-bearing surfaces

Wear mechanisms

Wear is usually either mechanical or chemical. Mechanical wear mechanisms include abrasive, adhesive, fatigue and third body wear. Chemical mechanisms involve corrosion.

The surface roughness of the two materials influences the amount of mechanical wear.

Abrasive wear

- Asperities on the harder material come into contact with the softer material
- The harder material asperities plough and cut the softer surface, causing grooves and detached particles
- The detached particles become third bodies, causing further wear (see fretting below)
- When abrasion occurs to the surface of a ceramic the dull appearance is termed *scuffing*

Third body wear

- This is really a form of abrasive wear
- Particles become trapped between the articulating surfaces
- Very high local stresses produced
- Cause localized abrasive and fatigue wear

Adhesive wear

- The softer surface forms a bond with the harder surface
- The intermolecular bonds cause friction and if the junction is stronger than the cohesive strength of the softer bearing material fragments of the softer material become adherent to the harder material or become smeared
- Tends to cause steady low rate wear

Fatigue wear

- Repetitive/cyclical stressing of the asperities causes accumulation of microscopic damage
- Decreased by greater conformity of the bearing surfaces
- Decreased by thicker bearing surfaces (in UHMWPE) due to less concentration of shear stresses near the surface
- Mainly a problem in knee replacements where there are less conforming joint surfaces

Corrosive/oxidative wear[24]

Corrosion results from degradation to oxides, hydroxides and other compounds. Corrosion is frequently associated with mechanical stress. There are many different kinds of corrosive processes and in many cases several of these processes may be occurring simultaneously:

- **Galvanic corrosion**
  - Two different conducting materials are in contact and form an anode and a cathode
  - Galvanic corrosion may be seen in many different settings; such as crevice and pitting corrosion discussed below
  - As a metal cools during manufacture, impurities and additional trace metals crystallize differentially in different grains and this allows galvanic currents between the grains; this can lead to ***intergranular corrosion*** due to galvanic currents at the grain boundaries
  - Galvanic currents can also occur within the grains of the metal – ***Leaching corrosion***
  - **Inclusion corrosion** can occur as a result of impurites left on the surface of the material, such as from surgical implements
- **Crevice corrosion**
  - Occurs as a result of galvanic currents formed due to different oxygen tensions in the superficial and deep parts of a crack or defect
  - Lack of Oxygen in the depth of the crack prevents Passivation
  - Can be exacerbated by mechanical factors such as abrasion removing the passivating layer
- **Pitting corrosion** – Where the passivating layer is removed in very localized areas and joint fluid (containing saline) gains access the exposed area of metal alloy re-oxidises, with the release of Hydrogen ions from the water. This results in a very small anodic area and a large cathodic area causing a galvanic current. The positively charged hydrogen ions are balanced by negatively charged chloride ions to form hydrochloric acid, which can dissolve titanium and cobalt chrome

Wear processes

Two wear process are of particular importance in Orthopaedic applications, in particular in relation to joint replacement arthroplasty; fretting and galling.

Galling

- Is a particular type of adhesive wear

- Occurs as a result of friction and adhesion between two surfaces sliding on one another, typically described as relatively converging contact; this can occur during assembly of a Morse taper or due to micromovement between the two surfaces of a Morse taper
- Part of the softer material will be gouged out and may form lumps stuck to the surface, whilst other parts of the softer material will become stuck to the harder material (a form of adhesive wear). Some materials are more prone to galling than others

Fretting

- Caused by micromovement between two surfaces resulting in abrasion of asperities
- Amount of movement may be as little as a few nanometres
- The abrasion results in loss of the passivating surface
- Depending on the tolerances joint fluid may ingress between the two fretting surfaces
- Where the passivating layer is lost and joint fluid gains ingress corrosion occurs due to galvanic currents causing pitting corrosion
- The loose particles caused by the abrasion of the surface become oxidised. Oxides of most metals are much harder than the non-oxidised metal
- Large quantities of wear debris can be produced which can then migrate between the bearing surfaces, causing third body wear
- Third body wear also occurs between the fretting surfaces, increasing the rate of fretting and causing '*false brinelling*'
- Fretting wear at Morse junctions has been implicated in adverse reactions to metal debris

Two further processes are of relevance in the manufacture of implants'

Polishing refers to the use of a fine abrasive glued to a work wheel.

Buffing refers to the use of a fine abrasive powder applied to a work wheel. Buffing results in a higher polished finish than polishing.

Measurement of wear

Linear wear and volumetric wear are two methods of measuring wear in an implant. The mechanisms of wear are the same in both.

Volumetric wear measures the volume of material lost (described as cubic millimetres per year or per million cycles)

Linear wear measures the penetration of one component into the other and is measured on a radiograph.

- For any given amount of linear wear in a hip replacement, the proportional volumetric wear will be greater for a large femoral head component than for a smaller one for geometric reasons
- Linear wear measurements cannot distinguish between true wear and creep

Laws of wear

The volume of material (V) removed by wear increases with load (L) and with sliding distance (X) but decreases as the hardness of the softer material (H) increases:

$$V \propto LX/H$$

Wear is also proportional to surface roughness:

$$V = kLX$$

Where k = a wear factor for a given combination of materials that incorporates the hardness of the softer material, material properties (stiffness, wear resistance), local environment (lubrication) and surface roughness.

Bearing couples in hip replacements

The four main bearing couples currently in use are metal/UHMWPE, ceramic/UHMWPE, metal on metal and ceramic on ceramic. The first two can be described as hard on soft and the latter two as hard on hard.

- Hard on soft bearing couples can, at best, achieve mixed lubrication, but some surface asperity interaction cannot be avoided
- Hard on hard bearing couples can potentially achieve true fluid film lubrication, at least during some parts of the gait cycle
- Both ceramic on ceramic and metal on metal bearing couples can theoretically achieve fluid film lubrication
- Metal on metal bearings are vulnerable to scratching
- Ceramic on ceramic bearings are more scratch-resistant and smoother than metal and ceramic also has high wettability
- If the lubrication mechanism changes from boundary or mixed to fluid film, the wear rates will reduce to very low levels; this can potentially be achieved in hard on hard bearings but only if the head diameter is large enough to give sufficient sliding velocity to entrain the synovial fluid

Wear rate

- For a hard on hard bearing couple debris volume will be highest in the first year (bedding in)
- For a hard on hard bearing couple the bedding-in wear volume will be greater with a greater radial clearance (radius of acetabular component minus radius of femoral head component)
- For hard on soft bearings wear is nearly linear over time but greatest creep occurs during the first year
- Once bedding-in has been achieved, wear rate will depend on the applied load and the sliding distance
- In a ball and socket joint the sliding distance will be greater with a larger head diameter, although the applied load per unit area will be smaller

- The overall sliding distance for an individual joint will be proportional to activity
- A small head diameter will, overall, lead to a reduced volume of wear particles for any given bearing couple

'The ideal hip implant'

Candidates become very anxious about what to say if they are asked what their ideal implant is or what they use. It must be realized that this question is just a 'setting' question to introduce the topic, and the question is really about the basic science underlying choice of implant. There is no right or wrong answer to the actual choice of implant.

In the basic science oral it would be reasonable to discuss the relative merits of existing implants, covering topics such as levels of evidence, survivorship analysis, etc. In many cases, however, the topic is going to move onto prosthesis design.

A number of factors could be considered in relation to implant design[25], but the overall aim is to have:

- Materials that are biologically compatible
- An implant that reproduces the 'normal' anatomy of the hip joint
- Both early- and long-term stability of fixation
- A stable articulation
- Low wear rates
- Minimal adverse effects on surrounding tissues, including bone and soft tissues

Aspects to consider include the following.

Stem geometry

- Stem geometry is interlinked with stem fixation
- *Double tapered stems* designed for cemented fixation require the necessary surface to allow slight subsidence to generate the appropriate hoop stresses
- Medial offset (horizontal distance between the long axis of the stem and the centre of rotation of the head) – Increasing the offset will reduce joint reaction forces but increase torque forces on the fixation interface and may also alter tension in the soft tissues
- Length of stem
- Whether or not to use a collar – In theory a collar transmits loads to the proximal part of the femoral stem, which avoids stress shielding, but in practice it is not possible to achieve reliable load transmission both through the collar and through the proximal part of the prosthesis itself and overall fixation may, therefore, be impaired

Stem fixation

- For cemented stems there are two fundamental philosophies
  - Tapered polished stems (e.g. Exeter) rely on slight subsidence to produce high radial compression forces in the cement, supported by hoop stresses in the bone, the shear forces are low and there are almost no tension forces. These stems must be polished to allow the slight subsidence[26]
  - Shape closed stems rely on shear forces between the implant and the cement. The compression forces on the cement are relatively low. The tensile forces are medium
- Cementless stems can have a porous surface or be pre-coated with hydroxyapatite, or both. Hydroxyapatite can either be confined to the proximal region or cover the entire stem. Long-term dissociation of coating from the stem has led some experts to conclude that the ultimate effect of hyroxyapatite coating is to generate an effect similar to a cemented polished stem

Head size

For hard on soft bearing couples there is a compromise to be made between conflicting biomechanical principles:

- Increasing head diameter results in
  - Reduced dislocation risk: The jump distance is greater for a large femoral head diameter
  - Increased range of movement before impingement (owing to the potential increase in the head : Neck ratio)
  - Increased volume of wear particles owing to increased sliding distance

For hard on hard bearing couples there is less of a conflict:

- Increasing head size results in
  - Reduced dislocation risk
  - Increased range of movement before impingement
  - Potential for fluid film lubrication, resulting in greatly reduced wear

Head fixation and taper junctions

- Monoblock femoral components avoid head/neck interface problems but prevent adjustment of neck length after stem implantation and require larger inventories
- Modular head fixation is achieved by one of the varieties of Morse taper

The concept of Morse tapers in general will now be discussed in more detail:

- A Morse taper is a method of joining to components together that is commonly used in engineering construction
- The Morse taper consists of a male (**trunnion**) and female component (**bore**), both of which have the shape of a truncated cone (**frustrum**). The angle of the taper is the angle of slope of the cone measured against the longitudinal axis of the taper. A stylized Morse taper is illustrated in Figure 30.14
- Contact is side to side – The Morse taper is not end loading
- Tapers with an angle of $>7°$ are called 'self releasing' or 'fast' tapers and can easily be pulled apart but If the angle of

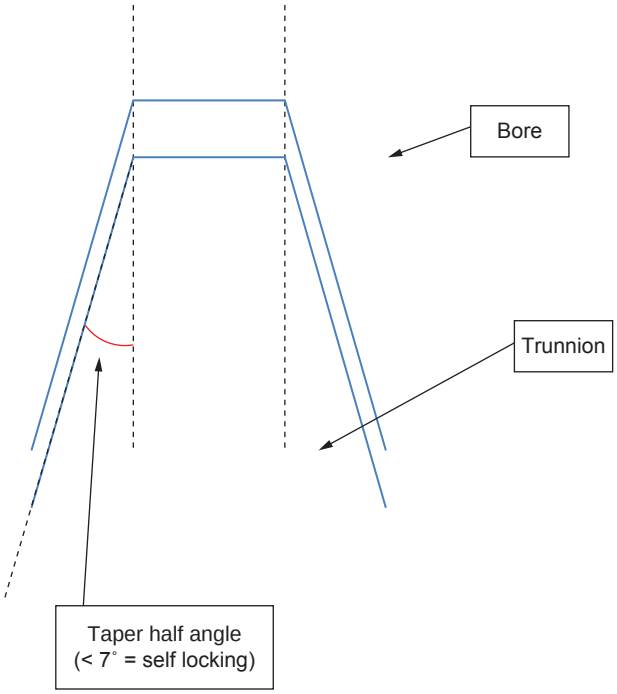

Figure 30.14 Morse taper

the cone is sufficiently small the taper will be **self locking** – I.e. it will not require an additional locking device to keep the two components together

- As a general rule a combined angle of 14° (7° each side) or less will result in a self locking taper. The smaller the angle the harder it will be to separate the two components. When the two components are forced together cold welds (transfer of the soft material to the harder one) occur at the interface
- Another factor influencing the tightness of fit include the surface finish of the two materials
- The two components of the Morse taper form a very rigid and firm fit that depends on friction between the two surfaces, Van der Waals forces, and the formation of cold welds
- Wear may occur at the Morse taper interface due to fretting and fretting corrosion
- The wear particles, typically oxides, are often hard and they can migrate between the bearing surfaces of the hip causing *third body* wear
- Wear at the Morse taper is Mode IV wear
- Wear at the Morse taper (*trunnionosis)* has been implicated in Adverse Reactions to Metal Debris and has been described as being characterized by a high level of cobalt ions in the blood
- Note that although wear at the Morse taper is often described as trunnionosis the main site of wear is often the bore rather than the trunnion despite the bore being in a harder material
- To minimize the risk of fretting occurring at the Morse taper dimensions and geometry and the surface finishes are critical. As it is practically impossible to achieve a perfect match. A design decision is, therefore, made about whether maximum contact loads occur near the tip of the trunnion (negative mismatch) or near the base of the trunnion (positive mismatch). There is some evidence to suggest that negative mismatch results in less fretting

Stem modularity at other sites –– Morse taper junctions are used at other sites as well as at the head–neck junction, particularly for revision components; these sites include the neck–stem junction and the mid-stem junction. These other junctions may also be the source of particles as a result of fretting and fretting corrosion.

Acetabular component geometry

- For cemented acetabular components a flange may provide better pressurisation of cement
- For non-cemented components supplementary screw fixation may be used or not
- For non-cemented acetabular components with UHMWPE inserts the presence of screw holes may allow polyethylene particles to pass to the interface and lead to cyst formation and/or loosening
- The geometry of the acetabular component may be a full hemisphere (for increased stability) or not a complete hemisphere (smaller jump distance but less potential impingement)
- Constrained components may increase stability but increase the load on the component-cement interface

Bearing couple

- Hard on soft or hard on hard bearing couples can be considered and their relative merits discussed[27]
- The wear rate for hard on soft bearings can be improved with the use of highly cross-linked polyethylene or polyethylene with antioxidant treatment, although consider the other mechanical consequences of increased cross-linking of the polyethylene
- Metal on metal bearings offer the potential for very low wear rates but they may be susceptible to scratching and cup orientation may be critical for low wear rates, open cups being associated with ALVAL – See below
- Ceramic on ceramic bearings have historically been associated with the problem of fracture of components. There are some concerns about stripe wear, which is probably caused by microseparation of the components, although the very small sized wear particles may be less problematic than the larger particles produced by polyethylene wear. A further problem with some ceramic on ceramic bearings is squeaking, which is probably due to impingement

ARMD and ALVAL

There has been a great deal of concern about metal on metal bearing couples and ARMD (adverse reactions to metal debris) and ALVAL (aseptic lymphocyte-dominated vasculitis-associated lesions).

Pathology

When excessive wear occurs at metal on metal bearing couples or junctions, nanometer-sized cobalt chrome wear particles become deposited in the periprosthetic tissue.

- **None specific reaction**: The metal wear particles are taken up by macrophages. The release of high levels of metal ions causes cell necrosis, which histologically is seen in the more superficial layers of membrane surrounding the implant
- Specific reaction in which high numbers of lymphocyts and plasma cells are seen in the perivascular areas in the per-implant tissues. The pathological process is thought to be a T-cell type IV delayed hypersensitivity to cells and tissues altered by the cobalt and chromium wear debris. The reaction causes further macrophage recruitment and further inflammation and damage

The process can lead to the formation of cystic and/or solid masses near prostheses, with marked necrosis and both the specific ALVAL and the non-specific macrophage responses outlined above.

The metal debris may arise both at the metal on metal bearing couple itself and at the site fretting corrosion at Morse tapers (either at the head/neck junction or at the neck/shaft taper in modular components.

- The probable sequence for production of metal wear debris at Morse tapers is fretting – Oxidation of wear particles – Further fretting and third body wear – Pitting corrosion – Further debris, etc, the whole process being called fretting corrosion. Third body wear also then occurs at the metal on metal bearing couple. The debris cause specific and non-specific inflammatory changes in the periprosthetic soft tissues, with tissue necrosis

Risk factors for adverse reaction to metal debris

There are a number of factors that may predispose to excessive wear of metal on metal hip replacements:

- **Acetabular component design**
  - Relatively shallow acetabular components predispose to edge loading
  - Suboptimal clearance – The clearance (the difference in radius between the acetabular and femoral head components) influences the lubrication of the bearing
- **Surgical factors**
  - Angle of inclination of the acetabular component – A high cup angle predisposes to edge loading
  - Retroversion of the acetabular component – This may predispose to impingement and wear
  - Size of component – For resurfacing arthroplasty problems have more commonly been associated with relatively small acetabular component sizes, which have usually been those used in female patients

Investigation of patients with suspected ARMD

Patients who have had hip replacements with metal on metal bearings should be regularly monitored. The recommendations for monitoring have been laid down by the Medicines and Healthcare products Regulatory Agency (MHRA). These include the following:

- Annual measurement of levels of chromium ions and cobalt ions in the blood. The blood tests must be taken in the specified way to avoid contamination of the sample by metal in the hypodermic needle
- The MHRA threshold is put at >7 ppb (μg/l) of either chromium or cobalt: 7 ppb equates to 134 nmol/l for chromium and 119 nmol/l for cobalt
- MRI scanning with MARS (metal suppression) protocol, or an ultrasound scan should be performed in symptomatic patients or patients with levels of chromium or cobalt elevated above the MHRA threshold

Biomechanics

Many candidates turn pale when asked to draw free body diagrams or when asked to explain the mechanics of constructs. The examiners are not expecting candidates to be qualified engineers but they do expect them to have some understanding of the basic concepts underlying orthopaedic constructs and locomotor systems. It is best to start with simple concepts and build them up progressively. It cannot be emphasized enough that practice at drawing vector diagrams and free body diagrams for different situations will make the answering of biomechanics questions much easier. Do not try to memorize specific diagrams; understand the concepts and be able to apply them to new situations.

Scalars and vectors

- A scalar is a value (e.g. temperature, speed)
- A vector has both a value and a direction (e.g. force, velocity)

Vector analysis

- Vectors may be represented by a scale drawing, where the length of the line represents the magnitude of the vector and the arrow its direction
- A vector direction must be referenced to a set of Cartesian coordinates (x, *y*, *z* axes)
- Vectors may be added and subtracted using scale drawings (Figure 30.15) – Using parallelograms or, in the case of forces at right angles to each other (such as along the *x*, *y* and *z* axes), using geometry
- A vector can be decomposed to give its components along individual *x*, *y* and *z* axes using right angled triangles. Figure 30.16 illustrates how a force V can be decomposed into two components along the *x* and *y* axes
- The magnitude of the components along the *x*, *y* and *z* axes can be found from a scale drawing or by utilizing geometry
- The mnemonic SOHCAHTOA is useful for remembering the geometry
- Sine = Opposite/Hypotenuse

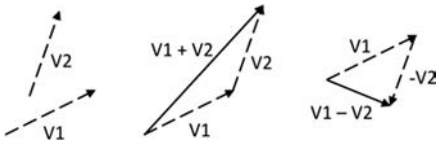

Figure 30.15 Vector addition

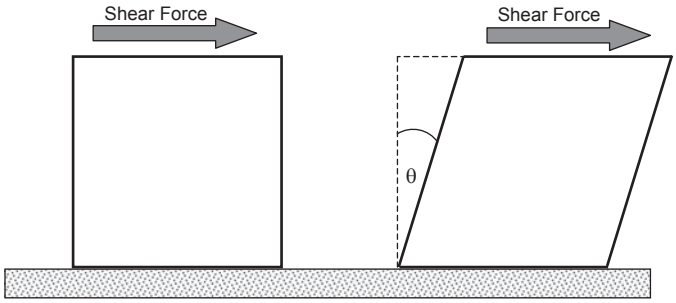

Figure 30.17 Deformation under shear

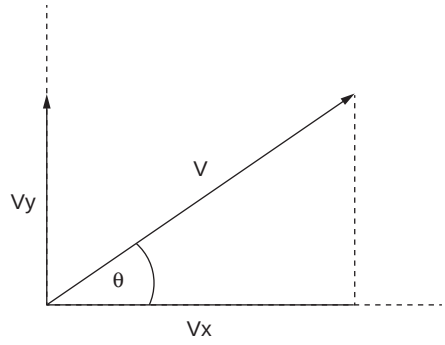

Figure 30.16 Vector decomposition

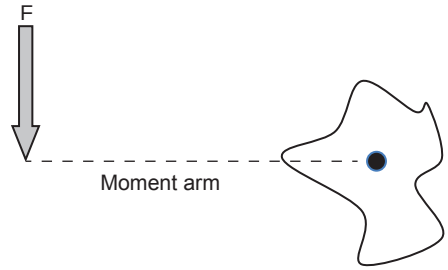

Figure 30.18 Moment arm and pivot point

- Cosine = Adjacent/Hypotenuse
- Tangent = Opposite/Adjacent

Newton's laws

Newton's first law: For a body in equilibrium the sum of forces and moments = 0

Newton's second law: Force = mass × acceleration

Newton's third law: To every force there is an equal and opposite force

Statics

- A force applied normal to the surface of an object will cause either compression or tension
- A force applied tangentially to the surface of a body will cause shear
- **Newton's second law** indicates that an object will remain in a state of equilibrium unless a (net) force is applied to it
- If a compression force is applied to an object and the force is opposed by an equal and opposite force, the object will shorten along the line of action of the forces; the shortening is described as **strain,** measured as change in length per unit length, or expressed as a percentage. The **stress** causing this strain is expressed as force/unit area (N/m^2 or Pascals, often expressed as MPa)
- If a tension force is applied and opposed by an equal and opposite tensile force, a tensile stress and strain will result
- If a force is applied tangential to the surface and opposed by an equal and opposite force applied to the opposite surface, a **shear stress** will occur and the object will deform as shown in Figure 30.17, the shear being expressed as an angle. When a cylinder is twisted at opposite ends, the result is a **torque shear stress**
- Shear stress causes compression and tension forces that lie at 45° to compression and tension forces resulting from forces applied normal to the surface

Kinematics

- **Newton's second law of motion** can be summarized as $F = m\times a$, where F = force, m = mass and a = acceleration
- Any shape can be represented as having a centre point, the centroid, which is calculated using calculus
- A solid body also has a centroid
- An 'extruded shaped object, e.g. a cylinder' with a regular cross-sectional area has a centroidal axis
- According to Newton's second law, when a net force is applied to an object that object will accelerate
- If the force acts through the centroid the object will accelerate in the direction of the applied force and a translatory movement will occur
- If the force does not act through the centroid then the imparted movement will be both translatory and rotatory; the rotatory component being imparted by the moment of the force
- The moment arm is the length of the line drawn perpendicular to the vector and passing through the pivot point (Figure 30.18)
- The moment is calculated by multiplying the magnitude of the force by the length of the moment arm

Bending forces

If two or more forces are applied to an object the object will deform. In orthopaedics the deformation behaviour of beams and cylindrical objects is of paramount importance, particularly for fracture fixation. The basic understanding of simple beam characteristics is, therefore, a common area that is explored in the basic science oral.

The engineering aspects of stresses and strains in beams are complex and depend upon many factors, such as the distribution of the application of the force, etc. A detailed analysis of beam mechanics is beyond the requirement of orthopaedic trainees and the descriptions given below are at a relatively simple level but sufficient for a working practical knowledge as necessary for the practice of orthopaedics. For more detailed analyses it would be necessary to consult with an engineer.

Cantilever bending

- A cantilever is a beam that is fixed at one end
- If a downward force is applied to one end of a cantilever, tension will occur on the upper surface and compression on the lower. The tension and compression forces will be maximal at the surfaces and running down the middle there will be a neutral axis (usually corresponding with the centroidal axis in a uniform beam) where the tension and compression forces are zero
- There is a linear relationship between the magnitude of the tensile and compressive forces within the beam and the distance from the neutral axis (Figure 30.19)
- The resistance to bending of a beam, or any other object, will depend not just on the modulus of elasticity of the material from which it is made but also on the distribution of the material around the centroidal axis; this distribution is described by the term **second moment of area**, or **second moment of inertia**
- For a solid beam the second moment of area $I = bh^3$, where b is the breadth and h is the height (or thickness). It can be seen that if the thickness of the beam is doubled the stiffness will increase by a factor of 8, whereas if the width is doubled the stiffness will just double
- For a solid rod the second moment of area $I = \frac{1}{4}\pi r^4$
- The tensile and compressive forces increase in a linear fashion with distance from the fixation point (assuming a weightless beam), as illustrated in Figure 30.20 a for cantilever bending
- The strain of the beam will be proportional to the square of the distance of the force from the fixation point
- For a weightless beam the sheer forces will be uniform along the length of the beam

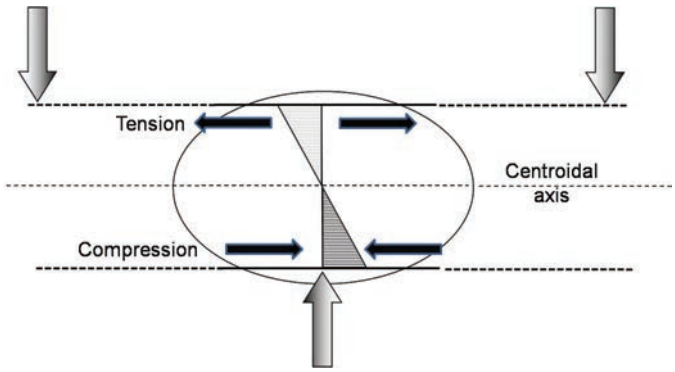

Figure 30.19 Stresses across a beam when being bent

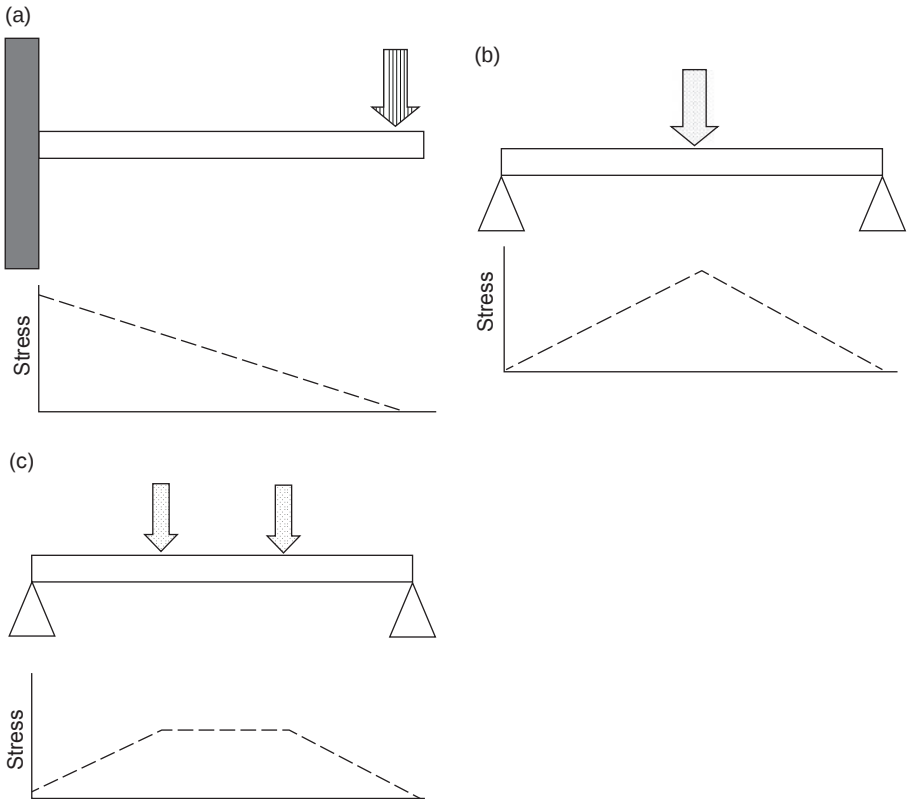

Figure 30.20 (a) Stresses along a (weightless) beam under Cantilever bending. (b) Stresses along a beam under three point bending. (c) Stresses along a beam under four-point bending

Three- or four-point bending

- A beam supported at both ends can be considered as equivalent to two cantilevers joined back to back
- The tensile and compressive forces applied to a beam when subjected to three- and four-point loading are illustrated in Figures 30.20 b and c

Torque forces

- When a turning couple is applied to an object torque forces result
- If the turning moment at one point is resisted by an equal and opposite turning couple at another point on the same axis, torque (shear) strain will be applied, resulting in torque shear
- The resistance to deformity when a torque strain is applied is also related to the distribution of material within the object; the term to describe this distribution is the **polar moment of inertia**
- For a rod-shaped object, the polar moment of inertia, $J = \pi r^4/2$
- It can be seen that the stiffness of a cylindrical object to twisting is proportional to the fourth power of the radius
- The stiffness of a hollow cylinder is proportional to the fourth power of the outer radius minus the fourth power of the inner radius
- A hollow intramedullary nail is less stiff than a solid nail *of the same diameter,* but if a constant volume of the same material is used to make two nails the hollow nail will be stiffer than the solid nail because of the greater outer diameter
- The same fourth power relationship applies to the bending stiffness of a bone screw, which is important in the design of the screws used in locking plates, which depend on their bending stiffness to fix the fracture, rather than on their pull-out strength; hence, locking screws have a larger core diameter and small thread depth than conventional screws

Table 30.6 gives the moments of area/inertia for elements of circular and rectangular cross-section[28].

Stiffness of constructs and working length

Another popular topic is the stiffness of constructs used for fixation of fractures.

- The stiffness of plates will depend on the *modulus of elasticity* of the material from which they are made and their *second moment of area* (which will be affected by the shape and profile of the plate)

Table 30.6 Moments of area/inertia for simple beams and rods

	Second moment of area (I)	Polar moment of inertia (J)
Rectangular beam	$\frac{1}{12}bh^3$	
Cylinder	$\frac{1}{2}\ \pi r^4$	$\frac{1}{4}\pi r^4$

- The stiffness to bending of screws, intramedullary nails and external fixation components will also depend upon the *modulus of elasticity* of the material from which they are made and their *second moment of area*
- The stiffness of intramedullary nails, etc to torque forces will depend upon their *sheer modulus and their polar moment of inertia*

When these device are applied or inserted, however, an additional concept will need to be considered: **The working length**

- **The working length of a device refers to the total unsupported length**
- Increasing the working length will decrease the stiffness of the construct, i.e. strain increases with increased working length
- For a cantilever beam loaded at its free end the maximum deflection at its free end is proportional to the length cubed – Maximum deflection = $FL^3/3EI$, where E is the modulus of elasticity and I is the second moment of area
- The angulation at the end of the cantilever beam, when loaded at its end, is proportional to the length squared
- The stiffness of an external fixation pin will be proportional to its Young's modulus, and the fourth power of its radius. The working length of the pin will be the distance between its attachment to the bar and the point at which it reaches the bone. Far cortex locking screws and dynamic locking screws deliberately lengthen the working length of the screws to allow more movement (strain) at the fracture site. By increasing the working length of either the entire device or of component parts of it the stiffness of the applied construct can be reduced, and vice versa
- The working length of an intramedullary nail will depend upon whether or not it is firmly wedged in the cortical bone near the fracture site. For an unreamed nail the working length will usually be the distance between the interlocking screws nearest the fracture but for a reamed nail the working length may be much shorter if the nail is firmly jammed in the bone at the isthmus on one or both sides of the fracture
- The working length of an intramedullary nail may differ for rotation and bending forces, as when the bone bends at the fracture site the nail may become fixed to the bone by three-point fixation
- For an external fixation device the working length is the distance between the two pins nearest to the fracture
- In theory a longer working length of a device decreases the stiffness of the construct but, for any given bending force applied to the fractured limb, the stress within the fixation device should decrease because the load is shared along the length of the device. Experimental results, however, have not always confirmed that the stress in plates reliably decreases as the working length increases and it is possible that the greater deflection with increasing working length may give rise to fatigue problems, particularly at the nearest fixation points

Free body diagrams

Free body diagrams are one of the most popular topics in the basic science oral. In the past this has usually involved the free body diagram for the hip, but other joints may be used as the basis for discussion.

Basic underlying limitations

Free body diagrams are a simple method for calculating the forces around a *stationary* structure or an element of that structure. It is important to realize the limitations of the method:

- The body must be in equilibrium
- The number of unknown forces must not be too great otherwise the problem becomes statically indeterminate
- The calculation only considers two dimensions

Underlying principles

- If a body is in equilibrium, the anticlockwise (positive) and clockwise (negative) moments must add up to zero
- If a body is in equilibrium, the sum of the vectors must add up to zero
- If a vector diagram is drawn, the result will be a closed polygon (triangle for three vectors)

Steps in drawing a free body diagram

- Select the area to be studied (this is your choice – As these are static free body diagrams all parts must be in equilibrium with all other parts; therefore, it is legitimate to select just one part of a complex system for analysis)
- Decide which is the moving part and which is the static part
- Draw the known forces and moment arms (when deciding in which direction a muscle vector should point, think of yourself inside the fixed part and imagine the moving part pivoting around you)
- Ignore forces passing through the pivot point as these do not produce any moments around that point
- Find any unknown force that exerts a moment by balancing the moments
- Now that the forces causing moments are known, the unknown translatory force passing through the pivot point can be calculated as the sum of the translatory forces must add up to zero
- In a simple see-saw example the forces are all along the same axis; when the forces are not along the same axis then the vectors must be decomposed to give the components along the *x* and *y* axes, or the resultant force can be worked out by making a scale drawing (remembering that the length of the line is proportional to the magnitude of the force and not, for example, the anatomical length of the muscle that may be producing it)

Common mistakes when drawing free body diagrams

- Forgetting that the moment arm meets the vector *at a right angle;* when drawing the hip diagram many candidates draw the line from the centre of rotation of the hip to the tip of the greater trochanter, or in the shoulder from the centre of the humeral head to the insertion point of the deltoid on the humerus, both usually incorrect
- Confusing the anatomical length of the muscle with the length of the line representing the magnitude of the vector
- Forgetting to establish which component is the moving part and which the fixed; the direction of the vectors is decided by their attachment to the moving part and not the fixed part (an important point when looking at the femoral/tibial articulation or the patellofemoral articulation, respectively)
- Making the diagram too small

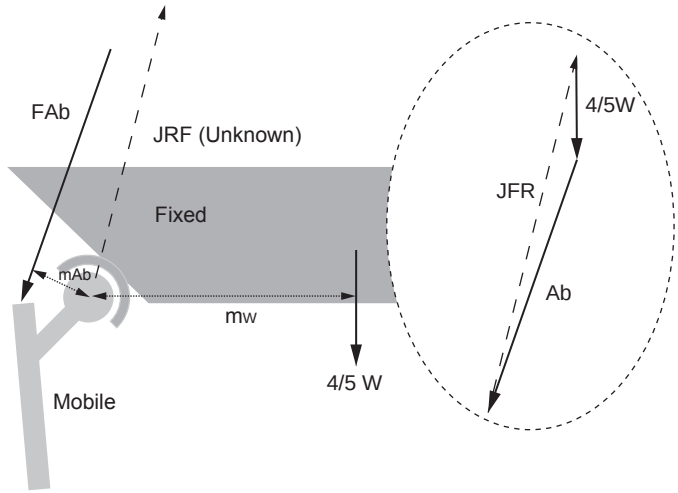

Figure 30.21 Free body diagram for the hip when standing on one leg

Example for the hip joint[30]

- Figure 30.21 shows a stylized free body diagram for the hip for standing on one leg
- The unknown abductor force (*F*Ab) can be calculated by balancing the moments

Force of abductors (FAb) × moment arm of abductors (m_{Ab}) = the weight of the body (minus the weight of the stance leg) × the moment arm m_w

- As the joint reaction force acts through the pivot point it has no moment arm and does not need to be considered when the moments are being balanced
- Once *F*Ab is known a force triangle can be drawn to scale to calculate the joint reaction force (JRF), which will be equal and opposite to the sum of the two vectors *F*Ab and *W*

Effect on hip joint reaction force of using a walking stick

- The use of a walking stick in the hand opposite to the affected hip provides a moment that partially balances the moment produced by body weight and the force required to be produced by the abductor muscle is, therefore, correspondingly reduced. The moment arm of the force

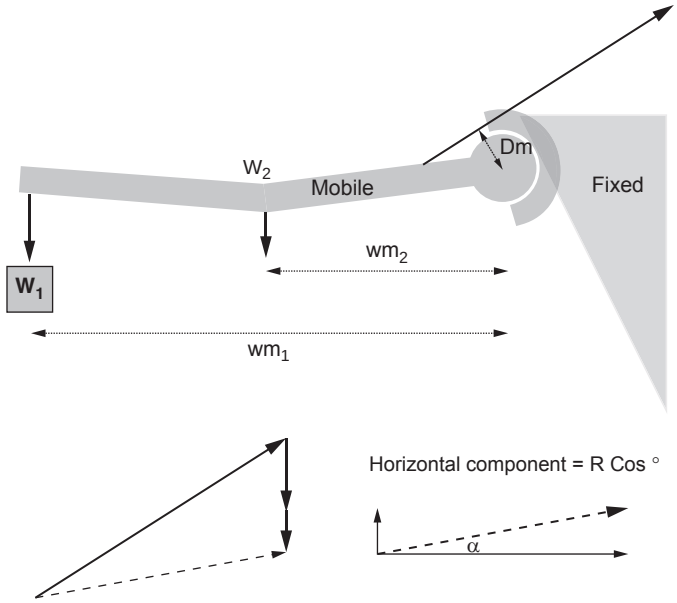

Figure 30.22 Free body diagram for the shoulder when holding a weight in the hand

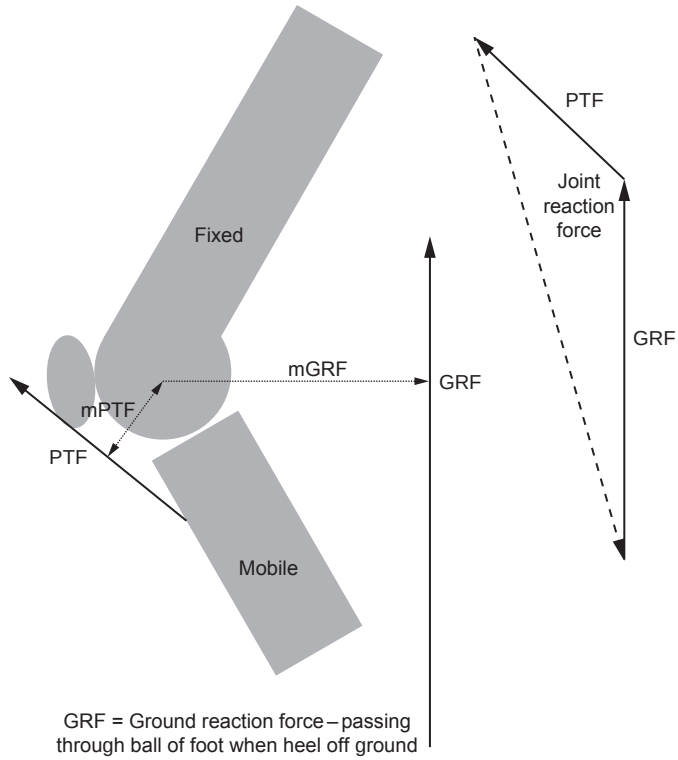

Figure 30.23 Free body diagram for the knee – Tibio-femoral joint reaction force when standing on one leg with knee flexed

through the walking stick is long, so as a result a force of 15% of body weight through a walking stick may reduce the JRF by up to 60%

Free body diagrams for other joints

Although the hip joint free body diagram is the most common one encountered in the exam, you should also be able to do similar diagrams for the ankle, knee, patellofemoral joint, elbow and shoulder. All are based on variations of the balance beam principle. Be prepared conceptually to 'turn the balance beam upside down' to do diagrams for, for example, the ankle, or to put both forces on the same side of the pivot point (but with opposite sign moments), for example, in the elbow and the shoulder.

Figure 30.22 shows an example for the shoulder. In this free body diagram, $w\mathrm{m}_1$ is the moment arm for the weight being carried and $w\mathrm{m}_2$ is the moment arm for the weight of the arm itself. *D*m is the moment arm for the deltoid muscle force. Once Dm has been calculated by balancing the moments, the JRF can be calculated by drawing a vector diagram. The horizontal and vertical components of the JRF can be calculated using geometry.

Figure 30.23 shows an example for the knee in equilibrium standing on one leg (such as on a step). In this particular example the femur is taken as fixed and the tibia as mobile. The ground reaction force (GRF) acts upwards through the ball of the foot and exerts a counterclockwise moment on the tibia, which is countered by the clockwise moment exerted by the quadriceps mechanism via the patellar tendon. The JRF can be calculated by drawing a force triangle.

When calculating the joint reaction force for the knee the *instantaneous* pivot point is the contact point between the femur and the tibia; this is because the free body diagram is looking at the situation in static equilibrium. The instantaneous pivot point is different from the axis of rotation of the knee, which considers the dynamic situation. It can be seen that in roll back the instantaneous pivot point moves backwards, thus, increasing the moment arm of the force being transmitted through the patella tendon, which in turn decreases the joint reaction forces when the knee is in deep flexion.

Once the patellar tendon tension has been calculated from the free body diagram as in Figure 30.23, the patellofemoral compression force can then be calculated by constructing a parallelogram from the two vectors made up of the tension in the quadriceps ligament and patellar tendon proximal and distal to the patella respectively (which are assumed to be of equal magnitude for this simplified calculation) then drawing the resultant force; the JRF will be equal and opposite to this force (Figure 30.24). *It should be noted that this is a simplication as in the actual knee the patella tilts and as a result the tensions proximally and distally are not equal.*

Hamilton–Russell traction

The principles of the free body diagram can also be used to show how traction works. A compound pulley can be analysed in the same way as any other system. It is important to establish which is the moving part and which is the fixed; the total traction force applied by the traction cord at the pulley

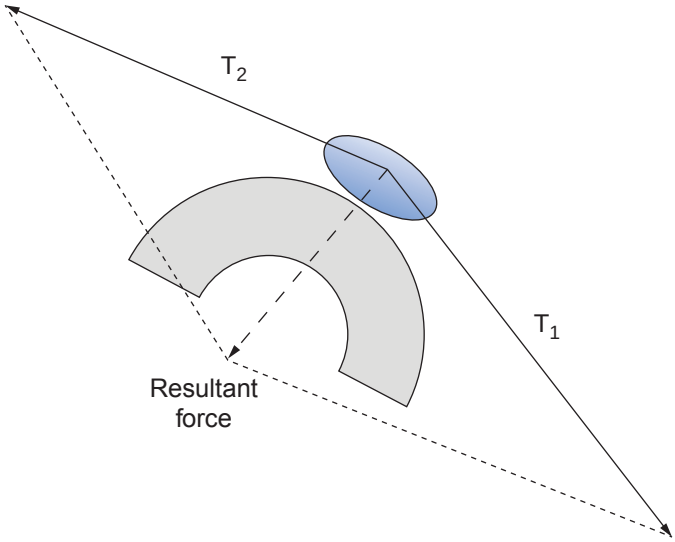

Figure 30.24 Free body diagram for the patellofemoral compartment of the knee

will depend upon the number of cords acting on that pulley so the number of cords reaching the moving pulley will be the number to use for calculating the total force. For a more detailed analysis allowance should be made for friction in the pulleys (allow for 10% loss in traction for each pulley) and for the effect of gravity when the foot end of the bed is tilted to prevent shear forces between the patient and the bed. The principle of Hamilton–Russell traction is shown in Figure 30.25.

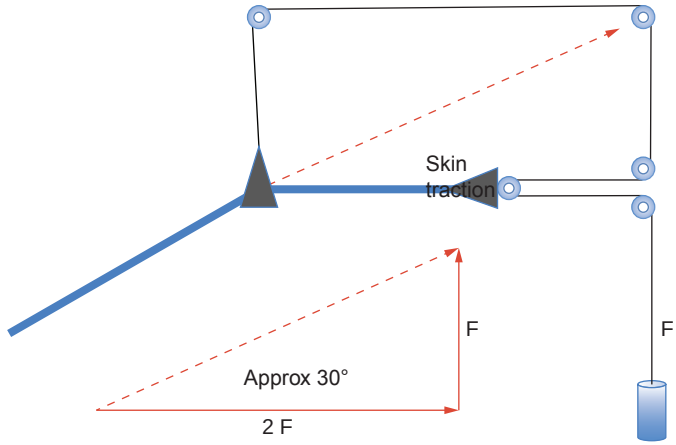

Figure 30.25 Hamilton–Russell traction

Screws

Definition

A screw is a machine that converts a rotational movement around an axis into a translational one along the same axis

Component parts

If asked to describe a screw consider the following component parts:

Head
Countersink
Shaft
Thread
Tip

Also consider the material the screw is made from.

Although screws are designed for specific purposes remember that a particular screw may nevertheless be used in different ways or to fulfill different functions depending upon the may it is inserted.

Key dimensions

Core or inside diameter
Outside diameter: The diameter across the threads
Shaft diameter
Pitch: The distance between the tips of two successive threads
Lead: The distance the screw advances with one complete turn of the screw
Length

Screw heads

The driver of the screw needs to be considered:

- Non-self aligning

  Slot
  Cross head
  Phillips
- Self aligning

  Hex
  Star – The star head is less likely to strip than the hex and, therefore, has better torque transmission

Shaft and core diameter

The resistance to bending is proportional to the fourth power of the core diameter.

For cannulated screws the resistance to bending is proportional to the fourth power of the outer diameter of the core minus the fourth power of its inner diameter

A larger core diameter screw allows a larger guidewire whilst maintaining the bending stiffness of the screw.

Locking screws need to be resistant to bending and are less reliant on pull out strength and are, therefore, designed with a relatively larger core diameter.

Thread

Compression screws may be designed partially threaded to ensure they only grip distally.

'Double start' screws have a lead that is twice the pitch – They have two parallel threads – Double start screws advance much quicker whilst retaining pull-out strength.

Conventional screws in plates work by applying compression force to increase the friction between the plate and the

bone – These screws, therefore, require high pull-out strength and are designed with a relatively high outer to core diameter ratio.

Locking screws require less pull-out strength as they work with a plate as a fixed angle device; they are, therefore, designed with a narrower outer (thread) width to allow a larger core diameter.

Tip

Self tapping screws have a cutting flute at the tip

The first few threads of self tapping screws are of progressively greater diameter and are designed for tapping the hole; these threads do not hold the bone well and the screw should be advanced beyond these threads

Self cutting, self tapping tips – These screws may strip their threads proximally if the screw does not advance sufficiently as the tip cuts the bone on the far cortex, they are, therefore, best suited for use in cancellous bone or as unicortical screws

Special screws

Dynamic compression screws – These have an outer cylinder with thread and an inner component that allow lateral movement proximally but not distally. They allow controlled axial movement at a fracture site.

Dual thread screws (such as Herbert screw) – These have a relatively large pitch smaller diameter thread distally and a narrower pitch wider diameter thread proximally. As a result of the differential pitch they compress the fracture surfaces together.

Conical screws – These screws become loose very quickly on being 'unscrewed'.

Kinematics of joints

Knowledge of the kinematics of some specific joints and structures is frequently explored in the basic science oral. The most popular topics are the knee, the subtalar joint and the spine

The wrist[30]

- Anatomically the bones of the carpus are usually considered as two rows: Proximal and distal
- Functionally the carpus can be considered as three columns
  - Central – The distal row and the lunate
  - Lateral – The scaphoid
  - Medial – The triquetrum
- Palmarflexion range is greater than dorsiflexion
- Ulnar deviation range is greater than radial deviation
- The volar ligaments are important stabilisers
- Volar extrinsic ligaments pass from the radius and ulna to the carpal bones
- Volar intrinsic ligaments pass between the carpal bones
- The carpal bones form a double hinge
- Activity of the wrist muscles tends to cause the double hinge to buckle
- The tendency to buckle is resisted by the shape of the articular surfaces and the ligaments
- The lunate and scaphoid are narrower on their dorsal surfaces than on their volar surfaces, tending to force the wrist into extension when compressed longitudinally. This is countered by the trapezium and trapezoid, which articulate with the dorsal aspect of the distal scaphoid
- Flexion/extension rotation and radioulnar deviation occur around instant centres of rotation in the proximal part of the capitate
- About two-thirds of flexion occurs in the midcarpal joint with one-third in the radiocarpal joint
- About one-third of extension occurs in the midcarpal joint with two-thirds in the radiocarpal joint
- On radial deviation the scaphoid and proximal carpal row flex and on extension they extend

The spine

- The motion segment consists of two adjacent vertebrae and the intervening soft tissues
- The motion segment can be divided into anterior and posterior columns; the anterior column contains the vertebral bodies, disc and longitudinal ligaments; the posterior column contains the facet joints, transverse and spinous processes, vertebral arches and intervening ligaments
- The movements of the motion segments are interlinked
- Movements in one plane involve obligatory motion in other planes
- Although six degrees of freedom are possible at all levels, the proportional freedom varies with the differing morphology of the vertebral bodies
- When considering the thoracic and lumbar spine
  - Flexion and extension movement increases progressively with more caudal motion segments
  - Lateral flexion is maximal in the lower thoracic spine motion segments
  - Rotation movement is maximal in the upper thoracic motion segments, decreasing caudally with the exception of the lumbosacral motion segment, where there is some increase in rotational movement
- The integrity of the curves (cervical, thoracic and lumbar) of the spine is an important consideration in its ability to withstand forces applied to it
- The compressive and shear forces on the intervertebral disc can be calculated using a free body diagram of a motion segment. The moments of the body weight and any weight being lifted are resisted by the spinal muscles, acting with a very short moment arm

The knee joint

The knee joint has a complex shape and motion pattern. When viewed from the anatomical Cartesian coordinates it is complex, with sliding and rolling occurring at the articular surfaces.

The instant centre of rotation around the transverse axis, seen within these coordinates, moves posteriorly during flexion, describing a J-shaped curve when seen from the side.

The rigid four bar mechanism

Classically the motion pattern has been modelled as a rigid four bar mechanism, in which the anterior and posterior cruciate ligaments form two bars of the mechanism and the bones connecting their proximal and distal attachments, respectively, form the other two rigid bars.

The kinematic model based on the rigid four bar mechanism is largely two-dimensional.

The transepicondylar axis and the medial pivot mechanism

More recent work has studied the rotation of the knee around the transepicondylar axis. When viewed along this axis the femoral condyles appear spherical rather than ellipsoidal in shape and during the functional arc of flexion/extension there is very little movement of the medial condyle on the medial tibial plateau in the AP plane.

The spherical radius of the femoral condyles, when viewed along the transepicondylar axis, has given rise to the concept of the *single radius* knee replacement[31].

Current thinking on knee kinematics divides flexion of the knee into three arcs:

- From full extension to 10° of flexion the 'screw home' mechanism operates
- The arc of flexion from approximately 10° to 120° is the functional arc', within which most activities of daily living occur. Within this functional arc there is very little AP movement in the medial compartment, most AP translation taking place in the lateral compartment; the knee can be seen as operating with a medial pivot, with rotation occurring around the longitudinal axis
- Beyond approximately 120° of flexion AP translation occurs in both the medial and lateral compartments, with the femoral condyles rolling back on the tibial surfaces

Anatomical correlations

- The medial tibial plateau has a concave contour
- The lateral tibial plateau has a convex contour, mainly caused by thicker articular cartilage in the centre of the plateau
- The lateral meniscus is more mobile than the medial
- The lateral collateral ligament is less closely attached to the periphery of the meniscus than the medial
- These anatomical features correlate with the greater AP mobility within the lateral compartment when compared with the medial

The ankle

- The distal tibial and corresponding upper talar articular surfaces slope upwards towards the lateral side by about 3°
- The talar body forms a truncated cone (frustum) with its apex facing medially
- The talar body is wider anteriorly than posteriorly
- The axis of rotation approximates to a line drawn through the tips of the medial and lateral malleoli although the position of the instant axis of rotation moves slightly during flexion and extension

The subtalar joint

- The orthopaedic' subtalar joint is composed of the posterior, middle and anterior subtalar facets
- The 'anatomical' subtalar joint consists only of the posterior facet; the middle and anterior facets and the talonavicular joint forming the 'talonaviculocalcaneal joint', which, together with the spring ligament, form a 'ball and socket' joint
- The axis of the subtalar joint varies widely from one person to another but averages about 42° directed upwards from the heel, and 23° directed medially
- The oblique axis of the subtalar joint results in the subtalar joint acting like a mitred hinge or a *torque convertor*; rotation around the axis of the tibia is converted to rotation around the long axis of the foot, and vice versa
- Mechanical junctions are not unidirectional and it follows that a rotation of the foot along its axis will lead to a rotation of the talus around the axis of the tibia; as a result of supination of the midfoot will cause external rotation of the talus within the mortice of the ankle and this can lead to external rotation fracture patterns in the ankle
- As a result of the axis of the subtalar joint extending laterally to the posterior aspect of the ankle the tendo-Achilles lies medial to the axis. The gastrosoleus, therefore, exerts a powerful inversion force on the hindfoot. If, however, the calcaneum goes into sufficient valgus this may convert the gastrosoleus into a powerful evertor. The work capacity of the gastrosoleus far exceeds that of tibialis posterior

The midtarsal joint

- The joints between the talus and navicular and between the calcaneum and cuboid form the midtarsal joint
- When the heel is inverted by tibialis posterior the midtarsal joint becomes 'locked' and the mid foot becomes stiff
- When the heel is everted the mid foot becomes flexible
- The locking of the midtarsal joint is caused by the calcaneocuboid joint moving beneath the talonavicular joints such that the two components of the midtarsal joint become incongruent with each other
- The interplay between the eversion/inversion of the hindfoot, the unlocking and locking of the midfoot and the windlass effect of the plantar fascia is integral to the foot's changing function during various stages of the gait cycle, ensuring flexibility for walking on uneven surfaces and rigidity for optimal push-off

The gait cycle

The gait cycle may be discussed in various ways in the basic science oral; the components of the cycle may be discussed, or the way that the gait cycle is affected by various interventions.

The gait cycle could also be used as a vehicle to discuss postural changes in the foot during activity, etc.

Background

- The centre of gravity of the body lies above the centre of rotation of the hip
- Translatory forces on the body tend to make the system less stable; in bipedal gait the system is **metastable**
- Falling is prevented by postural reactions and moving the lower limb to brace against the fall
- Walking can, therefore, be considered a series of controlled falls
- **Step** = initial contact of one foot to initial contact of the other
- **Cadence** = steps per minute
- **Stride length** = one full cycle
- **Foot progression angle** = axis of stance foot to axis of motion

The gait cycle

- Defined as the sequence occurring between two consecutive initial contacts of one foot
- Two main phases – **Swing phase** (35–40%) and **stance phase** (60–65%)
- Perry[32] divided the gait cycle into eight **subphases**, or **instants;** five in the stance phase and three in the swing phase
- In walking there is a **double stance phase** during which both legs are in the stance phase simultaneously
- In running there is an additional **float phase,** during which neither leg is in the stance phase
- During the stance phase the lower limb operates as a **closed kinetic chain**
- During the swing phase the lower limb operates as an **open kinetic chain**
- During the stance phase the kinetic energy of the lower limb is transferred upwards to the centre of gravity of the body
- During the swing phase, energy is transferred downwards through the lower limb

The subphases of the stance phase

Initial contact

- Foot makes contact with the ground
- Hip flexed, knee nearly extended
- JRF directed upwards and slightly posteriorly

Load response

- Ankle plantarflexes to allow foot to make full contact with ground
- Eccentric contraction of ankle dorsiflexors
- Hip extending – JRF passes anterior to hip
- Knee flexing
- Commencement of double stance phase

Midstance

- Centre of gravity passes forward over foot
- Ankle dorsiflexes
- Eccentric contraction of gastrocnemiu–soleus muscles
- Ground reaction force passes posterior to hip

Terminal stance

- Heel leaves ground
- Ankle plantarflexes
- Gastro-soleus contracts concentrically

Pre-swing

- Knee flexes
- Ground reaction force directed upwards and slightly anteriorly

The three rockers

An alternative way to break down the stance phase is to consider the three rockers. The three rocker concept is in may ways more useful for orthopaedic surgeons.

First rocker or heel rocker

- Initial contact to 'foot flat' – The ankle is plantarflexing
- Eccentric contraction of ankle dorsiflexors

Second rocker or ankle rocker

- Controlled ankle dorsiflexion, resisted by eccentric contraction of gastrocnemius – soleus, as body moves forwards over foot

Third rocker or forefoot rocker

- Ankle moves into slight dorsiflexion before unloading
- Windlass mechanism in foot 'winds up' plantar fascia
- Concentric contraction of gastrocnemius-soleus

Swing phase

Initial swing

- Knee and hip flex, ankle dorsiflexed
- In relaxed walking the momentum given to the lower limb during the initial swing subphase is one of the main sources of forward momentum of the body

Mid swing

- Tibia swings forwards under thigh

Terminal swing

- Prepositioning of foot prior to initial contact

Efficiency of gait

Efficient gait is achieved by minimizing the upward and downward excursion of the centre of gravity and by preserving forward momentum imparted to the lower limb by

transferring energy up and down the limb via the muscle contractions (particularly the eccentric contractions).

Gage[33] described the five prerequisites for efficient gait:

- Stability in stance
- Foot clearance in swing
- Adequate step length
- Appropriate pre-positioning of the swing phase foot
- Energy conservation

Radiology

X-rays

- X-rays lie at the upper end (high frequency, short wavelength) of the electromagnetic spectrum
- X-rays are produced by electrons, generated from an electron source (cathode), striking a rotating target (anode) made of tungsten
- The process is only 1% efficient, the remainder of the energy being dissipated as heat; hence, the rotating target to avoid meltdown!
- Parameters
  - kV – Unit of measurement of penetration ability of x-rays (i.e. how much energy they carry). Increasing kV increases the forward scatter
  - mA – Unit of measurement of the strength of the x-ray beam current (increasing the mA increases the exposure, which can also be increased by increasing the exposure time interval)
- Two types of x-rays result from the x-ray beam striking the patient
  - **Primary** – Direct from the tube to the x-ray plate, producing the desired image
  - **Secondary** – Reflected from and within the patient/other objects and can blur the image. Reduced by *grids* of lead/aluminium
- NB. When taking x-rays in theatre there may be a greater exposure of theatre staff to radiation from the x-rays reflected back from the patient than from those passing through the patient
- Resolution is the minimum separation between objects for their identification as separate objects
- Contrast indicates the ability to identify objects of differing density
- Contrast medium – A high-atomic-element substance used to enhance contrast between anatomical structures
- Analogue images have an infinite range of density between black and white
- Digital images have a discrete greyscale (levels of grey) between black and white
- Orthogonal images are captured at 90° mutual planes to convey three-dimensional images in two dimensions
- Dose
  - Total dose is measured in Grey
  - Effective dose is measured in Sieverts, enabling comparison of risk between procedures
  - Chest x-ray = 0.05 mSv
  - Flight to USA = 0.10 mSv
  - CT spine = 3.6 mSv
  - Bone scan = 5.0 mSv
- Remember – x-rays are a form of radiation; radiographs are the recorded images made with these x-rays

Image intensifier images

- X-ray photons are converted into light photons at the image intensifier input phosphor
- Visible light photons are converted into an electron beam at the photocathode, which passes by focusing electrodes and strikes the back of the output phosphor, which has a thin aluminium covering on the inner side to stop photons but allow electrons through
- Electrons are converted back into visible light at the output phosphor
- Input phosphor has larger area than output phosphor, resulting in a minification gain
- Each photon at the input phosphor generates about 100 photons at the output phosphor because of the increase in energy levels from acceleration; this is the flux gain
- Result is an increase in brightness of several thousand times
- The image can be magnified by increasing the voltage applied to the electronic lenses; a smaller area of input phosphor is used and the x-ray dosage must be increased to maintain the same noise levels

Computerized tomography

- X-rays in a fan-shaped rotating beam are received by a circle of stationary detectors
- Data is digitized such that every 'point' within the patient is a labelled pixel; because each slice has a depth, each unit of space is called a voxel
- Slices are imaged sequentially, or on faster and newer machines, in a helical or spiral fashion. The fastest machines use multiple slices in a spiral fashion
- Transverse anatomical sections can be produced with high resolution. Data can now be reformatted to any chosen plane, but coronal and saggital planes are the traditional ones
- CT is better at looking at bone than it is at looking at soft tissues
- Windows:
  - Hounsfield units (HU) are a measure of attenuation -the attenuation coefficient
  - Image is centred on a particular attenuation value and greyscale compressed within a window

  - The result is a contrast range appropriate to the desired tissues; 'bony windows' are usually centred on 300 HU with a width of 1200 (the greyscale of the scan is greater than that of the human eye)
  - Hounsfield units – Bone 1000, water 0, air 1000
- Resolution – Is less on CT than on plain radiography owing to the averaging within a voxel that occurs at the edges of objects

DEXA scans

- These have been described in the section on osteoporosis

MRI[34]

- Utilizes the fact that hydrogen atom nuclei (protons) spin on their axis and as they carry a charge they generate a small magnetic field (magnetic moment)
- Outside a magnetic field the protons are orientated randomly
- When the body is placed in a strong uniform magnetic field the protons align themselves with the field, although after about a second there is a slight difference (about one in a million) in the number aligning 'with' or 'against' the magnetic field, so the body becomes magnetized with a 'longitudinal' component
- Smaller electromagnetic coils, which can be turned on and off, provide magnetic field gradients to allow spatial localization when imaging
- If a radiofrequency pulse is applied at a frequency specific to the strength of the magnetic field (the Larmor frequency), the magnetisation is tipped into the 'transverse' plane
- A 90° radiofrequency pulse tips the magnetisation through 90° and a 180° radiofrequency pulse tips it through 180°
- Only transverse magnetisation sends back a radiofrequency signal (spin echo)
- Echo signal is composed of multiple frequencies according to the position along the field gradient
- The multiple spin echo frequencies are mathematically manipulated using a Fourier transform to produce an image, the signal strength at each frequency being dependent upon the local hydrogen density
- T1 relaxation time is the exponential time constant that represents the time to recover 63% of the equilibrium longitudinal magnetisation when the radiofrequency is turned off
- T1 relaxation tie depends on difference between frequency of molecular motions and the Larmor frequency; if they are similar, T1 is short
- Flips of <90° do not convert all the longitudinal magnetisation into transverse and the equilibrium is, therefore, regained more rapidly, allowing multiple rapid repeating of the flips
- Spin echo is produced by the sequential application of 90° and 180° pulses

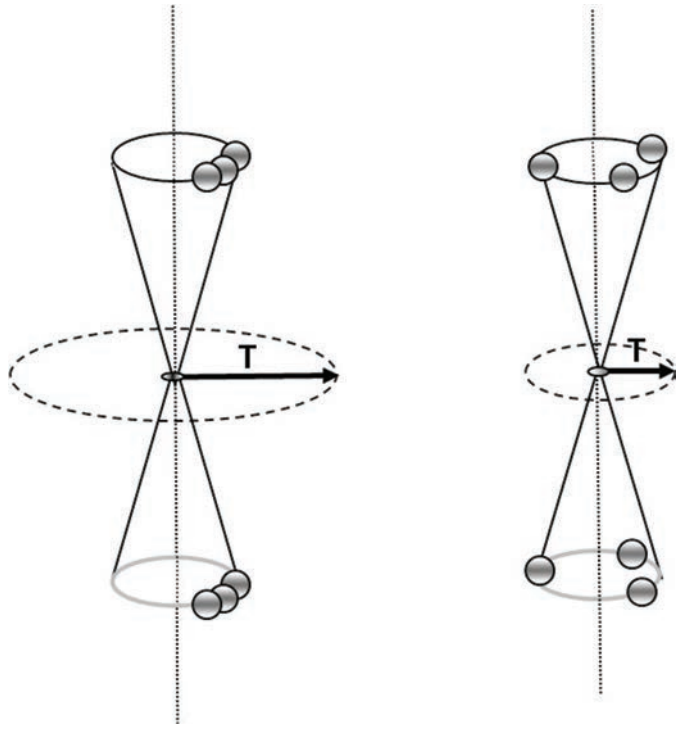

Figure 30.26 Precession

- Field echo is produced by symmetrically reversing the gradient fields
- Precession – The protons spin like a top and if they precess in phase they produce a signal. As the phase coherence is lost the signal decays. The time constant for the decay in transverse signal as phase coherence is lost is T2* (Figure 30.26.)
- T2* decay is a result of non-uniformities in the magnet as well as randomly fluctuating internal fields in the substance being scanned
- T2 relaxation time is the time constant for loss of transverse magnetisation resulting only from fluctuating fields in the substance
- T2 relaxation involves only loss of phase coherence, not loss of energy
- T2 tends to be long in tissues with highly mobile water molecules, e.g. in chondromalacia where there is loss of matrix structure – Hyperintense on T2 images
- Pulsed sequences can be chosen with different repetition or echo times
- TR = time to repetition of radiofrequency pulse (msec)
- Long TR times (>5 times T1) allow full T1 relaxation to occur before the next pulse
- With short TR times tissues with high T1 value (e.g. fat) appear bright
- TE = time to echo – From when the radiofrequency pulse stops to when the signal is measured (msec). Range 2–100 msec
- Short TE times are relatively insensitive to differences in T2

Summary

- **T1 relaxation time** = first order time constant for substance to become magnetized after being placed in a magnetic field, or to regain such magnetisation following a radiofrequency pulse. Energy is lost to the 'lattice' – The magnetic environment
- **T2 relaxation time** = first order time constant for loss of transverse magnetisation resulting from loss of phase coherence owing to local non-uniformities in the magnetic field
- **Tl-weighted images** – Short TR (<1000 msec), short TE (<60 msec)
- **T2-weighted images** – Long TR (> 1000 msec), long TE (TE >60 msec)
- **T2* gradient echo** – Variable TR, TE short (<60 msec), flip angle 10–80°
- **Proton density images** – TR long (>1000 ms), TE short (TE <60 ms)
- **STIR** – Short Tau inversion recovery
- **Fast spin (turbo) echo** – Can be used for T2 and PD images (T1 fast anyway)

Image interpretation

- T1-weighted images – **Fat bright,** subacute haemorrhage bright, free fluid dark, good anatomical detail
- T2-weighted images – **Fluid bright** – E.g. oedema
- Proton density – Good for menisci, low contrast
- T* gradient echo – Good for ligaments, articular cartilage and fibrocartilage
- Fast spin echo – Good for use in vicinity of metal prostheses

How to recognise type of sequences

- If fluid and fat both bright, probably T2-weighted
- If fluid bright and fat dark, probably fat-suppressed T2 (e.g. STIR)
- If fat bright and fluid dark, probably T1-weighted
- It should be noted that fat suppression sequences can be used with both T1 and T2 images
- If contrast low, may be PD

Nuclear medicine bone scans

Nuclear medicine scans involve the use of a radioisotope and a carrier substance that binds to the target tissue.

Technetium-99m scan

- This is the most commonly used 'bone scan'
- Tc^{99m} the 'm' indicates the metastable form of the isotope, which has a half life of 6 hours, compared with 200 000 years for the Tc^{99} isotope
- Technetium-99m is attached to MDP (methylene diphosphonate)
- The methylene diphosphonate interacts with the hydroxyapatite in bone to form insoluble technetium calcium phosphate complexes
- The technetium-99m emits gamma rays, which are detected with a gamma camera
- Uptake is found in areas of
  - Increased blood flow
  - Increased cellular activity with osteoid formation – In other words, osteoblastic activity
- Three phase scan
  - *First phase: Vascular phase*
    - 1–2 minutes; angiogram of blood in the arterial system
  - *Second phase: Blood pool*
    - After about 5 minutes; equilibrium of tracer throughout the extracellular space
    - Increased uptake in this phase indicates increased vascularization of the soft tissues (most commonly due to inflammation)
  - *Third phase: Delayed images – Bone phase*
    - After about 4 hours; tracer accumulates at sites of osteoblastic activity
- Potential false-negative results from technetium-99m scans:
  - Overwhelming bone destruction with no osteoblastic activity – Myeloma, thyroid and renal cell tumours, very aggressive secondary deposits – E.g. breast carcinoma
  - Superscan – Can be found with multiple secondaries (especially breast carcinoma). The entire skeleton is hot so can appear normal, but kidneys not seen as easily as normal (owing to lack of contrast between the normal increase in signal from the kidneys and that from the bone)
- Tc-99m scans can take more than a year to return to normal following hip replacements, and 18 months or more following knee replacements
- Tc-99m scans have high sensitivity but low specificity

Other radioisotopes

Gallium (67-citrate)

- Binds to plasma proteins
- Increased uptake found in areas of inflammation or neoplasia
- Less sensitive to blood flow than Tc-99 scans
- Delayed images at 24–48 hours required

Indium scan (indium-111-labelled white cells)

- Labelled white cells accumulate in areas of inflammation but not in areas of neoplasia

- Can be useful for diagnosing osteomyelitis or infection around joint replacements (but not completely reliable for this)

Radiolabelled monoclonal antibodies

- Labelling specific to particular cell lines, e.g. granulocytes

PET

- Positron emission tomography
- Specific radioisotopes, with short half lives (^{11}C, ^{15}O, ^{18}F), manufactured in a cyclotron
- When a positron is emitted by the isotope it interacts with an electron, to emit 2 gamma-rays at 180° to each other which can be detected by a detector
- Isotopes are used to label biologically active molecules that are introduced into the subject
- Good for active cells

SPECT

- Single photon emission computed tomography
- Tomographical images obtained by rotating a camera around the patient 360°. The images are then reconstructed in sagittal, coronal and axial planes
- Enhances sensitivity
- Improves anatomical localisation

Ultrasound

- Utilizes high frequency sound waves, which are reflected back from the tissues
- Sound waves are produced and received with a transducer containing a piezoelectric ceramic crystal
- Higher frequencies give higher resolution but greater signal attenuation so better for superficial tissues

Tourniquets

Tourniquets can be useful in providing a bloodless field but there are a number of complications that can arise from their use. Proper use of tourniquets is an important aspect of patient care.

Tourniquets may be non-pneumatic or pneumatic:

- Non-pneumatic tourniquets are only used for short operations on the digits
- Pneumatic tourniquets may be non-automatic or automatic. The non-automatic types have a hand-operated pump and a pressure gauge and they cannot compensate automatically for leaks in the system. The automatic type operate from either an air line or an electric pump
- Tourniquets not attached to a fixed air line carry a risk of being inadvertently left in place at the completion of surgery

Inflation pressure

- There is no absolute value for pressure of inflation; the surgeon should consider
  - Age of patient
  - Condition of the soft tissues
  - Intercurrent medical conditions (especially vascular pathology)
  - The circumference of the limb
- In the upper limb the inflation pressure should be 50 mmHg higher than the systolic pressure
- In the lower limb the inflation pressure should be double the systolic pressure

Contraindications

- Severe crushing injuries
- Sickle cell disease
- Peripheral vascular disease (relative)

Exsanguination

- Either by elevation or expression
- Expression should be avoided in the presence of venous thrombosis, malignancy or infection, all of which may be spread by embolism
- In frail patients cardiac arrest may occur from circulatory overload if both lower limbs are exsanguinated at the same time

Complications

Local

- Compression neurapraxia
- Bone and soft-tissue necrosis
- Direct vascular injury
- Postoperative swelling and stiffness
- Delayed recovery of muscle power
- Wound haematoma
- Wound infection

Systemic

- Cardiorespiratory decompensation
- Increased CVP
- Deep vein thrombosis
- Cerebral infarction
- Alterations in acid-base balance

Tourniquet paralysis syndrome

- Caused by cuff pressure rather than ischaemia
- Flaccid motor paralysis with sensory dissociation
- Pain sensation often altered although temperature appreciation is usually preserved
- Colour, skin temperature and peripheral pulses are usually normal

- EMG – Nerve conduction block at the level of the tourniquet
- May take up to 3 months to recover
- Nerves in patients with diabetes, alcoholism and rheumatoid arthritis have increased susceptibility

Post-tourniquet syndrome (tourniquet-induced skeletal muscle ischaemia)

This is a reperfusion injury and is due to ischaemia. After release of the tourniquet the following occur:

- Oedema
- Stiffness
- Pallor
- Weakness
- Subjective numbness

Myonephropathic metabolic syndrome

- Metabolic acidosis
- Hyperkalaemia
- Myoglobinuria
- Renal failure

Compartment syndrome

This is a very important topic and you *must* be able to explain the mechanism clearly.

Compartment syndrome is defined as increased pressure in an enclosed osteofascial space resulting in decreased capillary perfusion below that necessary for sustained tissue viability.

Normal compartment pressures are of the order of 5 mmHg.

Two possible mechanisms can precipitate compartment syndrome:

1. Increased content within the compartment – Haemorrhage, ischaemic swelling, reperfusion injury, etc
2. Decreased space – Tight cast, premature closure of fascia

The final common pathway involves:

1. Compartment pressure exceeds the venule/venous pressure
2. The venous outflow from the compartment is impaired and tissue ischaemia follows
3. Pressure within the compartment increases whilst arterial inflow is not impaired
4. As compartment pressure approaches systolic pressure, flow into the compartment will cease

NB. It is important to be aware that at a pressure well below arterial pressure there will be no perfusion of the tissues within the compartment, although at such a pressure there will still be a distal pulse.

Clinical presentation

- Pain out of proportion to the injury
- Pain on passive movement (pain on stretching the muscles of the compartment)
- Compartment palpably tight
- Paraesthesia
- Paralysis

Disproportionate pain, pain on passive movement and a tight compartment on palpation are the most important as all the others are *too late* and tissues may necrose even though a pulse is still present distally

- At 1 hour of ischaemia, a reversible neurapraxia develops
- At 8 hours of ischaemia, axonotmesis occurs

Measurement

Compartment syndrome is a clinical diagnosis except when pain cannot be assessed (e.g. in impaired consciousness or in the presence of regional anaesthesia), when a pressure monitor can be used.

- A catheter/needle and pressure transducer are used with simultaneous blood pressure measurement
- In trauma the measurement is taken within the zone of injury and should be undertaken in all relevant compartments
- The threshold can be an absolute value of 30 mmHg or pressure within 20–30 mmHg of diastolic blood pressure - Edinburgh group

Fasciotomies

- A diagnosis of acute compartment syndrome is a surgical emergency
- Leg compartments – Two-incision technique (anterolateral and medial) is used to decompress all four compartments
- Forearm – Volar decompression to include carpal tunnel, then check the dorsum and decompress if necessary
- Foot – Several different methods have been described, depending on the part of the foot affected (i.e. if calcaneal fracture or not). There is controversy over the best method of treating compartment syndrome in the foot. Nine compartments (possibly 10) have been described in the sole of the foot although it is not certain whether these are clinically and functionally separate. A three-incision technique has been described with two incisions dorsally and one medially but complications, including skin necrosis and below knee amputation, have been described, and some foot surgeons advise one incision only whilst others advocate a non-operative approach
- Closure – Not before 48 hours; there is a low threshold for skin grafts rather than delayed primary closure
- Late presentation – Once muscle necrosis is established there is no indication for fasciotomy, which may lead to rhabdomyolysis and infection. The definition of 'late' is controversial

Electrosurgery

- An electric circuit is made involving the patient, where the patient is the point of current resistance, generating heat

- Frequency chosen is above 100 kHz to avoid nerve and/or muscle stimulation
- Monopolar electrosurgery involves an active electrode (high current density) at the surgical site and a return electrode elsewhere on the patient. The return electrode must be of large surface area to reduce the current density and avoid burns
- Waveforms
  - 'Cut' – Involves continuous current to generate heat and vaporize tissue
  - 'Coag' – Involves intermittent current (on for <10% of the time) with less heat and this permits a coagulum to form. If this waveform is used to cut, higher voltages are required with more surrounding tissue damage
  - 'Blend' – Involves a longer 'on time' than coag
  - 'Fulguration' means the coagulation/charring of tissue over a wider area and employs a coag waveform with the diathermy point held slightly away from the tissue
- Safety
  - 'Grounded system' – Original technology; the risk of a return electrode being formed by patient contact with metal on the operating table resulting in iatrogenic skin burns
  - 'Isolated system' – The return electrode becomes the only route back to the generator so 'grounding' is no longer a risk
  - Return electrode placement should be over well-vascularized muscle mass. Most systems now monitor the impedance at the return electrode to reduce burn risk
  - Care should be taken with flammable prep solutions, which may soak into drapes and then catch fire – Alcohol burns without a visible flame
- Bipolar electrosurgery involves active and return point electrodes at the surgical site. The forceps points (electrodes) must be separated for current to pass through tissue. Advantage of bipolar – Avoids risk of damage from passage of current through surrounding tissues (particularly arteries in digits, etc), but still a risk of burns from alcohol-based prep solutions
- Note that, in electrocautery, direct current is used, in contrast to electrosurgery, which involves alternating current

Infection control

Two approaches are taken to address this issue:

- Reducing the size of the inoculum
- Enhancing the host defences

Reducing the inoculum

- Ward hygiene
- Screening/separation of infected cases
- Skin cleanliness (*not* antisepsis – As this encourages resistance)
- Theatre design and practice (see below)
- Limiting dressing changes

Enhancing host defences

- Good nutrition
- Antibiotic prophylaxis where appropriate
- Tetanus prophylaxis
- Optimize the skin preoperatively (e.g. psoriasis treatment, avoidance of blisters)
- Avoid unnecessary antibiotics (resistance)

Bacteria

Gram staining involves staining with crystal violet, fixing with iodine then washing with alcohol: Gram-positive retain dye; Gram-negative dye washes out and then re-stained with safranin O:

- Gram-positive cocci: Staphylococci, streptococci
  - Staphyloccoci may be coagulase-positive (*Staph. aureus*), or coagulase-negative (*Staph. epidermidis*)
  - Panton–Valentine leukocidin (PVL) is a toxin produced by *Staph. aureus,* which may cause necrotizing fasciitis
  - Streptococci may be alpha-haemolytic (*Strep. viridans, Strep. pneumoniae*), or beta-haemolytic (group A – *Strep. pyogenes,* group D – *Strep. faecalis*)
- Gram-negative cocci: *Neisseria*
- Gram-positive bacilli (rods): *E. coli, Proteus, Klebsiella, Pseudomonas*
- Gram-negative bacilli: *Clostridia* (*tetani, difficile, perfringens*)

Antibiotic actions

- Bacteriostatic
- Bacteriocidal
- Mixed
- Penicillin/cephalosporins – Prevent bacterial cell wall synthesis – Cell wall enzyme
- Glycopeptides (vancomycin, teicoplanin) – Interfere with cell wall enzyme
- Fucidin and clarithromycin – Block ribosomal peptides
- Linezolid – Inhibits protein synthesis

Bacterial resistance

Resistance acquired in one of two ways:

- Genetic – Resistance transferred via DNA — plasmids (small circles of double-stranded DNA), integrons and transposons
- Proteomic – Altered target site on bacterium or altered enzyme that is the target of drug actions

Skin flora

- Includes coagulase-negative *Staph. epidermidis, Staph. aureus* and Gram-positive diphtheroid bacilli. These are accessed by lipophilic antibiotics secreted in perspiration

MRSA

Acquired penicillin-binding protein PBP2a, encoded by gene *mecA*.

If found on screening swabs:

- 5 days intranasal mupirocin
- 4% chlorhexidine baths
- Re-swab and repeat if necessary

Treatment:

- Glycopeptides – Vancomycin and teicoplanin
- Oxazolidinones – Linezolid

Biofilms

- Biofilms are ubiquitous in nature and affect many other walks of life apart from orthopaedics
- Bacteria on surface of implants secrete a glycoprotein biofilm
- Within the film bacteria change from being planktonic to being surface attached community, in which many may be dormant
- This biofilm reduces access of antibiotics to bacteria (rifampicin has good penetration of biofilms)
- A proportion of bacteria within the biofilm are dormant so resistant to bactericidal antibiotics
- Bacteria within the biofilm can exchange resistance information
- Bacterial within a biofilm exhibit the phenomenon of **Quorum Sensing**, in which the members of the population are able to communicate with each other and to simultaneously transform into their planktonic versions when external conditions are favourable; this phenomenon explains recurrences of infection after long delays
- Biofilms may form within 3 weeks of initial infection and this will dictate methods used for treating periprosthetic infections, etc
- In most cases need to remove biofilm physically to allow adequate treatment of infection

Therapeutic index = effective concentration at site/minimum inhibitory concentration.

Instrument sterilisation methods

- Dry heat (ineffective, used for glass, liquid and powders)
- Moist head (under pressure, requires less heat for less time)
- UV light (surface sterilization only)
- Radiation (used commercially)
- Filtration (for sterilization of liquids)
- Gas (ethylene oxide – Slow)
- Liquid bath (4–8% glutaraldehyde, for heat-sensitive instruments – Carries risk of staff sensitisation)

Operating theatre design and practice

Source of pathogens

- Airborne contamination responsible for 95% of problem
- Floor is source of 15% of airborne contamination
- 98% of floorborne pathogens are from skin scales
- Axillae and groin heavily colonized – Most organisms from below level of neck

Theatre design[35]

- Separate preparation and disposal areas
- Four zones
  - Outer zone – Theatre reception and rest of hospital
  - Clean zone – Theatre reception to theatre doors
  - Aseptic zone – Theatre itself
  - Disposal zone
- Air flow
  - Plenum – Air pressure highest in preparation area > theatre > anaesthetic room > disposal room. Subject to turbulence and eddies
  - Laminar flow – Air moves at constant velocity along constant flow line with no turbulence
    - Horizontal
    - Vertical
  - Exponential (airflow like an inverted trumpet) – Minimizes entrainment of contaminated air from operating personnel
- Ultraclean air
  - HEPA (high efficiency particulate air filters) filter particles of 0.5 micron
  - Can achieve reduction to <10 colony forming units (CFU)/m^3 in centre of theatre
- Liddell showed reduction in deep sepsis by 50% with ultraclean air systems, a further 25% reduction with body exhaust suits, and 0.06% reduction with systemic antibiotics

Handwashing

- Bacterial counts are reduced by 99% with chlorhexidine, 97% with povidone-iodine
- Residual effect after time 97% with chlorhexidine, 90% with iodine

Theatre clothing

- Cotton clothing has pore sizes of 80 microns – Allow skin scales to pass through
- Single use non-woven clothing with spun-laced fibres impede bacterial passage but allows ventilation
- Gore-Tex® has very small pore size (0.2 micron) but is expensive

Orthotics and prosthetics

Orthotics is a subject often neglected during revision but is an important topic. Orthotics aspects may be incorporated into other topics, such as gait, hand injuries, etc.

Orthoses

Definition: *Orthotics are external devices that control the movement of segments of the body.*

Uses

Orthotics may be used to:

- Correct a flexible deformity
- Control motion at a joint
- Augment weak muscles
- Redistribute forces
- Relieve weight

Types of orthotics

Orthotics can be classified as *therapeutic* or *functional*:

Therapeutic orthotics

- Static
- Dynamic
- Static progressive – Serial casting
- Gravity drop out (upper limb)
- Articulated – Guide arc of motion

Static orthotics cannot be used to correct a fixed deformity.

Serial casting utilizes stress relaxation, a viscoelastic property of biological tissue.

Functional orthotics

- Stabilise proximal or distal joints
- Enable or maximize muscle action
- Assist movement
- Substitute for muscle action

Terminology

Orthotics are named by the joints that they cross

Upper limb:

- SO – Shoulder orthosis
- EO – Elbow orthosis
- EWHO – Elbow wrist hand orthosis
- WHO – Wrist hand orthosis
- HO – Hand orthosis

Lower limb:

- HO – Hip orthosis
- KO – Knee orthosis
- FO – Foot orthosis
- HKAFO – Hip knee ankle foot orthosis
- KAFO – Knee ankle foot orthosis
- AFO – Ankle foot orthosis

Spine:

- CO – Cervical orthosis
- LSO – Lumbosacral orthosis
- CTLSO – Cervical, thoracic, lumbosacral orthosis
- TLSO – Thoracic, lumbosacral orthosis

Orthotic materials

Orthotic materials need to be light, strong and sufficiently hard wearing to survive for the duration of their intended use.

Common materials include the following'

- ***Thermoplastic*** (low temperature thermoforming, e.g. Orthoplast)
  - Can remould to adjust, for example where there is pressure on bony prominences
  - Not as durable as thermosetting materials so usually used for temporary orthotics
- ***Thermoplastic*** (high temperature plastics – E.g. Polyproplylene)
  - setting temperatures above safe limit for contact with skin so made by taking a plaster of Paris mould and then fitting heated plastic to mould
- **Thermosetting materials** – composites, usually laminated fabric with resin. Example carbon fibre composites. Cannot be remoulded once made
- ***Leather*** – Traditional material, hard wearing and flexible, but skilled labour intensive so expensive
- Other materials, such as irons

Mechanism of action

Orthotics may use a number of different mechanism to achieve their effect. These are well illustrated by considering the different types of AFO in frequent use:

- ***Three-point pressure*** – Many orthotics use this principle, as used in casts for control of angulated fractures once they have been reduced. Applying force at three points enables a bending force to be applied to counter the intrinsic deforming force. To control a twisting force it is necessary to apply a turning couple, which requires a minimum of four points of application of force

A particular example of the three-point pressure principle is the use of irons, which fit into the heel of the shoe.- three point pressure is applied by the use of an iron and T-strap (e.g. inside iron and outside T-strap to apply a valgus force at the level of the ankle/hindfoot). The use of square sockets prevents ankle dorsiflexion and plantarflexion, round sockets allow such movement.

The same biomechanical principles as described for bones and joints apply to orthoses. Long lever arms help to minimize the contact pressures but increase the bulk and weight of the orthotic.

- ***Total contact*** – Total contact orthotics spread the loads across the entire surface
- ***Unweighting*** – The loads pass through the orthotic, thus, bypassing the joint – E.g. patellar tendon bearing AFO
- ***Immobilizing***
- ***Ground reaction*** – Ground-reaction orthotics are discussed below

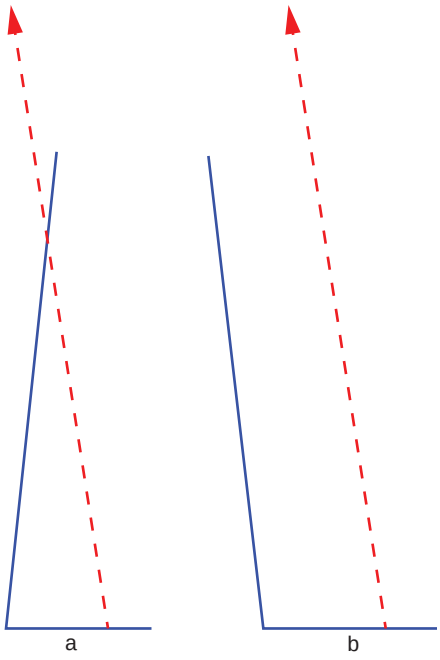

Figure 30.27 Ground reaction force orthotic

Ground-reaction orthotics

Three-point pressure orthotics must cross the joint being controlled. Ground-reaction orthotics control joint posture by positioning the ground-reaction force optimally; they do not cross the joint being controlled and are, therefore, smaller and lighter than three-point pressure orthotics. Ground-reaction orthotics suffer from the disadvantage that they are less reliable as changes in the slope of the ground or in, for example, trunk posture may reverse the effect. Examples of ground-reaction orthotics are:

- Shoe wedge – A lateral heel wedge will push the hindfoot into valgus, resulting in the ground-reaction force passing lateral to the axis of the subtalar joint, thus, helping to prevent the hindfoot going into varus
- Ground-reaction orthotics for controlling knee posture. In quads paralysis (usually from polio) the knee may collapse into flexion or may become hyperextended. By fixing the angle of the ankle the ground reaction force can be positioned anterior or posterior to the knee joint to encourage either flexion or extension; these orthotics utilize the principle of linked movement. Figure 30.27 illustrates the use of an AFO to control knee posture

Minimizing the risk of skin problems

Problems with orthotics are frequently at the orthotic–skin interface and it is important to understand the ways in which the interface pressures can be reduced:

- Maximizing the lever arm of the orthotic in relation to the lever arm of the deforming force
- Maximizing the surface area through which the forces are applied from the orthotic to the skin
- Maximizing the conformity between the orthotic and the underlying limb/trunk
- Minimizing pressure through unprotected bony prominences

The material at the interface should also be moisture-absorbant to avoid maceration of the skin.

Prosthetics

Prosthetics replace a body part. Things to consider:

- Functional vs cosmetic
- Suspension – Belt or socket
- Endoskeletal (modular) vs exoskeletal (formed plastic or wood)
- Limb-fitting for infants from 8 months (when they are starting to stand and can manage 2-handed play)
- Levels
  - Hip disarticulation
  - Transfemoral – Silicone Iceross® sockets
  - Through-knee – Problematic for artificial knee mechanism
  - Transtibial – Patellar tendon bearing, supracondylar suspension
  - Ankle disarticulation 'symes' – Silicone feet
  - Prosthetic feet – SACH (solid ankle cushion heel), multi-axis, dynamic response (carbon fibre springs)
  - Upper limb – Body powered (shoulder) or external power (myoelectric or switch)

If asked to describe a prosthetic think of the following components;

- Socket
- Suspension system
- Joint
- Terminal device/end effector

Wheelchair design

- Depends on daily use, fixed deformities, head/trunk control, environment
- Frame weight – Rigid/portable
- Wheel/tyres
- Back – Height, reclining or fixed
- Foot rests
- Seat cushion, trochanteric pads, scoliosis pads
- Strapping – Seatbelt, pelvis belts
- Ambulation – Hand-operated (requires good upper limb function), electric

Venous thromboembolism

You should make sure that you are familiar with the current NICE guidelines on DVT prophylaxis as this is likely to be a popular topic for discussion in the oral examination.

Epidemiology

- DVT occurs in 1/1000 of the general population but in up to 50% of lower limb arthroplasty patients without prophylaxis, but is usually asymptomatic
- Asymptomatic DVT is diagnosed by ultrasound, ^{125}I-fibrinogen or venography (the 'gold standard')
- Symptomatic DVT involves leg swelling and pain
- 90% of cases of pulmonary embolism are due to DVT but pulmonary embolism is a rare complication of DVT. Risk is increased 10 times by surgery or trauma
- Post-thrombotic syndrome (ulceration, dermatitis, chronic swelling) occurs in up to 10% of DVT patients within 10 years
- Routine prophylaxis is used to reduce the morbidity and mortality of thromboembolism; however, treatment of asymptomatic DVT has not been shown to be effective
- Aspirin is not as effective as heparins in reducing the risk of asymptomatic DVT
- Aspirin carries a lower relative risk of bleeding than the heparins (1.24 and 1.75, respectively)
- Fatal pulmonary embolism is catastrophic and, therefore, the most relevant endpoint

Risk factors

- Age – Exponential increase in risk
- Obesity – 3 times the risk
- Varicose veins – 1.5 times the risk
- Prior venous thromboembolism – 5% recurrence per annum, increased by surgery
- Thrombophilias – E.g. factor V Leiden, antiphospholipid syndrome
- 'Thrombotic states' – Neoplasia (7 times the risk), cardiac failure, recent myocardial infarction or cerebrovascular accident, infection, polycythaemia
- Combined oral contraceptive pill, hormone replacement therapy, high-dose progestogens
- Pregnancy – 10 times the risk
- Immobility – Bed rest for >3 days can increase risk 10 times
- Hospitalisation – 10 times the risk
- Anaesthesia – Risk associated with GA is twice the risk associated with a spinal

DVT/PE prophylaxis

DVT and PE are major risks for many orthopaedic operations but there is not universal agreement over certain aspects of treatment.

- Guidelines have been produced by NICE, AAOS, American College of Chest Physicians (ACCP), the BOA, etc. These guidelines continue to evolve as more research becomes available
- Increased bleeding risk has been found with all forms of chemical prophylaxis except aspirin
- One issue has been whether the goal of treatment is to minimize the incidence of deep vein thrombosis, or the incidence of symptomatic VTE. Current thinking is that the aim should be to minimize symptomatic VTE and bleeding complications
- Candidates should be familiar with the NICE guidelines and the BOA documents
- The most appropriate form of prophylaxis must be considered for all patients but the precise form of that prophylaxis will depend upon the relative risk of thromboembolism balanced against the risk of complications; in particular bleeding

Methods of prophylaxis can be classified into *mechanical* and *chemical.*

Mechanical methods

NICE guidelines recommend the use of mechanical methods from the day of admission. Options include:

- Anti-embolic stockings – Knee or thigh length
- Intermittent compression devices
- Foot impulse devices

Chemical methods

NICE guidelines are for chemical prophylaxis to be used (unless contraindicated) for 28–35 days

Meta-analysis results:

- Benefit in terms of reduction in DVT rates from the use of aspirin, LMWH and unfractionated heparin
- Benefit in terms of reduction in PE for all chemical methods in current use; the evidence is inconclusive on which regime is optimal
- All chemical regimes show a significant increase in the risk of major bleeding when compared with controls with no prophylaxis, in proportion to their effectiveness in preventing DVT

For every patient a DVT/PE risk assessment should be performed.

Where chemical prophylaxis is used in high risk patients treatment should be continued for four to six weeks.

Statistics

Although it is not necessary to have an in depth knowledge of specific statistical tests it is important to have sufficient understanding of statistics to be able to interpret and evaluate the claims made in research papers in the journals and claims made by manufacturers.

Statistics can be:

- ***Descriptive*** – Describes a population, study group, etc
- ***Inferential*** – Allows conclusions, or inferences, to be drawn about the populations from which samples have been drawn

Describing a set of data

There are numerous ways of describing a set of data. These methods depend upon the nature of the data that is available. The types of data will be described below. The measures used for describing the data will depend upon the type (level) of data involved. The tests used for drawing inferences will depend upon the type of data, the patterns of distribution of the data (normal or otherwise) and the questions being asked.

Types (levels) of data

- *Nominal* = Not ranked, just named (e.g. blue, green, red)
- *Ordinal* – Ranked (e.g. first, second, third . . ., or mild, moderate, severe)
- *Interval* (numerical)
  - Discreet integers (e.g. 1, 2, 3, 4 . . .)
  - Continuous (e.g. 0–100 and all decimals in between)
  - Ratio (this is a particular form of quantitative data in which there is an absolute zero

Although discreet and continuous data does not have to have an absolute zero it is only possible to compare proportions if there is such a zero – The advantage of ratio data is that they allow proportional comparisons between data – E.g. 6 is 50% larger than 4, etc.

- Nominal, Ordinal and Interval data describe increasingly high levels of data

Describing a population

When describing a set of data two important parameters are key to giving a mathematical meaning to the data set. These two parameters are the measure of central tendency and the measure of spread. The measures used to measure these parameters depend upon the level of data gathered.

The measure of central tendency

- **Mode** – The value that occurs with greatest frequency
  - Usually used for nominal data but can be used for all levels of data
  - Ignores extremes, can be used for all levels
- **Median** – The central value of the set of data; i.e. the value that has an equal number of values above and below it
  - Useful for ordinal data and skewed data
  - Ignores extremes, can be used for ordinal level or above
- **Mean** – The average
  - Used for interval data – Maximum use of information
  - Sensitive to extremes
  - Sensitive to skew
  - Cannot be used for nominal or ordinal data

Measures of spread/variability

The measure of central tendency will give an idea of the most common values but it is also necessary to describe how spread out the data are either side of the central area or values – The 'measure of dispersion'. The methods available again depend upon the level of data and the chosen measure of central tendency:

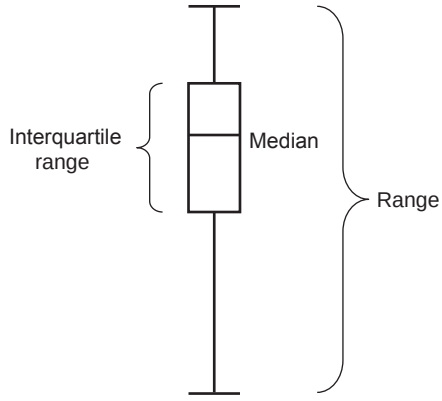

Figure 30.28 Box and Whisker plot

- **Variation ratio** – Used when the **mode** is the measure of central tendency. It is described as the percentage of values not at the modal value expressed as a percentage of the total values
- **Range**: Extreme values of the data set. The lowest and highest values of the data. The range does not give much information about the spread of data about the central value
- **Interquartile range (IQR)** – Often used when the **median** is the measure of central tendency. The first quartile point has one quarter of the data below it and the third quartile point has three quarters of the data below it so the interquartile range (from the first to third quartile points) contains half the data. When the median is used the spread is often expressed as a 'box and whisker' plot – With the box representing the IQR and the whisker the range. Figure 30.28 illustrate a Box and Whisker plot
- **Variance**: The measure of the spread, where the mean is the measure of the central tendency. Variance is the corrected sum of the squares about the mean of the data. The F-test is used to compare the variance of two populations. The advantage of using Variance to describe spread of data is that it distinguishes between a relatively 'smooth spread' either side of the mean and an 'irregular spread', which a simple mean deviation would not
- **Standard Deviation (SD)**: The square root of the variance – The Standard Deviation gives more information about the nature of the spread either side of the mean

The above measures of Central Tendency and Spread can be used as **descriptive statistics** to describe an entire population. Usually, however, entire populations cannot be measured and measurements are made on a sample drawn from the whole population. The question then arises as to how representative the sample is of the population from which it has been drawn, or if two samples are drawn from different populations; these questions are answered by **inferential statistics.**

Inferential statistics

A common sequence in performing a study is:

1. Take a sample of measurements
2. Describe the data in the sample mathematically (measure of central tendency and measure of spread
3. Take a further sample, or further samples
4. Describe the further sample or samples mathematically
5. Perform tests to determine whether the different samples were drawn from the same, or different, parent populations
6. Assess sources of error (Bias)
7. Draw conclusions

The following measures and terms are used in inferential statistics:

- **Standard Error of the Mean**
- Use Corrected SD – For calculating the SD of a sample use the variance squared divided by the number in the sample – 1 (because the variance of a sample is nearly always less than the variance of the population from which it is drawn)
- If multiple samples are taken from the population of data the means of each of the samples can be calculated and the Variance of these sample means can be measured
- The *Central Limit Theorem* shows that if multiple samples are taken from a population with any distribution (i.e. the distribution does not have to be Normal) the distribution of the sample means will be nearly Normal
- The SEM measures how closely a sample mean approximates the population mean. The SEM is a probabilistic statistic describing how far the sample mean probably differs from the sample mean
- The larger the sample size the more likely is the sample mean to match that of the mean of the population from which it has been drawn – With very large samples the SEM approaches 0
- The larger the sample size the more the SD of the sample will approach the SD of the population from which it is drawn
- SEM is calculated as the SD divided by the square root of the sample size
- It is important to realize that the Standard Deviation is a descriptive statistic that describes, *for a particular sample or population,* the distribution of the data about the mean. The Standard Error of the Mean, however, is a probabilistic statistic that describes how far the mean of a particular sample probably differs from the mean of the entire population of data from which that particular sample is drawn

Confidence intervals:

Confidence intervals are the ranges on either side of a sample mean indicating the probability that the true figure lies between these two values.

- Only applicable to normal distributions (parametric data)
- Confidence Intervals can be calculated from the Standard Errors of the Mean (SEM)
- Confidence intervals are equal to the values between the confidence limits and are a chosen number of Standard Errors of the Mean on either side of the mean
- For a large sample 95% confidence intervals are 1.96 SEM from the mean on either side
- When comparing two groups, if the 95% confidence intervals do not overlap there is a significant difference between the two study groups. If there is an overlap of up to 25% there may be a significant difference and this can be calculated using a two tailed t-test

Outcome measures

- Outcome measures need to be valid, reproducible, responsive to change, clinically relevant and easily measured
- Outcome measures may be primary or secondary, objective or subjective

Data interpretation

- **Null hypothesis:** That no difference exists between two groups (hence that any difference seen has occurred purely by chance). Tests including outcome measure are then employed to disprove the null hypothesis
- **Alternative hypothesis:** That there is a difference between the two groups
- ***P* value:** The probability that the difference seen has occurred by chance. The level of significance is usually set at a 5% probability ($P = 0.05$) that the difference was due to chance. Other levels can be set
- **Type I (α) error**: A false-positive result; incorrectly rejecting the null hypothesis, i.e. deciding there is a difference when there isn't one. Reduced by setting the *P* value smaller but then bigger sample sizes are needed to protect against a type II error
- **Type II (β) error**: A false-negative result, incorrectly accepting the null hypothesis, i.e. finding no difference when there is one. Increased risk if sample size too small or by setting the *P* value too small
- **Type III (γ) error**: Occurs when the researcher correctly rejects the null hypothesis but incorrectly attributes the cause. In other words the researcher misinterprets cause and effect

Power analysis

- **Power = 1–β**: The probability of demonstrating a true effect and correctly rejecting the null hypothesis i.e. *the ability of a study to detect a difference of a certain size*. The method of determining the number of subjects needed in a study to have a reasonable chance of showing a difference, if one exists. Usually set at 80%

Factors affecting power analysis:

- Significance level chosen (*P*-value)

- Sample size (power increases with increasing sample size)
- Variability in observations (power decreases with increasing variability)
- Size of the difference between the means considered to be the smallest acceptable difference
- Spread of the data
- Experimental design
- Type of data (parametric vs non-parametric)

The most important factors to consider in setting up a study are the variance of the subjects and the smallest difference that is considered relevant; hence, the value of pilot studies in establishing the variance.

- *It is essential to distinguish between Clinical Significance (how important a difference is clinically) and Statistical Significance (a mathematical calculation) – A result may be statistically significant but not clinically significant*

There are a large number of statistical tests. Although it is not necessary to know the details of how these tests work it is important to have an understanding of when different tests are appropriate.

Parametric and non-parametric tests

Parametric tests

- Used when the data is predicably distributed – This usually means *Normally distributed; i.e.* it is distributed on a Gaussian curve
- The *Kolmogorov–Smirnov* test can be used to analyse a set of data to assess whether it is drawn from a normally distributed population
- More powerful than non-parametric tests
- Observations must be independent
- Populations under study must have similar variance
- Examples include

Student's t-test for comparing the means of two samples:

- A one tailed t-test can be used if the alternate hypothesis allows a difference in only one direction (e.g. x is bigger than y) but one tailed tests should be used with caution
- A two tailed t-test is used if the alternate hypothesis allows a difference in both directions (e.g. x differs in size from y; it may be larger or smaller)
- For the two sample t-test to be used the variables must be independent
- If multiple comparisons are made with a t-test there is an increasing chance of a type II error (because with a *P*-value of 0.05 there is, by definition a 1 in 20 chance of an apparent difference between the two groups when none really exists. The chosen *P*-value should, therefore, be divided by the number of comparisons made (this is the **Bonferroni correction**)

ANOVA: Analysis of variance for comparing the means of two or more samples.

Chi-squared test to compare the difference between actual and expected frequencies (*contingency tables)*

Fisher's exact test – Also used with contingency tables – Useful when the numbers are small

Non-parametric tests

- Used when data is not normally distributed; e.g. when the data is skewed
- Less powerful than parametric tests
- Examples include the Mann–Whitney U test, the Wilcoxon Signed Rank test and the Kruskal–Wallis test
- Less likely to be find a significant difference between the groups
- No assumptions made about origins of the data
- Less likely to give type II errors
- Use rank order of value
- Cannot relate back to any parametric properties of the data

Transformation: A process by which non-parametric data are converted to a parametric form to permit more powerful analysis, e.g. logarithmic scale.

Note: Scoring systems (e.g. Harris Hip Score, etc) include ordinal data. Even if the final values appear to be continuous they remain non-parametric data and appropriate non-parametric tests must be used.

Some other terms and concepts that are useful are given below:

- **Accuracy:** How often the test is correct
- **Constructive Validity**: Evidence that a test measures what is intended to measure
- **Precision**: Repeatability of measurement
- **Incidence**: The rate of occurrence of new disease in a population previously free of the disease. It is found by dividing the number of *new* cases per year by the number of the population at risk
- **Prevalence**: The frequency of a disease at a given time. Found by dividing the number of *existing* cases by the number of the population at risk
- **Surveillance**: The study of trends in a population

Correlation and regression

Correlation coefficient (r): Measures the degree of association between two parameters and varies from complete association (= +1 or –1) to no association at all (= 0). NOTE a correlation coefficient is a *descriptive statistic* and does not establish cause and effect.

- Pearson's correlation coefficient – A measure of linear (parametric) association
- If one parameter increases as the other does, then the correlation is positive (and vice versa)
- If a curved line is needed to express the relationship then more complicated measures of correlation must be used, such as Spearman's rank for non-parametric data

- A correlation coefficient should always be accompanied by a scatter plot
- R^2 indicates what percentage of the variation in the one value is caused by the other

Regression: Once correlation is established, regression quantifies the correlation. it is the line drawn over the scatter plot, using the regression equation $y = a + bx$; regression coefficient = direction coefficient of the regression line.

- Interpolation – Measurements made on slope within data range
- Extrapolation – If the line is continued beyond the data range and the relationship is inferred – This should be done only with great caution

KAPPA analysis: Involves adjusting the observed proportion of agreement in relation to the proportion of agreement expected by chance and it is a measure of the repeatability of a test:

- 1 indicates complete agreement
- 0 indicates agreement could be expected purely by chance
- A negative value suggests systematic disagreement

Study types

Studies can be:

- *Retrospective* or *prospective*
- *Observational* or *experimental*
- *Cross-sectional* or *longitudinal*
- *Randomised* or *non-randomised*

Study types are commonly divided into:

- Case study
- Case series
- Case-control study
- Cross-sectional study
- Cohort study
- Randomised controlled trial (RCT)

Case series –– The outcomes of a group are reported but there is no comparison group. Weak.

Case-control study –– Retrospective form of a cohort study. Patients with an outcome of interest and a control group are followed backwards from some point in time to ascertain whether some early treatment or other exposure had a relationship to that outcome.

Cohort (longitudinal) study –– A cohort is a group of patients. In a cohort study two groups, one of which has undergone an intervention or treatment, are followed up over time in order to compare outcomes such as onset of disease or adverse events.

Cross-sectional studies –– Patients or events examined at one point in time.

Randomised controlled trial (RCT) –– The 'gold standard'. Groups of patients are randomised to receive or not to receive an intervention or treatment. The outcomes are compared in a prospective manner. The aim of the study and the hypothesis to be tested are clearly stated. Important features of RCTs include the following:

- **Randomization:** Avoids bias. Also ensures that all prognostic variables, both known and unknown, will be distributed 'fairly' between the study groups. Types include:
  - *Simple*: Treatment allocations assigned by computer-generated tables
  - *Block*: Treatment is allocated by blocks of set size. This ensures that equal number of patients are assigned to each treatment
  - *Stratified*: Ensure prognostic variables which are extremely important are equally distributed between the treatment groups
- **Inclusion/exclusion criteria**
- **Outcome measure:** Measures of outcome should be valid, reproducible and responsive to change
- **Bias**: A systematic source of error: Potential source of bias should be analysed and, where possible, eliminated. Bias will be discussed further below
- **Power analysis**
- **Ethical approval**
- **Informed consent**
- **Collection of data** and **results**
- **Analysis**
- **Conclusions**
- **Publication**: Important to know where the paper is intended to be published
- **Clinical significance vs statistical significance**
- **Masking/blinding**: This protects against bias. Can be single (patient) or double (patient and investigator)
- **Distorting influences**
  - Extraneous treatment
  - Contamination
  - Changes over time
  - Confounding factors; these are independent variables that interfere with the drawing of statistically valid conclusions from the study

Sequential analysis: Performed if there is a very important outcome, e.g. cancer. Two modalities are compared, power is determined, analysis is performed at predetermined points and the trial is stopped when statistical significance is reached

Equivalence study: Opposite to the null hypothesis. RCT where the two treatments are expected to have the same outcome. Hypothesis is that there is a difference

Intention to Treat Analysis (ITT)[36]: This is not an intuitive concept. Two of the problems that may arise during a RCT are non-compliance and missing outcomes. In an ITT the study subjects are treated as if they had the treatment that

they were randomly allocated to even if they did not receive the treatment or if the outcome is not known. ITT avoids misleading interpretation due to differential drop out from the groups being compared (see Bias section). ITT analyses are good for studying the effect of a treatment protocol as it reflects the real day-to-day situation more accurately than if these patient are excluded from the study and preserves the benefits of randomization

Survival analysis

This is a popular topic in the basic science oral.

Advantages

- Maximizes use of data
- Provides a graphical comparison of the survival (or failure to survive) of different groups over time
- Patients can be followed up for different lengths of time
- New patients can continuously be added to the analysis
- Useful when event being studied is relatively rare

Disadvantages

- More data is included in the left side of the plot (i.e. shorter follow-up) so the data towards the right side of the plot may become unreliable
- Usefulness depends on how failure is defined (e.g. radiological loosening, symptomatic loosening or revision in hip replacements)
- Measures only the time for a single event to occur

Types

- **Life table**: Survival probability is calculated at set time intervals. The graph can be recognised because it is stepped at regular intervals
- **Kaplan–Meier**: Survival probability is calculated when an event occurs. The graph can be recognised because the horizontal divisions or steps are at irregular time intervals

Underlying method

- Probability is calculated at each time or event interval as the probability of survival of *those at risk* – This is called a *conditional probability*
- To reach the final figure the conditional probabilities are multiplied together, i.e. the probability of surviving to point d is the probability all those surviving to point c surviving to point d times the probability of those surviving to point b surviving to point c times the probability of those surviving at point a surviving to point b, etc
- As new patients are continuously added to the left side of the plot the graph will need recalculating for all parts at each calculation point in time
- Patients who are lost to follow-up or who drop out of the study are **censored** (this is usually marked with a small tick or an asterix on the plot); their data is still used in the study up until the time that they are censored

Cautions when interpreting Kaplan–Meier plots

- As there are more patients in the shorter follow-up left side of the graph the data becomes less reliable to the right side of the graph – You should look for the confidence intervals
- If two study groups are being compared and the confidence intervals overlap you cannot conclude that there are any differences between them
- Never extrapolate beyond the right margin of the graph; to do so is invalid
- The outcomes for different groups can be analysed using the **Cox Proportional Hazards model (also called the Cox Regression model)** or the **Log Rank** test**.** These test is used for studies where 'time to event' is the measure being used. The Log Rank test will give a *P*-value that indicates the statistical significance of any difference between the groups. The Cox Proportional Hazards model will give a Hazard Ratio indicating the chance of something happening in one group divided by the chance in another group

Appraising a paper

It is essential to be able to appraise a paper logically and meaningfully.

Factors to consider when appraising a paper include:

- Research question – Was it valid?
- Study design – Was it appropriate to the research question?
- Study sample – Was it representative of the target population?
- Control group – If there was one was it acceptable?
- Sampling methods – Were they randomised and unbiased?
- Measurements and outcomes – Were they valid and relevant?
- Completeness – What was the compliance; what was the drop out rate; were there missing data?
- Distorting influences – Were there extraneous treatments; contamination; changes over time; other confounding factors?
- Conclusions – Were the conclusions related to the research questions posed in the introduction?
- Opinion – Was there a clear distinction between what could be concluded directly from the results presented and what was merely reporting the results of others

Levels of evidence

Different research groups have produced different lists of the levels of evidence. In the UK the most commonly used ranking is that produced by the Cochrane collaboration:

1. *Systematic reviews/meta analyses*
2. *Randomised controlled trials*
3. *Non-randomised well-controlled trials, time series, cohort studies, case-controlled studies*
4. *Non-experimental studies from >1 centre*
5. *Expert committees, descriptive studies*
6. *'Read it somewhere, someone told me'*

Bias

When studies are performed bias can be introduced at any stage. Bias refers to a *systematic* source of error (i.e. not random variations). Sources of bias can be considered under a number of different headings; many different classifications and terminologies are to be found. The following list illustrates common potential sources of bias:

- **Selection bias**
  - Choosing which subjects go into which group. Selection bias affects internal validity by producing confounding variables. This is minimised by careful recruitment and by randomisation with adequate concealment
  - There are many possible sources of selection bias
- **Exposure bias**
  - ***Intervention bias*** – There are other differences between the treatment received by the two study groups apart from the intended difference under study – 'co-treatment'
  - ***Compliance bias*** – Different compliances with treatment in the two groups
  - ***Wthdrawal bias*** – Different withdrawal rates or 'lost to follow up' rates in the two groups
- **Measurement bias**
  - Those performing the measurements are not completely blinded to the treatment given
- **Analysis bias**
  - Post-hoc significance bias – Significance levels chosen after data collected
  - Data mining without pre-determined hypothesis
- **Publication bias**
  - It can be easier to achieve publication of papers with positive results than those without. Publication bias can be assessed by *Eggers test* or by constructing **Inverted Funnel Plots**

Funnel plots

- Funnel plots are used to compare outcomes in groups of different sizes
- It was seen in the section on SEM that with increasing sample size the SEM approaches zero; where this does not occur it is likely that some effect is being observed. This can be used to detect 'outliers' in outcome studies, such as when comparing mortality rates for different surgical centres or surgeons
- Inverted funnel plots can be used to detect publication bias; the SEM should be larger for studies with smaller sample sizes – If it is not this may indicate publication bias. Figure 30.29 illustrates two inverted funnel plots; if there is an effect of the intervention there will be a shift of the data points to the left but there should still be a greater scatter of results for the studies with a larger SEM. The plot on the left shows no such increase in scatter with increasing SEM, suggesting publication bias

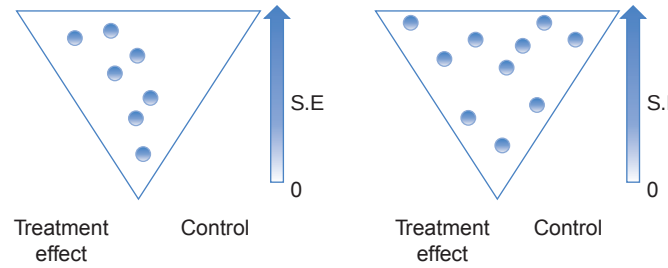

Figure 30.29 Inverted funnel plot – Testing for publication bias

Using and interpreting clinical tests

When a test is available it is important to be able to advise a patient on the meaning of the test result. The terms Sensitivity, Specificity, Positive Predictive Value and Negative Predictive Value are used to interpret the test results but they are often confused.

There are four possible outcomes for a test: True positive (a), false positive (b), false negative (c) and true negative (d).

Sensitivity: This measures how often a test will be positive if a patient has the condition it tests for. It is calculated by dividing the true positives by the total number with the condition (i.e. test positive/(true positives plus the false negatives): A/(a + c)

Specificity: This measures how often a test will be negative if a patient does not have the condition it tests for. It is calculated by dividing the number of true negatives by the total number who do not have the condition (i.e. test negative/(true negative + false positive): D/(d+b)

It can be seen that Sensitivity and Specificity are both test specific – They are seen from the point of view of someone who either does, or does not, have the condition – As a result they are not influenced by the frequency of false positives or negatives; they are not, therefore, influenced by the prevalence of the condition in the population

Positive Predictive Value (PPV): This measures how likely it is that someone with a positive test result actually as the disease. It is calculated by dividing the true positives by all who test positive: *A/(a+b)*

Negative Predictive Value (NPV): This measures how likely it is that someone who has a negative test result really does not have the condition. It is calculated by dividing the true negatives by all who test negative: *D/(c+d)*

PPV and NPV are seen from the point of view of someone who does, or does not, have a positive test result. For rare conditions a relatively high number of false positives may occur for every true positive and as a result PPV and NPV are influenced by the prevalence of the condition, particularly for rare conditions

Liklihood Ratio – This measures, given a test result (Table 30.7), how much more likely a patient is to have the condition than not have the condition. It is calculated by dividing the sensitivity by (1 – Specificity)

Table 30.7 Test results

Sensitivity =	*a/(a+c)*
Specificity =	*d/(d+b)*
Positive predictive value =	*a/(a+b)*
Negative predictive value =	*d/(c+d)*

Risk Ratio and Odds Ratio

These are used to compare a group with some intervention with another group without that intervention or with a different intervention.

Hazard = Instantaneous event rate = Probability of an event happening at any given time if has not happened so far. The word Hazard is derived from the name of a French game of chance and describes a danger that exists in the present

Risk = The probability that an event, or outcome, will occur *over a defined period of time*. It is calculated by dividing the number of events/outcomes by the number at risk at a fixed point in time (see discussion on Survivorship analysis). Risk *anticipates* a future danger whereas hazard refers to an immediate danger that already exists

Risk Ratio – Calculated by dividing the risk in one group by the risk in another group. A result of 1 means there is no difference in risk. The Confidence Interval can be calculated and if the 95% confidence interval does not include 1 then the difference is statistically significant

Odds = the number of times an event happens divided by the number of times it does not happen

Odds Ratio – Calculated by dividing the odds in one group by the odds in the comparator group. The statistical significance of any difference can be calculated in the same was as for Risk Ratio

When risk is very low the Risk Ratio and the Odds Ratio will be similar but for high frequency events they differ considerably

Absolute Risk Reduction (ARR) – The difference between the event/outcome rate in one group compared with that in another. The result is usually expressed as a percentage

Relative Risk Reduction (RRR) – The proportion by which an intervention reduces the event/outcome rate compared with the comparator group

Number Needed to Treat (NNT) – The number of patients needed to be treated to achieve a benefit in one. Calculated by dividing 1 by the ARR (or 100 by the ARR if ARR is expressed as a percentage)

Screening

Testing of asymptomatic people to look for disease/carrier status.

Criteria

- Valid
- Test acceptable – No harm to study group
- Specific, sensitive
- Natural history of the condition is known
- Early pick up – Leads to intervention
- Intervention – Leads to improve outcomes
- Potential yields, cost effective
- Incidence known

Setting up a research project

A popular question in the basic science oral is about how you would go about setting up a research project. The answer to this question should not focus solely on statistics but should start at the beginning and systematically work through the various stages of a project.

- Defining a research question
  - *Who/what* will be the subject of the study (inclusion and exclusion criteria)?
  - *What intervention* is planned?
  - Who or what will the study be compared with – I.e. what will be the *comparator group?*
  - What will be the *outcome data* that are collected? Will the data be quantitative or qualitative?
- Doing a literature search to ensure that the study has not already been done
- Deciding on the size of the study – This may need to involve a statistician and in some cases a pilot study to establish the variance of the population under study
- Deciding how the data will be analysed
- Finding funding for the study
- Ensuring that patient safety is ensured at all times
- Ensuring that ethical approval is obtained
- Planning a publication route to disseminate the results

Ethics and consent

This may appear at first sight to be a somewhat 'soft' topic and can easily be overlooked in preparation for the examination; yet it is on the curriculum and, therefore, 'fair game' as an examination topic.

Duties of care

The essential document to refer to is *Good Medical Practice*, published by the General Medical Council (GMC). This document sets out the duties and responsibilities of a doctor. It is available from the GMC website[a].

Consent

The reference document for issues relating to informed consent is *Consent: Patients and Doctors Making Decisions Together*, June 2008. This is also available from the GMC website.

- Information required for consent – You must explain:
  - What treatment aims to achieve

[a] www.gmc-uk.org

  - What treatment involves
  - What levels of pain are involved
  - If treatment involves serious risks
- If the patient insists that they do not want the information – You must:
  - Explain that they may not have given valid consent
  - *Record reasons* and be prepared to justify them
  - Advise that they can change mind
  - Advise that information can be given at any later stage
- The only other reason to withhold information is if it will cause serious harm to the patient – This means more than becoming upset or declining treatment
- You are required to inform patients if there is treatment likely to have greater potential benefit than the treatment you are able to give (either from another doctor or at another institution)
- You should usually discuss with the patient:
  - Complications (you must inform the patient of a chance of serious complication even if that chance is very small)
  - Side effects
  - Failure to achieve desired aim
- If there is a foreseeable possibility of future impairment of capacity, the patient can decide in advance:
  - Who they would like to be involved in future decisions
  - What procedures they would not want
  - Should be recorded in writing
  - Should be available to patient
  - Should be reviewable
- Mental Capacity Act 2005 (England and Wales)
  - Capacity is defined as the ability to:
    - Understand
    - Believe
    - Retain
    - Weigh up the information and
    - Communicate their wishes
- If a patient lacks capacity you should:
  - Consider whether the incapacity is temporary or permanent
  - Consider views expressed in advance by the patient
  - Consider views of legally appointed representative
  - Consider views of relatives, carers, etc, on what they consider to be the patient's preferences
  - Consider the views of the healthcare team on what they consider to be the patient's preferences

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Chapter 31

Applied basic science oral topics

Paul A. Banaszkiewicz and Stan Jones

The basic science viva is generally considered the most difficult of the four vivas stations to pass. Whilst on the day any viva can be failed, the basic science viva is straight on the money with requests to 'draw me the stress/strain curve' in the first sentence rather than the history/clinical examination default fallback that can be used in the adult and pathology viva station. Although the trauma viva may catch you out with the biomechanics of intramedullary nails or massive transfusion protocols, the familiarity of bread-and-butter fracture-clinic material daily encountered by trainees generally ups a candidates final viva mark and may prevent a failure. In addition the experience gained as a trainee working in an MTC should not be under estimated.

Candidate feedback from the second edition had asked for more worked through basic science examination corner material. We set about the challenge of writing an applied basic science chapter by including material that we felt represented the most common questions that regularly appeared in the exam.

It initially appeared a daunting task but at some point we realized that there were about 10 basic science topics that almost always got asked at each viva sitting, around 10 topics that were usually asked and another 10 or so topics that more often than not cropped up. In working through each of these topics we would hope that the chapter will give you a good half decent chance of at least scrapping through the viva with a pass.

One of the major difficulties with the basic sciences is that you can never fully predict how a particular viva topic will be asked in the exam. You can rehearse a set viva answer but this can all go pear-shaped as these viva question are about applying general principles your have learnt to particular situations, some lateral or out of the box thinking and invariably some thinking on your feet.

A useful starting point is to be familiar with the basic science exam syllabus as this can help to plan your revision timetable and ensure no parts of the syllabus are neglected or forgotten about:

1. **Anatomy**
    1. Clinical and functional anatomy with pathological and operative relevance
    2. Surgical approaches to the limbs and axial skeleton
    3. Embryology of musculoskeletal system
2. **Structure and function of connective tissue**
    1. Bone
    2. Cartilage – articular, meniscal
    3. Muscle and tendon
    4. Synovium
    5. Ligament
    6. Nerve
    7. Intervertebral disc
3. **Pathology**
    1. Thromboembolism and prophylaxis
    2. Principles of fracture healing
    3. Biology of wound healing
    4. Tendon and ligament injury and healing
    5. Nerve injury and regeneration
    6. Shock – Types, physiology, recognition and treatment
    7. Metabolism and hormonal regulation
    8. Metabolic and immunological response to trauma
    9. Blood loss in trauma/surgery, fluid balance and blood transfusion
    10. Osteoarthritis
    11. Osteoporosis
    12. Metabolic bone disease
    13. Rheumatoid arthritis and other arthropathies (inflammatory, crystal, etc)
    14. Haemophilia
    15. Inherited musculoskeletal disorders
    16. Osteonecrosis
    17. Osteochondritides
    18. Heterotopic ossification
    19. Infection of bone, joint, soft tissue, including tuberculosis, and their prophylaxis
    20. Prosthetic infection
    21. Surgery in high-risk and immunocompromised patients
4. **Prostheses and orthoses**
    1. Principles of design
    2. Prescription and fitting of standard prostheses
    3. Principles of orthotic bracing for control of disease, deformity and instability
5. **Pain**
    1. Anaesthesia – Principles and practice of local and regional anaesthesia and principles of general anaesthesia
    2. Pain management programmes and management of complex regional pain

3. Pain and pain relief
4. Behavioural dysfunction and somatisation

6. **Musculoskeletal oncology**
   1. Presentation, radiological features, pathological features, treatment and outcome for common benign and malignant tumours
   2. Principles of management of patients with metastatic bone disease in terms of investigation, prophylactic and definitive fixation of pathological fractures and oncological management
   3. Presenting features, management and outcome of soft-tissue swellings, including sarcomas
7. **Biomechanics and biomaterials**
   1. Bone grafts, bone banking and tissue transplantation
   2. Biomechanics of musculoskeletal tissues
   3. Biomechanics of fracture fixation
   4. Tribology of natural and artificial joints
   5. Design of implants and factors associated with implant failure (wear, loosening)
   6. Biomaterials
8. **Genetics and cell biology**
   1. Application/relevance of modern genetics to orthopaedic disease and treatment
   2. Molecular genetics and molecular biology in T&O
   3. Cell biology in T&O
   4. Cellular and molecular basis of wound healing
9. **Diagnostics**
   1. Musculoskeletal imaging: X-ray, contrast studies, CT, MR, ultrasound, radioisotope studies
   2. Assessment of bone mass and fracture risk
   3. Effects of radiation
   4. Blood tests
   5. Kinematics and gait analysis
10. **Clinical enviroment**

Theatre design

1. Design of theatres

Equipment design and use

1. Tourniquets
2. Sterilisation
3. Infection prevention and control
4. Patient warming methods and rationale
5. Skin preparation

Medical ethics

1. Duty of care
2. Informed consent

11. **Evidence management**

Data analysis

1. Data analysis and statistics – Principles and applications
2. Principles of epidemiology

Clinical trials

Design and conduct of clinical trials

12. **Quality improvement**
    1. Quality improvement projects including principles, methods and reporting
    2. Audit
    3. Clinical governance

Structure and function of connective tissue

Bone

The bone section is detailed and it is difficult to cut corners for high-yield topics as you also need an general understanding of the subject to feel confident when answering specific viva questions. In addition, a lot of the peripheral material is asked in the MCQ/EMI part 1.

A good starting point is to talk around a picture of bone structure (Figure 31.1). Score 8 candidates would be able to

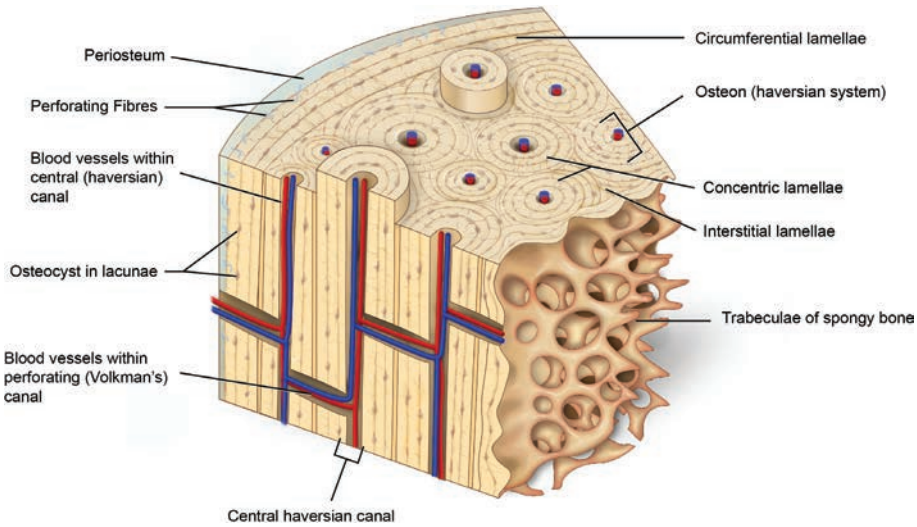


Figure 31.1 Cortical bone structure. Cortical bone is arranged in layers or lamellae concentrically around the centeral haversian canal which provide the vascular, lymphatic and nerve supply to the osteocytes via the canaliculi

talk about bone structure and function without any significant prompting for 5 minutes (if allowed). Score 6 candidates would need some prompting as they may dry up and not react fast enough when deciding which subtopic to switch to. A score 5 candidate may not have understood the topic in sufficient detail and as the viva progresses will get stuck when probed for more specific details. A score 4 candidate would not have read the topic in enough detail, be under rehearsed, hesitant and need multiple prompts. With bone composition it is more likely candidates will be asked to discuss the cellular components of bone and/or osteoclastogenesis rather than extracellular matrix although the structure and synthesis of type1 collagen in bone is fair game.

Examination corner

Basic science oral 1

EXAMINER: What factors inhibit osteoclast function?

CANDIDATE: I was not sure if the examiner had said osteoclasts or osteoblasts. I decided not to ask them to repeat the question and set about trying to remember the inhibitors of osteoclast function.

I mentioned calcitonin and oestrogen but the examiner seemed unimpressed and was looking I think for a big list of various hormones, growth factors and cytokines that could be reeled off verbatim. The only difficulty was that I didn't know any!

We ended up discussing vitamin D metabolism and again the examiner seemed unimpressed by my answers. Looking back at the question I think the examiners were after quite a complex answer, which involved the interplay between osteoblasts and osteocytes in bone resorption.

Bone resorption is primarily carried out by osteoclasts. The overall rate of osteoclastic bone resorption is regulated at two main levels: (1) determining the number of osteoclasts through the regulation of the osteoclastic precursor pool and its rate of differentiation, i.e. regulating osteoclastogenesis; (2) regulation of osteoclastic activity. Both involve activation of RANK signalling by its extracellular ligand RANKL and osteoprotegerin, a decoy RANK. Many of the previously known regulators of bone resorption, such as PTH, 1,25-$(OH)_2D_3$ or several interleukins, affect osteoclastic differentiation and/or function through their impact on RANKL or OPG production by stromal cells or osteoblasts and not, as initially thought, by a direct action on the osteoclast or its precusors. The only known hormone that directly interacts with osteoclasts is calcitonin but this hormone is thought to have only a minor role in bone turnover.

Basic science oral 2: Osteoclast bone resorption (Figure 31.2)

A typical osteoclast develops a ruffled border that increases its surface area and a sealing zone at the plasma membrane. The cytoplasm adjacent to the ruffled border is devoid of cell organelles. Bone is resorbed in a hollowed pit (Howships lacuna). A low PH enviroment is created through the production of acid and the transport of protons into the Howships lacuna that helps dissolves the inorganic apatite matrix. The organic matrix is removed by preoteolytic digestion through the activity of the collagenases produced in lysosomes which fuse with the ruffled border and thereby release their contents into the lacuna. Lysosomal enzyme cathepsin K degrades type 1 collagen and other non-collagen proteins. Mutation of cathepsin K leads to pycnodysostosis, a rare autosomal recessive skeletal dysplasia in which osteoclast function is defective. It is characterized by short stature, osteopetrosis, acroosteolysis (resorption of distal phalanges), spondylolysis, delayed cranial suture closure, and bone fragility.

Basic science oral 3

EXAMINER: Describe the structure of collagen in bone?

CANDIDATE: Composed primarily (90%) of type I collagen (b-one) which consists of a triple helix of two alpha-1 chains and one alpha-2 chain arranged in a quarter staggered structural array producing single fibrils. Collagen is responsible for the tensile strength of bone. Mineral deposition (calcification) occurs in the hole zones that exist between the ends of fibrils and the pore zones that lie between the sides of fibrils of collagen. Cross-linking increases the tensile strength of collagen.

Basic science oral 4

EXAMINER: (*Laminated picture handed to candidate*; Figure 31.1) What is this?

CANDIDATE: This picture demonstrates cortical lamellar bone structure. The basic structural unit of cortical bone is the osteon or Haversian system. Each osteon is an elongated cylinder running parallel to the long axis of the bone. The central (Haversian) canals run longitudinally through the bone that serves as a passageway for blood vessels and nerves. Perforating, or Volkmann's, canals lie at right angles to the long axis of the bone, and connect the blood and nerve supply of the periosteum to that of the central canals and medullary cavity. Osteocytes occupy lacunae at the junctions of the lamellae, and are connected to each other and the central canal via a series of hair-like channels, canaliculi.

Circumferential lamellae are located just beneath the periosteum, extending around the entire circumference of the bone, while interstitial lamellae are incomplete or fragmented osteons that lie between intact osteons, filling the spaces in between. They represent the remnant osteons left from partial resorption of old osteons during bone remodelling.

COMMENT: This viva question is about the structure of bone. Practice talking around the diagram for 5 minutes. This should polish up your answering skills for any possible exam viva question on the subject

Basic science oral 5

EXAMINER: What cells are found in bone and what is their function?

CANDIDATE: The five primary cell types in bone tissue are: Osteocytes, osteoblasts, osteoclasts, bone lining cells and osteoprogenitor cells.

Osteoclasts are multinucleated cells of haematopoietic origin that degrade and resorb bone. Osteoclasts are generated from myeloid progenitors through a progression that involves the fusion of mononuclear precursor cells.

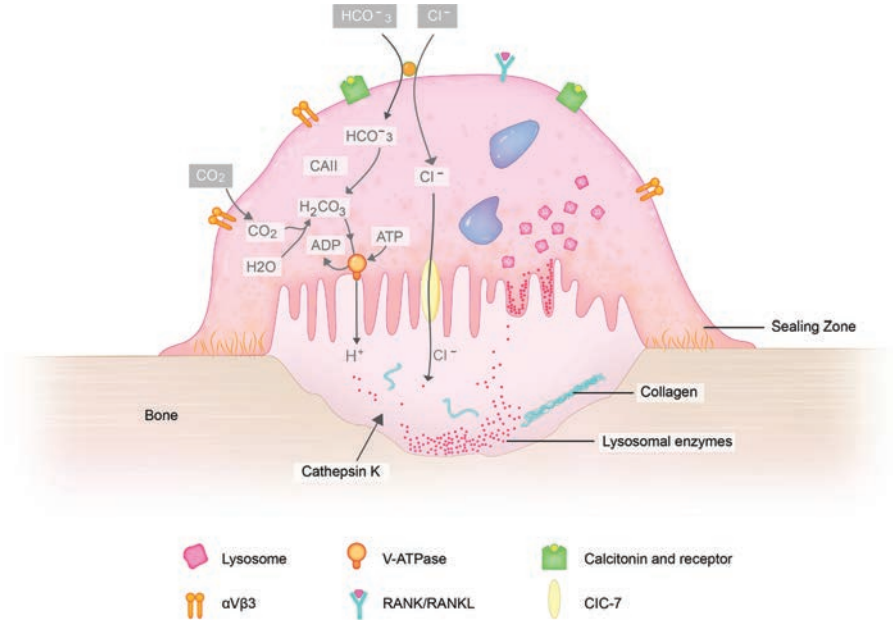


Figure 31.2 Osteoclast bone resorption. Osteoclasts form a seal around the resorptive compartment. The positioning of the sealing zone is mediated by integrins expressed on the osteoclast surface

Osteoprogenitor cells derive from mesenchymal stem cells and are the precusor cells for osteoblasts under conditions of low strain and increased oxygen tension.

Osteocytes are former osteoblasts surrounded by newly formed matrix and are important for tissue maintenance. Osteocyte communication plays a critical role in the response of the skeleton to mechanical loading.

Osteoblasts are found on the surface of bone and form mineralized bone matrix.

Bone lining cells are flat cells lining the surface of bone. They respond to mechanical forces on the bone and maintain the bones enviroment through synthesis and resorptive-like activities.

EXAMINER: What do we mean by osteoclastogenesis?

CANDIDATE: Osteoclast formation and activity is mediated by osteoblasts, which have receptors for hormones involved in bone remodelling (PTH, vitamin D). The production and activity of osteoclasts is controlled by two cytokines produced by osteoblasts: (1) monocyte colony-stimulating factor (M-CSF); and (2) RANKL.

M-CSF stimulates proliferation and osteoclastogenesis (fusion of monocytes and diffentiation).

RANK is found on osteoclast progenitor cells and mature osteoclasts. The identification of RANK–RANKL signalling as the main signal regulating osteoclast differentiation and activity was a major breakthrough in the bone biology field.

Osteoblasts also secrete osteoprotegerin (OPG), a molecule that blocks RANKL–RANK interaction and, therefore, inhibits osteoclast formation and activity. Discussion of RUNX2 gene in regulation of osteoblast function. Discussion of bisphosphonates and inhibitory action on osteoclasts.

EXAMINER: What receptors are present on osteoblasts?

CANDIDATE: Receptors found on osteoblasts include:

PTH receptor

- Stimulates alkaline phosphatase and type I collagen production

1,25 dihydroxyvitamin D receptor

- Stimulates matrix and alkaline phosphatase synthesis and production of bone specific proteins (osteocalcin)

Oestrogen receptor

- Inhibits bone resorption and stimulates bone production by inhibiting adenylyl cyclase

Glucocorticoid receptor

- Inhibit collagen and bone matrix production

Prostaglandins

- Stimulates bone resorption by activating adenylyl cyclase

COMMENT: This question is about the cellular components of bone and osteoclastogenesis. There is enough material for the examiners to stay on this topic particularly if osteoclastic bone resorption is included or it could be asked more superficially as part of a bigger section on the structure of bone.

Basic science oral 6

EXAMINER: Explain how an osteon is formed.

CANDIDATE: The chief structural unit of compact bone is an osteon, which consists of concentric bone layers called lamellae which surround a Haversian canal. Osteons are formations characteristic of mature bone and take shape during the process of bone remodelling. New bone may also take this structure as it forms, in which case the structure is called a primary osteon.

COMMENT: This is not quite the straightforward 'describe the structure of cortical bone' question it has a different twist to it.

EXAMINER: How does remodelling of bone occur?

Table 31.1 Components of bone matrix

Cells
• Osteocytes
• Osteoblasts
• Osteoclasts
Extracellular matrix
Organic (35%)
Collagen (type I) 90%
Matrix proteins(non-collagenous) • Osteocalcin, osteonectin, osteopontin
Proteoglycans
Growth factors and cytokines
Inorganic (65%)
Primarily hydroxyapatite $Ca_5(PO_4)_3(OH)_2$

CANDIDATE: Cortical bone remodelling occurs by osteoclastic tunnelling (cutting cones). The osteoclasts dissolve the inorganic matrix by acid secretion and as they move forwards the resorption cavity is occupied by osteoblasts that lay down osteoid before it is calcified.

Basic science oral 7: Blood supply of a long bone

This is another fairly common question. Break it down into the three systems: (1) nutrient artery (high pressure); (2) metaphyseal–epiphyseal system; and (3) periosteal (low pressure) system.

There is not enough info here for a full 5 minute viva particularly for good score 8 candidates so this topic will be part of a wider bone-related topic:

Describe the blood flow changes that occur with fracture healing?

This may lead into a discussion on the stages of fracture healing and then possibly into general management principles for a non-union

Discuss the changes that occur with blood flow after IM nailing of a long bone?

Basic science oral 8

Candidates may be asked about bone composition (Table 31.1). This should be relatively straightforward, although extracellular matrix organic components can be slightly confusing as different textbook give slightly different lists.

Growth plate

This is a very popular topic and candidates will need to practice drawing out the growth plate whilst simultaneously describing its components. There are a number of different questions that lead on from this.

EXAMINER: Can you draw and describe the physis for me as you go along?

COMMENT: Alternatively a candidate will be given a laminated histological photograph of a growth plate and be asked to describe in detail (Figure 31.3).

CANDIDATE: The basic histological appearance of the physis is demonstrated, with the top of the diagram nearest the joint surface.

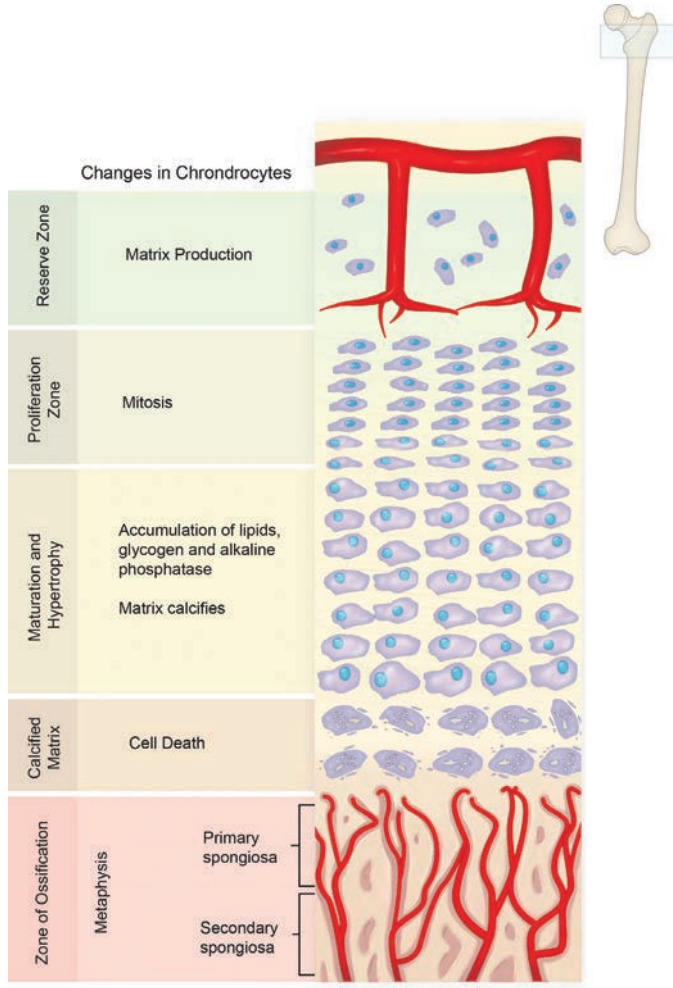


Figure 31.3 Typical growth plate. The epiphyseal plate is composed of five zones of activity

COMMENT: We have seen candidates describe the diagram in the opposite direction[a].

The epiphyseal plate (or physeal plate, physis or growth plate) is a hyaline cartilage plate. It is an area of developing tissue located between the epiphysis and the metaphysis. It can be divided into a number of zones. It is important to learn a description of each cell layer and be aware if you are talking about *cell function* or *morphology* or both. The cartilage

[a] Postgraduate Orthopaedics Sept 2015 viva day. Two candidates described a histological laminated photograph of the growth plate backwards i.e. mixing up the secondary spongiosa with the reserve zone. Both candidates were allowed to continue talking for about 20 seconds before the mistake was pointed out. Both recovered well but in general terms this does not inspire confidence. The 'catch out' was to hand the diagram to the candidates the wrong way round. This was unintentional. In the real exam the consequences may depend on how long an examiner allows a candidate to continue talking before they point the mistake out. In theory this type of mistake should be pointed out almost immediately as the examiners are not there to catch you out.

cells follow a cycle that applies to most cartilage cells in the body: Rest, proliferation, hypertrophy and programme cell death (apoptosis). Matrix synthesis occurs throughout with variations in cell content according to cell layer. The chondrocyte is the only cell present in the growth plate and is responsible for the synthesis, maintenance and breakdown of the collagen and extracellular matrix.

Zones of the growth plate[b]:

- Reserve zone
- Proliferative zone
- Hypertrophic zone
  - Maturation zone
  - Degenerative zone
  - Zone of provisional calcification
- Zone of vascular invasion (primary spongiosa)
- Secondary spongiosa

EXAMINER: Which layer of the physis is affected in a physeal fracture?

CANDIDATE: The provisional calcification zone is thought to be where a physeal fracture occurs although fractures are rarely limited to just one zone only. Despite this zone being strengthened by the mineralisation process it is still relatively weak as the extracellular matrix content is less due to chondrocyte hypertrophy.

EXAMINER: Describe the Salter–Harris classification for grading physeal fractures[c,d]. Draw the Salter–Harris fracture classification.

COMMENT: If you are asked to describe the Salter–Harris classification offer to draw it out as it is much quicker and easier to explain when using a diagram.

CANDIDATE: The Salter–Harris classification categorizes fractures according to their involvement of the epiphysis, physis and metaphysis.

Type I fractures are transverse entirely within the physis without involvement of the epiphysis or metaphysis.

Type II fractures involve part of the physis for a variable distance and then exit through the metaphysis. This produces a triangular shaped fragment of metaphysis often referred to as Thurston Holland's fragment.

Type III fractures are intra-articular extending from the physis into the epiphysis. This type of fracture generally occurs when the growth plate is partially fused.

Type IV fractures extends from the joint surface, crosses the physis and exits via the metaphysis. These fractures can interfere with normal growth and if displaced require ORIF.

Type V fractures are often an unrecognised crush injury with initially normal radiographs. The force is applied through the epiphysis to one area of the epiphyseal plate.

Several modifications to this classification have occurred. Ogden[1] added 6–9 subtypes.

Type VI: An injury to the periphery of the physis with stripping of the perichondral ring that allows bone bridging between the metaphysis and epiphysis.

Type VII: Isolated osteochondral injury of the epiphysis.

Type VIII: Isolated injury of the juxtaphyseal metaphysis with impairment of vascular supply and ossification.

Type IX: Injury of diaphyseal or metaphyseal periosteum with or without a fracture, compromising vascular supply to physis.

Peterson[2] also described two additional types:

1. Peterson I – A transverse metaphyseal fracture with longitudinal extension into the physis
2. Peterson II – Open fracture with partial physeal loss

COMMENT: Candidates are generally not asked these further classifications and the usual situation is that a candidate will volunteer them. Do not mention if you are only vaguely familiar as you then present the examiners with a good oportunity to probe you further.

EXAMAINER: What are Harris growth arrest lines?

CANDIDATE: They are thought to represent temporarily slowing or cessation of growth. They are transverse metaphyseal condensations of normal bone caused by a transverse rather than longitudinal, orientation of trabeculae within the primary spongiosa during periods of slowed or arrested longitudinal growth. If normal growth resumes, the line should appear to move away from the physis with time.

EXAMINER: How does rickets affect the physis?

CANDIDATE: In rickets there is failure of mineralisation leading to changes in the physis in the **zone of provisional calcification** and the bone. This results in increased width and disorientation of the physis since little or no provisional calcification occurs, and cortical thinning with bowing of the bone.

EXAMINER: How is the physis affected in the case of achondroplasia?

CANDIDATE: In achondroplasia there is a physeal dysplasia involving the cartilaginous **proliferative zone** of the physis with reduced chondrocyte proliferation and column formation. The genetic defect resulting from a single amino acid substitution alters the production of fibroblastic growth factor 3. As a result limbs are short although the trunk is of normal size.

EXAMINER: What other conditions affect the physis and how? What diseases affect the growth plate?

Growth is intensively anabolic with the production of cells that then produce structures composed of proteins, proteoglycans and non-protein substances. Disorders of growth, therefore, represent problems in cell multiplication and differentiation or in the formation of elements of structure[3].

CANDIDATE: The following diseases affect the physis (Table 31.2):

Lysosomal storage disorders: Reduced oxygen tension in the reserve zone.

Mucopolysaccharide disorders: Lead to chondrocyte degeneration in the hypertrophic zone.

Diastrophic dwarfism is possibly due to a type II collagen synthesis disorder affecting the reserve zone in addition to defects in other zones.

Effect of hormones and growth factors

The growth plate is affected by:

- Hormones (growth hormone, thyroxine, insulin, PTH, calcitonin)

[b] Described in detail in Chapter 29.

[c] If all else fails SMACK- **S**lipped (type I) **M**etaphyseal (type II) **A**rticular-epiphyseal (type III) **C**omplete-metaphysis and epiphysis (type IV) **K**rushed! (type V).

[d] With the children or fracture viva, candidates may end up hovering around this topic.

Table 31.2 Zone structure of the growth plate

Reserve zone	• Attaches growth plate to epiphyseal bone • Storage of lipids, glycogen, and proteoglycan aggregates for later growth and matrix production • Low oxygen tension	• Diastrophic dwarfism, Pseudo-achondroplasia, Gaucher's disease
Proliferative zone	• Proliferation of chondrocytes with longitudinal growth and stacking of chondrocytes. • Highest rate of extracellular matrix production • Increased oxygen tension in surroundings inhibits calcification	• Achondroplasia, gigantism, malnutrition, irradiation
Hypertrophic zone • zone of maturation	• Chondrocytes become swollen and vacuolated • Cellular division stopped • Marked decrease in type II collagen synthesis	• Mucopolysaccharidosis (Hurler syndrome, Morquio syndrome)
Hypertrophic zone • zone of degeneration	• Chondrocytes show metabolic changes of impending cell death • Proteoglycans are degraded • Low oxygen tension	• Mucopolysaccharidosis (Hurler syndrome, Morquio syndrome)
Hypertrophic zone • zone of provisional calcification	• Formed from last 2–3 cells in the column • Chondrocyte death increases alkaline phosphatase enzyme activity • Release of calcium mineralizes cartilage matrix	• Rickets, osteomalacia, SUFE
Primary spongiosa	• Vascular invasion and resorption of transverse septa • Bone formation	• Metaphyseal chondroplasia, acute oteomyelitis
Secondary spongiosa	• Remodelling • High oxygen tension	• Ostopetrosis (abnormality of osteoclasts, internal remodelling), osteogenesis imperfecta, scurvy, metaphyseal dysplasia

- Growth factors (TGF-β, PDGF, EGF and FGF)
- Vitamins (Vitamins A, C and D)

These factors influence chondrocyte proliferation and maturation and matrix synthesis and mineralisation. Some factors have a specific effect on a particular zone whilst others affect the entire growth plate.

Reserve zone

- Parathyroid hormone, IL-1

Proliferative zone

- Thyroxine, TGFβ, growth hormone, insulin

Hypertrophic one

- TGFβ, BDGF, vitamin D, calcitonin

Specific hormone effects

Thyroxine – Increases DNA synthesis in cells in the proliferative zone. Stimulates cell maturation
Parathyroid hormone – Stimulates chondrocyte proliferation and causes an increase in proteoglycan synthesis
Calcitonin – Accelerates growth plate calcification and cellular maturation
Glucocorticoids – Decrease proliferation of chondroprogenitor cells
Growth hormone – Stimulates cellular proliferation and production proteoglycans
Vitamin D – Vitamin deficiency results in an elongation of the cell columns in the growth plate and a decrease in mineralisation

Basic science oral 1

'Draw and describe the growth plate.'

Just like 'Sit down and draw articular cartilage', this is a top-10 high-yield topic for the basic science oral and is an absolutely classic question favoured by examiners. Practise drawing it out and explaining what you are drawing beforehand; it can be surprisingly easy to lose one's way when explaining out loud what you are drawing to someone.

Basic science oral 2

'Draw the growth plate.'

- Discussion of cell maturation through the layers of the growth plate

- Effect of various hormones on the growth plate
- Location of various disease processors in the growth plate: SUFE, rickets, etc (see above)

Basic science oral 3
Histology picture of the growth plate.

EXAMINER: What is this? Can you name the various parts?
EXAMINER: What are the resting cells?
CANDIDATE: Pluripotent stem cells.
EXAMINER: Where does the blood supply come for this layer?
EXAMINER: What is the pinkish staining in this layer between the cells?
EXAMINER: Why are the cells hypertrophic in this layer (hypertrophic layer)?
CANDIDATE: I wasn't sure. I said because they are rich in stored material probably glycogen granules.
EXAMINER: What happens to cells after hypertrophy?
EXAMINER: Where do fractures occur and why?

Basic science oral 4
Electron micrograph picture of the growth plate.

EXAMINER: What is the structure the picture is demonstrating?
CANDIDATE: The growth plate.
EXAMINER: Can you point out the various layers?
COMMENT: A bit trickier than expected. Slightly different in appearance than the classic textbook drawings. Halfway through I got a bit lost but managed to recover with some prompting. Not asked anything else and we quickly moved on to another topic.

Basic science oral 5
Draw and explain the architecture of the physis and its relation to fractures.

Basic science oral 6
Growth plate and its blood supply.

Cartilage – Articular, meniscal

Articular cartilage

This is an A-list topic. The challenge for the FRCS (Tr & Orth) exam board is how to re-invent the same question in a different way for each diet of exams.

Candidates may just be handed a laminated photograph of articular cartilage at the beginning of the viva question or be asked a couple of warm up questions before being asked to draw it out.

EXAMINER: What are the functions of articular cartilage?
CANDIDATE: The functions are

- To resist the compressive forces encountered across the joint under loading (shock absorber)
- To provide a smooth low friction surface for joint motion

EXAMINER: Can you draw the histological appearance of articular cartilage?
Draw the structure of articular cartilage[e].
What are the articular cartilage layers?
COMMENT: Candidates should be able to draw out the various layers of articular cartilage. Candiates may then be asked to explain why the layers appear like this, with reference to the three-dimensional ultrastructure[f].
CANDIDATE: The histological appearance of articular cartilage would be . . .
COMMENT: Concentrate on the: (1) differing orientation of type II collagen fibrils; (2) orientation and cellular features of the chondrocytes.

Structure. The histological structure can be divided into zones.

Superficial (tangential) zone– 10–20% of thickness

- Provides resistance to stress
- High concentration of collagen fibres arranged **parallel** to the joint surface forming a dense mat
- The most superficial part is called the lamina splendens and containing no cells, a clear film of collagen fibrils with little proteogylcan. This dense collagen arrangement probably reduces leakage of proteoglycans from the articular surface and protects it from the effect of harmful enzymes
- Below this is a cellular layer with chondrocytes parallel to surface, flat shaped, high density, many cells one to three cells thick
- Good resistance to shear forces due to tangential arrangement, greatest tensile strength
- Low metabolic activity, hence, low healing potential
- Thinnest layer with the highest concentration of collagen and the lowest concentration of proteoglycan
- High concentration of water at that can be squeezed out to help create lubrication

Middle (transitional) zone – 40–60% of thickness

- Provides resistance to compression
- Collagen fibres arranged obliquely at right angles to each other
- High concentration of proteoglycan
- Chondrocytes arranged in random orientation, round shape, progressively lower density and fewer cells
- Lowest concentration of water

[e] A score 8 candidate would be able to continually talk away describing each layer (and fast as well) with the examiners keeping silent, not interrupting and pleased that all the hard work is being done instead of them having to pull out and extract the info from the candidate. A score 6 candidate would discuss enough detail of each layer for a safe pass but need a little prompting from time to time. A score 4 or 5 candidate would struggled with naming the layers, structure and function and require a lot of prompting giving the impression they hadn't really learnt the subject.

[f] If you are proactive you should be able to draw and talk at the same time so practice this beforehand. If you are given a laminated diagram you have to get on the money straightway and keep talking.

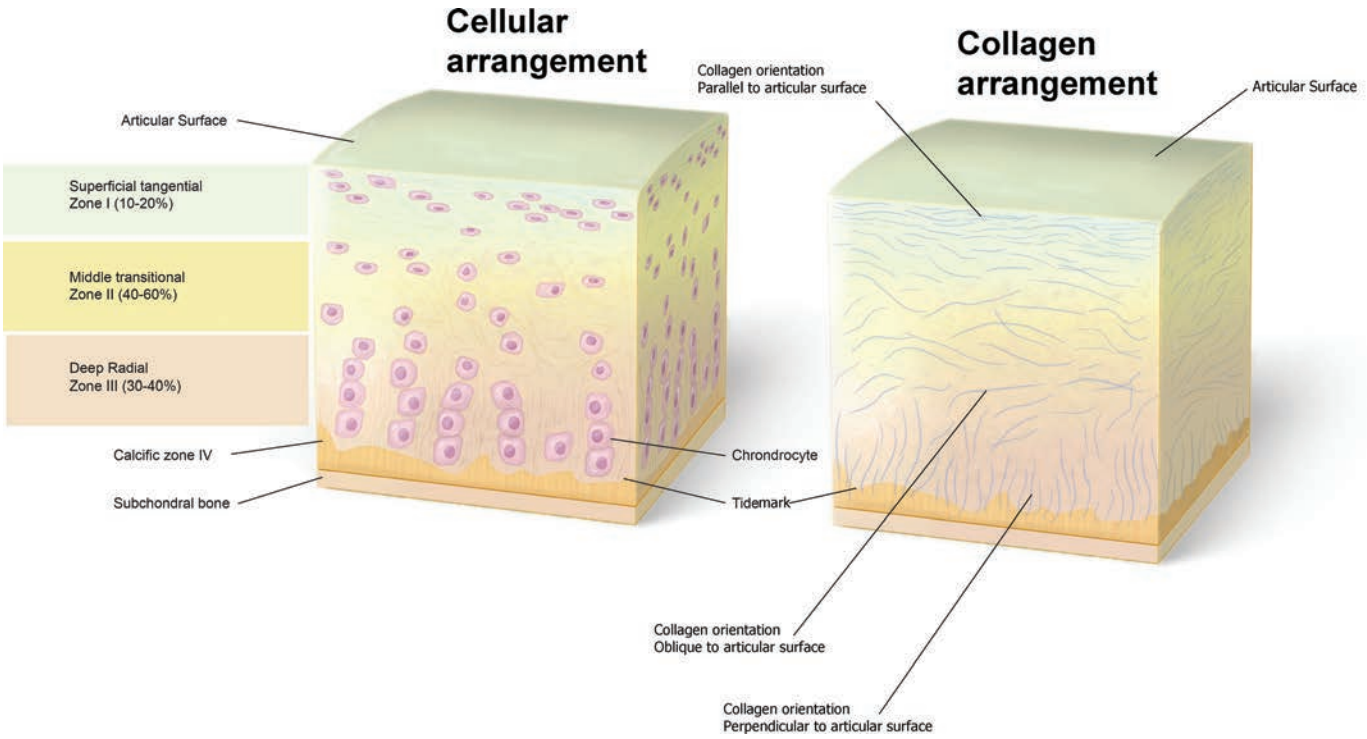


Figure 31.4 Layers of articular cartilage seen on histological section. Chondrocyte and collagen fiber arrangement

Deep (radial) zone – 30% of thickness

- Provides resistance to compression
- Collagen fibres vertically arranged (perpendicular to articular cartilage)
- High concentration of proteoglycans
- Cells spherical arranged in vertical columns

Tidemark

- Provides resistant to shear
- It is the boundary between the calcified and uncalcified cartilage. It is cell free and represents a calcification front
- The collagen fibers in the deep zone penetrate through the tidemark into the calcified cartilage to provide structural stability for articular cartilage on the subchondral bone

Calcified zone

- Anchor for the various layers
- Hydroxyapatite crystals anchor articular cartilage to subchondral bone
- Forms a barrier to blood vessels supplying subchondral bone

EXAMINER: What is the composition of articular cartilage?

CANDIDATE: The wet-weight proportions of articular cartilage are: water (65–80%); collagen (10–20%); proteoglycans (10–15%); chondrocytes (5%); and other matrix components such as adhesives and lipids.

EXAMINER: What are the contents of articular cartilage? (See Table 31.3 and Figure 31.5.)

Table 31.3 Constituents of articular cartilage

Cells (chondrocytes) (5%)			
Extracellular matrix	Fibres	Collagen (10–20%) Almost exclusively type 2	Type II, IX, XI
			Type VI, X
		Elastin	
	Ground substance	Water (65–80%)	
		Proteoglycans and glycosaminoglycans (10–15%)	
		Glycoproteins	
		Degradative enzymes (matrix metalloproteinases)	

Chondrocytes:

- Involved with the synthesis of matrix components and regulation of matrix metabolism
- Deeper cartilage zones contain no chondrocytes
- Low metabolic rate

Water:

- Up to 80% of the extracellular matrix
- Permits load dependent deformation of articular cartilage by its movement both in and out and within cartilage
- Increased water content leads to increased permeability, decreased strength and decreased elasticity
- Provides a medium for lubrication

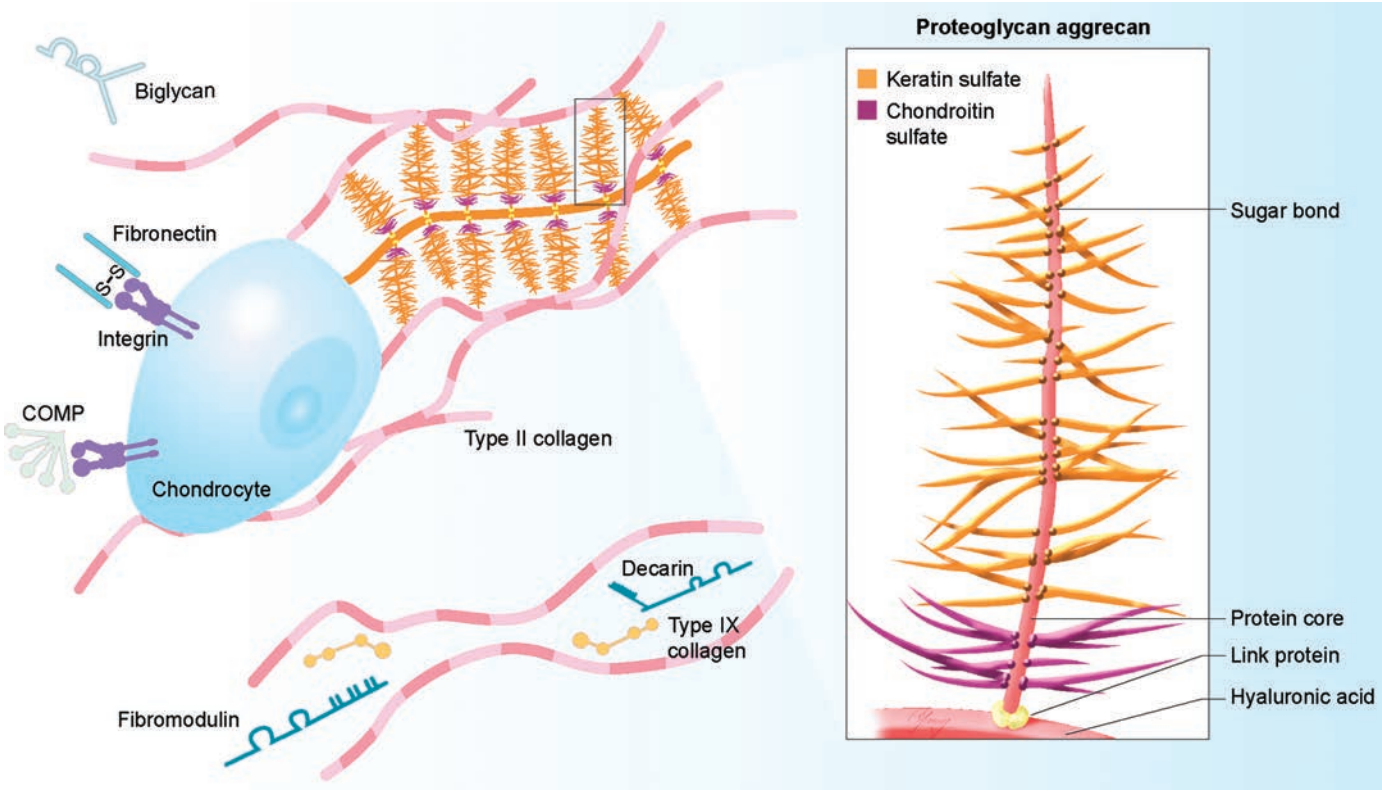


Figure 31.5 Three classes of proteins exist in articular cartilage: collagens (mostly type II collagen); proteoglycans (primarily aggrecan); and other noncollagenous proteins (including link protein, fibronectin, cartilage oligomeric matrix protein) and the smaller proteoglycans (biglycan, decorin and fibromodulin). The interaction between highly negatively charged cartilage proteoglycans and type II collagen fibrils is responsible for the compressive and tensile strength of the tissue, which resists load *in vivo*.

Collagen:

- About 10–20% wet weight, 60% dry weight
- Gives the articular cartilage its tensile stiffness
- The main collagen in articular cartilage is type II accounting for 90–95% of the collagen
- Types II.IX and XI form a mesh that serves to trap proteogylcans providing for stiffness and strength
- Type VI helps chondrocytes adhere to the matrix. Increases in early OA
- Type XI constrains proteoglycan matrix
- Type X only found near calcified zone

Proteoglycan:

- Proteoglycans are complex macromolecules that trap and hold water, providing the tissue with its turgid nature that resists compression
- They give articular cartilage its compressive strength and elasticity
- Consist of a protein core with covalently bound glycosaminoglycon chains
- The most common glycosaminoglycan in articular cartilage is chondroitin-sulphate (there are 2 subtypes, chondroitin-4-sulphate and chondroitin-6-sulphate), then keratin-sulfate and dermatan-sulphate
- Chondroitin-4-sulphate is the most abundant and decreases over the years; chondroitin-6-sulphate remains constant; and keratan-sulphate increases with age

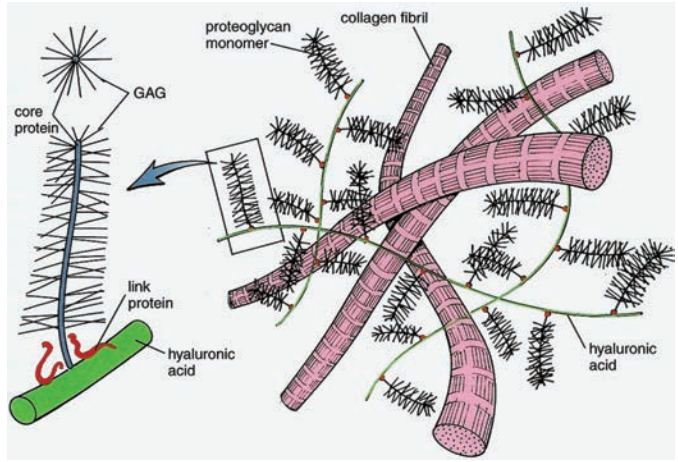


Figure 31.6 Proteoglycan aggregate and aggrecan. The aggrecan molecules are stabilised by link proteins onto hyaluronic acid to form proteoglycan aggregate.

- Glycosaminoglycan link to a protein core by sugar bonds to form a **proteoglycan aggrecan** (Figure 31.6)
- Aggrecan molecules do not exist in isolation within the extracellular matrix, but as **proteoglycan aggregates**
- Each aggregate is composed of a central filament of hyaluronic acid with up to 100 aggrecan molecules

Table 31.4 Differences between changes in articular cartilage due to ageing and those due to degeneration in osteoarthrosis

	Ageing	Osteoarthrosis
Structure	Stable, localized, superficial fibrillation	Progressive, superficial fibrillation; fibrillation and fragmentation extending to subchondral bone; loss of tissue (decreased cartilage thickness with complete cartilage loss in some regions); formation of fibro-cartilaginous repair tissue
Cells	Decreased density of chondrocytes with skeletal growth, alteration in synthetic activity (smaller, more variable aggrecans), decreased response to growth factors, decreased synthetic activity	Initial increase in synthetic and proliferative activity, loss of chondrocytes, eventual decreased synthetic activity, increased degradative enzyme activity, appearance of fibroblast-like cells in regions of fibrocartilaginous repair tissue
Matrix	Decreased concentration of water, loss of large proteoglycan aggregates (decreased stability of aggregates), increased concentration of decorin, accumulation of degraded molecules (aggrecan and link protein fragments), increased collagen cross-linking, increased diameter of and variability in collagen fibrils, decreased tensile strength and stiffness in superficial layers	Initial increase in water content and, in some instances, in concentration of proteoglycans; disruption of collagenous macromolecular organisation; progressive degradation and loss of proteoglycans, hyaluronan, and collagens; increased concentration of fibronectin; increased permeability and loss of tensile and compressive stiffness and strength

radiating from it, with each interaction stabilised by the presence of a link protein

EXAMINER: What is the structure of a proteoglycan[g]?

COMMENT: This involves differentiating between proteoglycan aggrecan[h] and proteoglycan aggregates. It is certainly known for candidates to be asked to draw out the structure of a proteoglycan aggrecan molecule. Volunteer to draw it out for the examiners (Figure 31.6). Rehearse your answer.

EXAMINER: How does collagen synthesis take place?

COMMENT: This question may be asked if a candidate has breezed through the topic with a score 8. This answer will involve discussing polypeptide chain formation, pro-collagen synthesis, cleavage to tropo-collagen fibrils, etc. It is more likely that the examiners will prefer to ask you about age changes of articular cartilage especially if time is tight.

EXAMINER: Describe the changes in articular cartilage with ageing.

CANDIDATE: Articular cartilage undergoes significant structural, matrix composition, and mechanical changes with age. There is disruption of the collagen–proteoglycan matrix. This reduces its compressive stiffness. Leeching out of the proteoglycan leads to increased permeability and reduced stiffness. Increased permeability leads to loss of lubricant.

COMMENT: Candidates may be asked to directly compare the biomechanical changes with ageing and osteoarthritis in cartilage. This can be rote learned from various tables but candidates should aim for a more detailed understanding as many follow up questions focus towards more in depth knowledge. This question is known to be asked without any lead and then moves into a more detailed discussion of treatment of cartilage lesions.

EXAMINER: Describe the changes in articular cartilage with osteoarthritis? What pathological processes are involved in the development of osteoarthritis?

CANDIDATE: The process can be divided into three overlapping stages: (1) cartilage matrix damage; (2) chondrocyte response to tissue damage; and (3) the decline of the chondrocyte synthetic response and progressive loss of tissue.

In the early stages of disease, loss of proteoglycan is reversible, whereas at later stages there is irreversible loss. The earliest visible change is loss of collagen integrity resulting in tissue fibrillation and increased water content. Decreased aggrecan concentration and aggregation, decreased glycosaminoglycans chain length all increase the permability and stiffness of the matrix and make it vulnerable to further mechanical damage. Despite chondrocyte proliferation and increased collagen and proteoglycan synthesis this response fails to halt disease progression. There is progressive loss of articular cartilage and a reduced chondrocyte anabolic and proliferative response. This is related to a downregulation of chondrocyes function.

COMMENT: Have an answer rehearsed. Whilst tables provide a succinct summary they do not give a candidate the opportunity to practice their answer (Tables 31.4 and 31.5).

EXAMINER: What are the options for treating an articular cartilage defect?

CANDIDATE: There are three main types of cartilage injury: (1) superficial matrix disruption; (2) partial thickness defects; and (3) full thickness defects. Superficial matrix disruption arises from blunt trauma whereby the ECM is damaged but viable chondrocytes aggregate into clusters and are capable of synthesizing new matrix. Partial thickness defects disrupt the cartilage surface but do not extend into the subchondral bone. These defects are unable to self-repair. Full thickness defects arise from damage that penetrates deep into the subchondral bone. These defects can elicit a repair response due to access to the marrow cells; however, they are typically filled with fibrocartilage. This type of repair tissue is much weaker than hyaline cartilage and displays poor long-term performance due to poor compressive strength and durability.

EXAMINER: What are the options for treating an articular cartilage defect in the medial femoral condyle of the knee[i]?

CANDIDATE: Although the natural history of an isolated articular cartilage lesion is not completely understood, these defects may to lead to significant morbidity and progress to diffuse osteoarthritis.

There are two broad categories of treatment:

1. Articular cartilage repair
2. Articular cartilage regeneration

[g] You may be asked to draw out a proteoglycan.
[h] Looks like a test-tube brush.
[i] Testing clinical application of basic science.

Table 31.5 Stages in the development and progression of degeneration of articular cartilage in osteoarthrosis[15]

Stage	Description
I: Disruption or alteration of cartilage matrix	Matrix disruption with increased water content. Possible causes include mechanical insults, degradation of matrix macromolecules, or alterations of chondrocyte metabolism. There is disruption of the collagenous macromolecular organization leading to increased permeability and decreased stiffness of the matrix. The concentration of aggrecans and degree of proteoglycan aggregation decrease.
II: Chondrocytic response to tissue damage	When chondrocytes detect a disruption or alteration of their matrix, they can respond by increasing synthesis and degradation of the matrix and by proliferating. Their response may restore tissue, maintain tissue in an altered state, or increase volume of cartilage. They may sustain an increased level of activity for years.
III: Decline of the chondrocytic synthetic response and progressive loss of tissue	Failure of chondrocytic response to restore or maintain tissue leading to loss of articular cartilage accompanied or preceded by decline in chondrocytic response. Causes for this decline remain poorly understood, but may partially result from mechanical damage to tissue with injury to chondrocytes and down-regulation of chondrocytic response to cytokines.

Repair refers to the healing of a damaged articular surface with a neo-cartilage tissue, that resembles native cartilage, but does not necessarily duplicate its structure, composition and function. Regeneration refers to the formation of an entirely new articulating surface that essentially duplicates the original articular cartilage.

Repair techniques (mesenchymal stem cell stimulation techniques such as abrasion chrondoplasty, driling and microfracture) penetrate the subchondral bone and induce the formation of fibrocartilage repair tissue. Although excellent short-term clinical outcomes have been reported, the clinical durability of marrow-stimulated repair tissue declines with longer follow up.

Restoration techniques such as osteochondral autograft, transfer system (DATS), mosaicplasty, and osteochondral allograft attempt to replace the cartilage defect with host or donor articular cartilage in a single stage

Osteochondral autograft transfer system (OATS) is recommended for smaller lesions, lesions in high-demand athletes, and lesions with associated bone loss.

Microfracture is suited for medium-size defects with little or no bone loss in lower demand older patients.

COMMENT: Score 8 candidates may be asked more specific details about each option such as indications, complications, results, etc, if they cover the earlier material in good time.

Examination corner

Basic science oral 1

Sit down and draw articular cartilage.

High-yield orthopaedics. One of the examiner's favourite questions. Numerous candidates have been asked to do this in the basic science oral. Don't forget to practise your drawing skills beforehand rather than in the examination itself.

Basic science oral 2

Discuss the mechanism of repair of cartilage lacerations.

Another fairly common question that turns up in both the basic science and general orthopaedic and pathology orals. The examiners tend to probe quite deep with this topic.

Basic science oral 3

Cartilage changes with age compared to osteoarthritis. (Table 31.4).

Basic science oral 4

- Articular cartilage – Draw the layers
- Discussion on types of cartilage
- Asked about different types of collagen and the basic structure of collagen

Basic science oral 5

Articular cartilage

- Draw the structure
- Types of collagen in cartilage
- Role of type IX collagen
- The majority of type IX collagen molecules exist in the extracellular matrix covalently bound to the surface of type II collagen fibrils. It is thought to have an important role in stabilising the three-dimensional organisation of the collagen network. Reduction in function of type IX collagen has the potential to contribute to articualr cartilage degeneration
- Pathogenesis of osteoarthritis

Muscle and tendon

Ligament

Examination corner

Basic science oral 1

Q. Please draw me the stress/strain curve of a ligament.

A. The candidate performed poorly, drawing the curve incorrectly, and with this the examiner took over and drew it himself to demonstrate to the candidate the correct diagram. He then began to explain the significance of each particular area on the graph.

Basic science oral 2

Q. Draw the stress/strain curve of a ligament/tendon and explain its various parts.

COMMENT: This is covered in Chapter 30. There are some slightly different stress/strain curves in various textbooks but the question is more concerned with principles and describing what is happening to the tendon. Stick with one of the standard curves that are easy to draw out and not so complicated that the examiners are unfamiliar with it.

Pathology

Thromboembolism and prophylaxis

Basic science oral 1

Q. Tell me about your postoperative plan for a patient following THA?

A. In some ways this question is a gift, inviting you to discuss your day-to-day practice. Beware, however, and lead with your strongest suit; if you start with your anti-thrombotic plan be prepared to be interrupted and to have to discuss it and the controversies that surround it. Pain control is a good starter as it's not very controversial and will get you beyond the awkward silence that follows the question.

Tendon and ligament injury and healing

Examination corner

Basic science oral 1

- Mechanisms of tendon repair
- Theories of intrinsic and extrinsic tendon repair

Basic science oral 2

Q. How do tendons and ligaments heal after injury?

Ligament and tendon healing occurs in three distinct but overlapping periods, similar to bone healing – The acute inflammatory phase, the proliferative/regenerative phase and the remodeling phase

Inflammatory phase: Starts at the time of injury and lasts up to 48 hours. Platelets form a clot and release growth factors (including platelet-derived growth factor, transforming growth factor-B and fibroblast growth factor) leading to accumulation of neutrophils and monocytes to remove debris and damaged cells

Proliferative/repair phase: Locally released growth factors lead to fibroblast proliferation and formation of an initially disorganized scar tissue with predominantly type III collagen, blood vessels and matrix. Over several weeks, this becomes more organized with collagen fibres aligning with the long axis of the structure and release of proteoglycans into the matrix. However, the newly formed collagen fibrils are still abnormal and smaller than normal. The proliferative stage is important as if the repair is too slow, the immature repair tissue will result in gapping of the tendon ends and possible failure. If this stage is too vigorous, the tendon will scar to its shealth or surrounding structures

Remodelling phase: After a few weeks, the proliferative phase merges into the remodelling phase often lasting for months or even years after the initial injury. During this phase, cellularity and both collagen and glycosaminoglycan synthesis decrease. The repair tissue changes from cellular to fibrous. Encompasses two phases:consolidation and maturation.

The first sub-stage, consolidation, is characterized by a decrease in cellularity and matrix production, as the tissue becomes more fibrous through the replacement of the weaker type III collagen by type I collagen. Cross-linking of fibrils and alignment of the fibres along the direction of any applied stresses further strengthen the healing structure. After approximately 10 weeks, the maturation stage starts, which includes an increase in collagen fibril crosslinking and the formation of more mature tendinous tissue. During this phase, the fibrous tissue is digested and replaced by more normal scar-like tendon tissue. It is important to note that most healed ligaments and tendons never achieve the same mechanical properties as a normal structure

Nerve injury and regeneration

Operative photograph shown of a divided nerve (median) at the wrist ready for repair.

Q. What factors affect the prognosis of nerve recovery following repair?

A. Functional recovery of a nerve injury depends on a number of variables

- Age of patient – Younger patients have a greater chance of functional recovery

(a) (b)

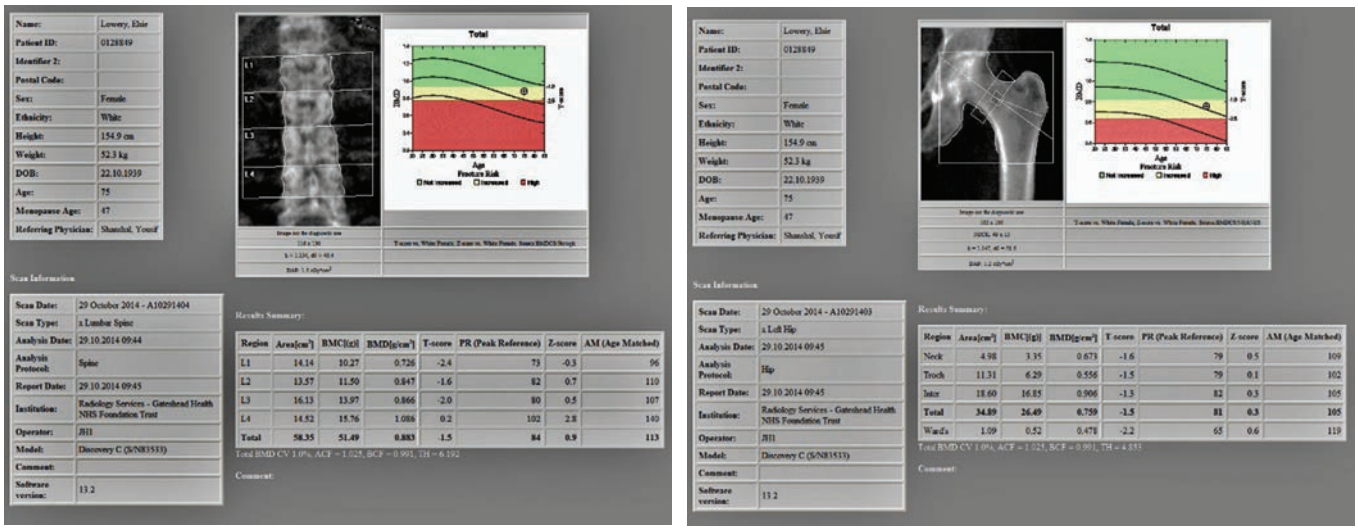


Figure 31.7 (a,b) DEXA scan. Lumbar spine and proximal femur

- Type of injury – A clean laceration is more likely to be repairable than a stretch injury or extensive area of nerve damage
- Surrounding tissues – Deep wounds with surrounding tissue damage, sepsis, scar tissue or contracture may adversely affect nerve regeneration
- Type of nerve injured – Pure motor or pure sensory nerves have better healing potential. Mixed nerves may suffer axon transposition during regeneration, resulting in improper end-organ re-innervation and, therefore, a poor functional recovery
- Level of nerve injury – Proximal injuries place a greater metabolic burden upon the nerve cell body and may result in cell death and thus, poor recovery
- Timing of repair – As time from injury to repair increases, irreversible changes occur both in the nerve cells and trunk, combined with fibrosis in the denervated muscles and joint contractures, leading to poor functional recovery potential

COMMENT: This should be a relatively straightforward question but candidates will have to talk around each variable.Ideally all variables should be remembered as forgetting two or three allows the examiners to probe deficencies and will generally lose a candidate a mark.

EXAMINER: What happens to the cell body after injury?

CANDIDATE: It undergoes hypertrophy during the healing process to accommodate greater biosynthesis of metabolites necessary for nerve regeneration. The neuron shifts from nerve conduction mode to repair mode.The cell nucleus migrates to the periphery of the cell body and chromatolysis occurs.The production of ribonucleic acid (RNA) and regeneratic enzymes increases with a corresponding decrease in neurotransmitters and neurofilament proteins

EXAMINER: What else happens to the nerve?

COMMENT: It wasn't that obvious but the candidate should have perhaps kept talking. Injury to a peripheral nerve results in structural and biochemical changes within each component of the nerve (the ventral cell body in the spinal cord, the proximal nerve stump site of injury, the distal stump and associated end-organs).

CANDIDATE: Existing Schwann cells proliferate and line up on basement membrane.

EXAMINER: What happens to the distal segment?

CANDIDATE: It breaks down with axonal disruption with axoplasm and myelin degraded distally by phagocytes.

EXAMINER: What is this called?

CANDIDATE: Wallerian degeneration

COMMENT: The candidate possibly should have been more descriptive about Wallerian degeneration and then go on to describe the other changes within the nerve.

Osteoarthritis

Basic science oral 6

'What pathological processes are involved in the development of osteoarthritis?'

COMMENT: See page 771. Remember the 3 stages of osteoarthritis development (Table 31.5) described by Buckwalter and Mankin

Osteoporosis

Although osteoporosis for most candidates is a dull topic it is an important A list high-yield key topic with great relevance to clinical practice. A DEXA scan is usually shown as a prop to lead into a discussion about osteoporosis and treatment (Figure 31.7).

One worry with osteoporosis is that one or two examiners have been known to ask candidates what seem like unclear or vague questions. This is tricky and candidates may inadvertently

lose marks if they struggle understanding the thread of the question. Pain, biomechanics and osteoporosis are three classic basic science viva topics that spring to mind but any topic can become unclear if the examiner's questions are phrased in an imprecise or obscure manner.

We use the tree analogy with this viva topic-starting off at the bottom is simply and the examiners branch out into three or four main themes whilst starting at one smallish branch in the middle of the tree can lead onto many unexplored smaller twigs.

EXAMINER: What is osteoporosis?

CANDIDATE: The WHO consensus definition states that osteoporosis is a systemic skeletal disease characterized by low bone mass and microarchitectural deterioration of bone tissue, leading to enhanced bone fragility and a consequent increase in fracture risk

Quantitatively osteoporosis is defined by the World Health Organisation (WHO) as a bone mineral density that is 2.5 standard deviations or more below the peak bone mass of an average young, healthy adult as measured on DEXA scan (dual-energy x-ray absorptiometry)[j].

EXAMINER: What are the risk factors for osteoporosis[k]?

CANDIDATE: Risk factors for osteoporosis include:

You can generate a random list of factors or attempt to structure your answer better into either:

- Primary osteoporosis vs secondary osteoporosis
- (Or) Non-modifiable vs modifiable risk factors

Primary osteoporosis is idiopathic or age related. Secondary osteoporosis occurs when underlying agents or disease induce bone loss.

Random risk factors[l]:

- Early menopause <45 years. Osteogen blocks the effect of parathyroid hormone (PTH) on osteoclasts
- Prolonged amenorrhoea in the absence of pregnancy

Primary osteoporosis

- Genetic: Positive family history, white or Asian, thin
- Hormonal: Loss of oestrogen protection
- Environmental/lifestyle: Smoking, excessive alcohol, inactivity
- Diet: Deficiency of calcium or vitamin D

Secondary osteoporosis

- Chronic medical conditions: Rheumatoid arthritis, hypothyroidism, Cushings disease, chronic renal disease, chronic liver disease, malignancy
- Drugs: Steroids, thyroxine, heparin, phenytoin, chemotherapy. Corticosteriods therapy with a prednisolone dose >7.5 mg/day for over 6 months

Non-modifiable

- Gender: Women are at greater risk of osteoporosis. Lower peak bone mass, bone loss at and after menopause, longevity relative to males
- Age: BMD decreases with age
- Ethnicity: Afro-Caribbean women have a higher BMD than white women at all ages due to higher peak bone mass and slower rates of loss
- Reproduction factors: BMD decreases most rapidly in the early postmenopausal years, early menopause
- Positive family history of osteoporosis
- Small size: This is due to a combination of low osteogen levels and reduced impact loading. Body Mass Index (BMI) <19

Modifiable

- Smoking
- Alcohol excess
- Lack of exercise
- Diet
- Drugs

EXAMINER: What is this?

You may be shown a DEXA scan and have to describe its features (Figure 31.7).

The technique involves simultaneous measurement of the passage through the body of x-rays with two different energies resulting in rapid pulses of different frequencies. By using two different energy beams it is possible to minimize the effect of soft tissues, particularly fat, on the result

EXAMINER: Can you interpret this DEXA scan?

COMMENT: The report shows:

1. A sketetal image(lumbar spine or proximal hip)
2. Bone mineral densities(g/cm^2)
3. The patient result plotted against a normal population

CANDIDATE: The T-score is the number of standard deviations (SD) above or below the mean peak bone mass for a sex- and race-matched healthy normal **young adult** population. This is the value the WHO takes its definition of osteoporosis from. The T-score is approximately what the patient should have been at their peak bone density at about age 20 years.

The Z-score is the number of standard deviations below the mean for an **age**-, **sex**- and **race**-**matched** population. As the Z-score is measured against an age-matched group it cannot detect age-related osteoporosis. Elderly white women have weak bones even if the bone density is average. Low Z-scores in young patients may suggest anorexia, chronic ill health or vitamin D deficiency

Bone mineral density(BMD(g/cm^2) is most useful when values are compared to a previous earlier scan result after starting treatment to quantify any improvement.

When looking at the lumbar spine DEXA the area should gradually increase from L1 to L5 with any deficiency usually secondary to a compression facture that would require futher evaluation with plain films.

EXAMINER: How would you investigate a patient with osteoporosis?

[j] An absolutely need to known definition. Deliver this in a pinpoint precision manner in the viva to avoid dropping marks.

[k] Predictable question so rehearse your answer to avoid strugling to articulate if you get affected with stress and forget your way.

[l] We find it difficult to remember a random-generated list of risk factors for osteoporosis and strongly recommend structuring your answer better.

CANDIDATE: I would take a full clinical history including family history, medication taken, and history of previous fractures. I would examine the patient looking for thoracic kyphosis (vertebral compression fracture), loss of height, low body weight, scoliosis and signs suggestive of secondary osteoporosis. Investigations would include blood tests to exclude second causes of low bone mass:

- Vitamin D deficiency
- Hyperparathyroidism
- Cushing's syndrome
- Hyperparathyroidism

I would order:

- FBC
- U&Es
- LFT
- Ca^{2+}
- Alkaline phosphatase
- Myeloma screen
- Vitamin D levels

Laboratory tests are usually unremarkable in osteoporosis.

EXAMINER: How do you classify osteoporosis?

CANDIDATE: Osteoporosis can be classified into type I and type II (Riggs and Melton).

Type I: Affects mainly cancellous bone at the time of menopause, so vertebral and distal radial fractures are common: Related to loss of oestrogen at the menopause; high turnover osteoporosis. Enhanced osteoclastic bone resorption with more and deeper lacunae. Osteoblasts unable to fully replace resorbed bone

Type II: Age-related (senile osteoporosis) and affects cortical and cancellous bone; occurs 10–15 years later than type I and is seen in both females and males at a ratio of 2 : 1; poor calcium absorption; low turnover osteoporosis. Hip and pelvic fractures are common. Often occurs from chronic predisposing medical conditions or prolonged use of medications such as steroids. Failure of osteoblasts to form bone. Osteoclastic bone resorption is normal or slightly decreased

Examination corner

EXAMINER: Good morning this is the basic science oral.

I am Mr Nut and this is Mr Bolt

Your name is Mr Bone – Is that correct?

Can you hand me your topic sheet please. The first thing I want to talk about is osteoporosis. You see patients in the fracture clinic. Which patients do you send for a DEXA scan[m]?

CANDIDATE: Indications for DEXA scan include long-term steroid use, early surgical menopause, and postmenopausal women with a family history of fractures, alcoholism, heavy smoking or a Body Mass Index <18.5

EXAMINER: How would you decide whom to treat with osteoporosis?

CANDIDATE: The FRAX tool is useful. It has been developed by the WHO to evaluate fracture risk of patients. Algorithms give the 10-year probability of fracture. The output is a 10-year probability of hip fracture and the 10-year probability of a major osteoporotic fracture (clinical spine, forearm, hip or shoulder fracture).

EXAMINER: Say there is an 80-year-old woman with a fractured wrist. Would you send her for a DEXA scan?

CANDIDATE: No, not if she had established osteoporosis.

EXAMINER: What about an 80-year-old woman with a fractured neck of femur for a DEXA scan?

CANDIDATE: If there is established osteoprosis and a fragility fracture there is no need for a DEXA scan and you can start immediate treatment for the condition. As a general rule if a DEXA result does not alter a patient's management then a DEXA scan is not indicated.

NICE guidelines recommend bisphosphonates (alendronate, etidronate and risedronate) as treatment options for the secondary prevention of osteoporotic fragility fractures:

1. In women aged 75 years and older, without the need for prior dual-energy x-ray absorptiometry (DEXA) scanning
2. In women aged between 65 and 74 years if the presence of osteoporosis is confirmed by DEXA scanning
3. In postmenopausal women younger than 65 years of age, if they have a very low bone mineral density (BMD, that is with a T-score of approximately –3 SD or below, established by a DEXA scan), or if they have confirmed osteoporosis plus one, or more, additional age-independent risk factors
   - Low Body Mass Index (<19 kg/m^2)
   - Family history of maternal hip fracture before the age of 75 years
   - Untreated premature menopause
   - Certain medical disorders independently associated with bone loss such as:
   - Chronic inflammatory bowel disease, rheumatoid arthritis, hyperthyroidism or coeliac disease)
   - Conditions associated with prolonged immobility

EXAMINER: Can you interpret this DEXA scan for me?

CANDIDATE: The T-score compares to peak bone mass of young normal adults

The T-score values are:

- Normal – BMD <1 standard deviation (SD) below young normal mean

[m] Examiners introduce themselves, check your name and make sure you know what viva table you are at – all within 10–15 seconds.The argument that you can lose 90 seconds at the start of the viva with introductions is generally misleading. You are down to quite a difficult first question with the Basic Science viva question almost immediately without time to catch your breath.

- Osteopenia – BMD 1–2.5 SDs below young normal mean
- Osteoporosis – BMD >2.5 SDs below young normal mean
- Severe osteoporosis – As above plus previous fragility fracture

The T-score predicts fracture risk. For every –1 SD the fracture risk doubles.

Abnormal Z-scores suggests an aetiology independent of age.

EXAMINER: How would you treat osteoporosis?

CANDIDATE: Treating osteoporosis is a multi-disciplinary approach and involves adopting NICE guidelines. These are divided into **lifestyle changes** and **pharmacological treatments**.

Lifestyle changes involve stopping smoking, reducing alcohol consumption, weight-bearing exercises and reducing falls risk.

Drug treatments would include calcium (1500 mg) and vitamin D (800 IU) supplements.

For definite treatment:

- First-line treatment would be bisphosphonates. Alendronate 70 mg once a week
- Second-line treatment would be strontium ranelate 2 mg once a day
- Third-line treatment would be raloxifene (selective oestrogen receptor modulators-SERMs)

NICE have published separate guidelines on primary and secondary prevention of osteoporotic fractures in postmenopausal women who have osteoporosis.

Essentially NICE recommends using alendronate for first-line treatment for the primary prevention of osteoporotic fractures, followed by etidronate and risedronate for second-line treatment and strontium for third-line treatment[4].

EXAMINER: Does calcium and vitamin D supplementation really work[n]?

CANDIDATE: Although the paper by Jackson from Ohio published in *The New England Journal* in 2006 showed no statistical significant difference when compared to placebo, treated women did have 12% fewer hip fractures, the type of fracture associated with the largest morbidity and mortality. Plus bone density at the hip increased slightly. So there was probably at best a modest protective effect in decreasing bone resorption, but it is less clear whether supplementation can increase bone mass or density. In addition, the dose of vitamin D in the study was probably too low at 400 IU/day it should be double this (700–800 IU/day).

Classic reference

Jackson RD, LaCroix AZ, Gass M, et al. Calcium plus vitamin D supplementation and the risk of fractures. *N Engl J Med*. 2006;354:1102.

Design: Randomised, placebo-controlled trial, 36 000 women at 40 different sites, healthy, postmenopausal aged 50–70 years (of note, corticosteriod use was an exclusion criteria)
Mean follow-up period: 7 years
Intervention: CaCO3 1000 mg plus vitamin D 400 IU daily. Personal use of calcium, vitamin D, bisphosphonates, and calcitonin was allowed. 52% of women were taking hormone therapy at baseline
Outcomes: No difference in number of hip, wrist, vertebral or total fractures. At year 6, Calcium plus vitamin D did increase BMD by 0.9% at the hip but not at the spine
Conclusions: No significant benefit, slight increase in risk of kidney stones

EXAMINER: How do bisphosphonates work?

CANDIDATE: Bisphosphonates decrease osteoclast mediated bone resorption. They attach to the osteoclast and prevent the attachment of its ruffled brush border to the bone.

EXAMINER: What are the difficulties of treating patients with bisphosphonates?

CANDIDATE: Oral bisphosphonates are associated with gastric and oesophageal irritation. They should be taken after food. Atypical femoral fractures with long-term use. Intravenous bisphosphonates can cause osteonecrosis of the jaw, atrial fibrillation, flu-like symptoms

EXAMINER: And the other treatment options?

CANDIDATE: Alternatives to bisphosphonate therapy for the treatment of osteoporosis include strontium ranelate that stimulates the proliferation of osteoblasts, as well as inhibiting the proliferation of osteoclasts. It has a dual effect on bone metabolism, increasing bone formation and decreasing bone resorption

Selective oestrogen receptor modulators (SERMs) include raloxifen. Prevents vertebral osteoporotic fractures in women with osteoporosis, and stabilises bone density. They work by attaching to osteogen receptors in the bone, stimulating the production of new bone. There is an increased risk of thrombosis and menopausal symptoms can worsen.

Teriparatide is a synthetic PTH. It stimulates both osteoclasts and osteoblasts and improves microarchitecture. Reduces the likelihood of both vertebral and non-vertebral fractures

Hormone replacement therapy (HRT) may be beneficial in primary prevention of osteoporosis but is not a proven benefit in established disease.

EXAMINER: What is the difference between osteoporosis and osteomalacia[o]?

CANDIDATE: Usually osteoporosis is painless and insidious until a fracture develops. It is commoner with advancing age. There is an imbalance between osteoclastic bone resorption and osteoblastic bone formation. In general,osteomalacia is a painful bone disorder at onset, which can present

[n] These are the types of quirky questions that can really catch you out if you are unprepared for them.

[o] This is a common viva question. It can be thrown in at various stages of a basic science viva when discussing osteoporosis or more general metabolic bone disease.

at any age. Other features of osteomalacia include proximal myopathy and paraesthesia or tetany (due to low calcium)

Osteomalacia patients may report a history of renal failure, anticonvulsant use, or malabsorption. Osteoporosis typically presents with normal biochemisty-a normal serum calcium, phosphorus, alkaline phosphatase, vitamin D, and PTH. In contrast, osteomalacia is characterized by low or normal calcium and phosphate, increased alkaline phosphatase levels, low levels of vitamin D, and secondary hyperparathyroidism. Urinary calcium levels may be normal in osteoporosis but are often low in osteomalacia. Both conditions appear as low bone mass on radiographs and DEXA scan. However, specific radiological findings unique to osteomalacia include Looser pseudofractures. On x-ray, the coarseness of the trabeculae in osteomalacia may differentiate the two diagnoses.

COMMENT: The examiners can end up asking questions on osteoporosis that seem quite wide off the mark and you can't always predict the twists and turns than may occur with this viva topic. To confuse matters material from some textbooks can drill down into obscure (half remembered for the exam) epidemiological details and facts about osteoporosis that are not always relevant to a real-life viva question.

British Orthopaedic Association Blue Book: *The Care Of Patients With Fragility Fracture*[P]:

Summary

Key elements of good hip fracture care include:

- Prompt admission to orthopaedic care
- Rapid comprehensive assessment – Medical, surgical and anaesthetic
- Minimal delay to surgery
- Accurate and well-performed surgery
- Prompt mobilization
- Early multi-isciplinary rehabilitation
- Early supported discharge and ongoing community rehabilitation
- Secondary prevention, combining bone protection and falls assessment

Six standards for hip fracture care:

1. All patients with hip fracture should be admitted to an acute orthopaedic ward within 4 hours of presentation
2. All patients with hip fracture who are medically fit should have surgery within 48 hours of admission, and during normal working hours
3. All patients with hip fracture should be assessed and cared for with a view to minimizing their risk of developing a pressure ulcer
4. All patients presenting with a fragility fracture should be managed on an orthopaedic ward with routine access to acute orthogeriatric medical support from the time of admission
5. All patients presenting with fragility fracture should be assessed to determine their need for antiresorptive therapy to prevent future osteoporotic fractures
6. All patients presenting with a fragility fracture following a fall should be offered multi-isciplinary assessment and intervention to prevent future falls

Secondary prevention of fragility fractures:

- Ideally, comprehensive secondary prevention should consist of osteoporosis assessment and treatment together with a falls risk assessment, in a 'one-stop shop' setting
- A Fracture Liaison Service, delivered by a Nurse Specialist, is a proven approach to the identification, assessment and treatment of fracture risk

Using audit, standards and feedback to improve care and secondary prevention:

- The national hip fracture database

National Institute for Health and Clinical Excellence (NICE) clinical guidelines 146. Osteoporosis assessing the risk of fragility fracture August 2012:

The guidance states that two tools, FRAX and Q Fracture, are available for use in the UK and that it is not clear whether these tools are equally accurate and whether choice of tool should depend on circumstances.

EXAMINER: What is the pathophysiology of osteoporosis?

CANDIDATE: Osteoporosis is a complex disorder characterized by an imbalance in the coupling mechanism governed by the intricate interactions between various hormonal factors, cytokines, and the RANK/RANKL/OPG regulatory system. Bone remodelling is regulated by various cytokines, including IL-1, 6 and 11, colony-stimulating factors and calcitrophic hormones, such as PTH, 1,25-dihydroxy vitamin D, calcitonin and oestrogen. Members of the TNF and TNF-receptor superfamily, receptor activator of nuclear factor-kappa B (RANK), RANK ligand (RANKL), and osteoprotegerin (OPG), play an essential role in osteoclastic bone resorption in postmenopausal osteoporosis

EXAMINER: What are the changes in peak bone mass with respect to age?

CANDIDATE: BMD is usually maximal by age 35 years and declines in women after age 40 years when the rate of new bone formation no longer equals the rate of bone resorption. The rate of decline in BMD is most rapid in women within 2 years of menopause and averages 2–4% a year during the first 7 years after menopause. BMD may decline by 25–33% during this period. Afterward, loss continues, but at a slower rate of 1–2% a year.

EXAMINER: Is there a national screening programme for osteoporosis?

COMMENT: This is a complicated question and may lead onto discussing criteria for establishment of a screening programme as applied to osteoporosis.

Osteoporosis is a common disease that affects mainly postmenopausal women. It is estimated that >2 million women have osteoporosis in England and Wales. After the menopause, prevalence rises with age from approximately 2% at 50 years

[P] See website for full summary – www.postgraduateorthopaedics.com

to >25% at 80 years. More than one-third of adult women and one-fifth of men will sustain one or more osteoporotic fractures in their lifetime.

The UK national screening committee have concluded it is not appropriate to implement a national screening programme for osteoporosis[5].

(1) There is no RCT assessing the clinical and cost effectiveness of any current approach to screening for osteoporosis. Two Scottish trials that were identified that assessed the affectiveness of osteoporosis screening on reducing fracture incidence which were conducted well over a decade ago and the results are no longer applicable, mainly due to changes in the recommended treatment[6,7].

(2) There is a lack of consensus between two leading UK sources of guidance in this area (the National Osteoporosis Guideline Group (NOGG) and the National Institute for Health and Clinical Excellence (NICE)) regarding which women should be eligible for treatment. NOGG recommends primary preventative treatment for women whose fracture risk is equivalent to the fracture risk of women of the same age who have already had a fracture. NICE's treatment thresholds are fundamentally different, because they are based on identifying groups of women for whom treatment is expected to cost no more than £20 000 per QALY gained.

(3) The long-term clinical and cost effectiveness of osteoporosis treatment is not known.

Metabolic bone disease

Examination corner

Adult and pathology oral 1

Classical radiographic changes of rickets at the wrist.

- Spot diagnosis
- Describe the radiographic features

Osteopenia of the bones and widening of the growth plates of the distal radius and ulna with flaring of the metaphysis

- What is rickets?
- What types of rickets exist?
- How do you manage it?

Adult and pathology oral 2

Classical radiographic changes of rickets of the lower limbs.

- Spot diagnosis
- Distal tibial bowing

Oral question

EXAMINER: What are stress fractures?

CANDIDATE: Stress fractures, which resemble Looser's zone, occur on the 'tension/convex side bone'. These are small horizontal stress/fissure fractures sometimes called banana fractures. They tend to be painful and locally tender and persist despite anti-Pagetic treatment.

Examination corner

Basic science oral 1

Classic AP radiograph of Paget's disease affecting the proximal femur shown.

A candidate would be expected to pick up the spot diagnosis. If you do not, it isn't a good start and candidates are on the backfoot.

EXAMINER: What is Paget's disease?

CANDIDATE: Paget's disease is bone disease that results from rapid turnover of bone. There is increased bone formation and bone resorption. There are increased numbers and size of osteoclasts

EXAMINER: Are you sure about this?

(The examiner was not convinced/happy with the answer and attempted to prompt the candidate. We think the examiner wanted a more detailed initial answer but didn't communicate this particularly well to the candidate)

COMMENT: At least mention '**haphazard** increased **osteoblastic** bone formation[q]. There are increased numbers and size of osteoclasts. Bone is enlarged, hypervascular, deformed and biomechanically weak prone to fracture.

CANDIDATE: (Hesitating as they were unsure whether their answer was wrong, what exactly the examiner wanted to hear and what they could say next to redeem the situation.) The osteoclasts and osteoblasts are both overactive in Paget's disease.

The examiner mumbles something incoherent and then goes onto the next topic.

COMMENT: This is a gift topic and the candidate should have given a much more detailed definition of Paget's disease and continued to talk on. The examiners can always stop the candidate talking if they want to.

(Fail)

Examination corner

Basic science oral osteopetrosis

Radiograph of either the pelvis, spine or legs could be shown.

Spot diagnosis. An exercise in radiographic pattern recognition. There are several rare but commonly shown radiographs of various bone diseases that candiates have to pattern recognise, i.e. Paget's, osteogenesis imperfecta, melorheostosis (candle bones).

This may lead on to a discussion of pathogenesis of the disorder.

In less severely affected individuals it is not unusual to find areas of affected bone alternating with apparently normal bone, particularly in the pelvis. Lack of metaphyseal remodelling in the femur gives a characteristic Erlenmeyer flask deformity of

[q] Try to be more detailed in your answer – the two extra words make a big difference in impressions.

the distal end of the femur. There is a failure of osteoclasis. Osteoclasts are dysfunctional mainly due to a lack of carbonic anhydrase that is needed for dissociation of calcium hydroxyapatite from bone matrix. Bone resorption fails whilst formation continues.

Rheumatoid arthritis and other arthropathies (inflammatory, crystal, etc)

Basic science oral 1

EXAMINER: This man presented with acute onset of pain in his foot (photograph shown).

CANDIDATE: This is a picture of a left foot. It shows an inflamed and swollen big toe, and the surrounding skin is erythematous and shiny. There are no other obvious features of note. The diagnosis would be suggestive of gout. My differential diagnosis would include cellulitis, infected bunion and septic arthritis.

This was a classic picture of gout affecting the big toe.

A candidate would be expected to describe the photograph, say that the picture is suggestive of gout and give a differential diagnosis. This type of question is fairly straightforward but there is little margin for error in your answer.

A candidates answer needs to be smooth and polished quickly describing what they see in the picture, the probable diagnosis and differential diagnosis. The examiners will then ask you specific questions about the condition which can go down a number of different avenues.

COMMENT: If you do not recognise the condition, have to be prompted or are slow off the mark, you will be marked down and lose a point (not recognising a common condition demonstrates a lack of basic, core knowledge).

- How do you diagnose gout?

Crystals not elevated urate levels are diagnostic!

Thin, tapered, needle shaped intracellular crystals negatively bifefringent. Candidates may be shown a picture of these crystals as a lead in viva prop.

Other clinical pictures that candidates may be shown include:

- Tophi deposited in an olecranon fossa or pinna of ear
- Ulcerated tophi through the skin with surrounding chalky material
- Radiograph of gouty arthropathy in a big toe: The periarticular erosions or cysts are larger and slightly more peripheral than those in rheumatoid arthritis and are filled with uric acid deposits

Basic science oral 2

- Calcium pyrophosphate dihydrate arthropathy (pseudogout)

Arthroscopic picture of a knee with small whitish crystal deposition on the menisci (Figure 31.8 a and b).

EXAMINER: This is an picture we took of a knee arthroscopy performed last week. Can you see anything unusual?

CANDIDATE 1: There are white bits around the meniscus

EXAMINER: What do we call this?

CANDIDATE 1: Chondromatosis

EXAMINER: That's the wrong condition. Chondromatosis is when foci of cartilage develop in the synovial membrane of joints, bursae, or tendon sheaths as a result of metaplasia of the subsynovial connective tissue. This is called chondrocalcinosis. It is very different. What do you think causes it?

CANDIDATE 1: Calcium is deposited on the meniscus.

EXAMINER: What causes the calcium to be deposited. What is the pathology of the condition?

CANDIDATE 1: You get elevated levels of calcium.

EXAMINER: There is a critical change in ionic calcium and pyrophosphate equilibrium in cartilage allowing deposition to occur. (Fail)

CANDIDATE 2: Meniscal calcification is seen.

EXAMINER: What do we call this?

CANDIDATE 2: This is called chondrocalcinosis.

EXAMINER: What is chondrocalcinosis?

CANDIDATE 2: Chondrocalcinosis occurs with calcium and pyrophosphate crystal deposition usually in cartilage. Pyrophosphate is generated in abnormal cartilage by enzyme activity at chondrocyte surfaces. It combines with calcium ions in the matrix where crystal nucleation occurs on collagen fibres. There are rare familiar forms of chondrocalcinosis; however, the vast majority of cases follow some local change in the cartilage due to ageing, degeneration, enzymatic degeneration or trauma.

EXAMINER: Any other places that can be affected besides menisci?

CANDIDATE 2: The triangular cartilage wrist, pubic symphysis and intervertebral discs can be involved.

EXAMINER: How does it present?

CANDIDATE 2: Calcium pyrophosphate dihydrate(CPPD) can present in three different ways: (1) Chondrocalcinosis – Deposition of calcific material in articular cartilage and menisci; (2) pseudogout – A crystal-induced synovitis; (3) chronic pyrophosphate arthropathy – A type of degenerative joint disease.

COMMENT: Again this picture is a spot diagnosis, the condition needs to be recognised immediately or the question is usually lost. A useful tip would be to spend time pattern-recognising pictures of the condition from two or three larger textbooks.

Basic science oral 3: gout and pseudogout

Gout: Smaller joints, pain intense, joint inflamed
Pseudogout: Larger joints, pain less severe, joint swelling and tense (less so than gout), x-rays chondrocalcinosis

Q. Difference between the microscopic appearances of the crystals?

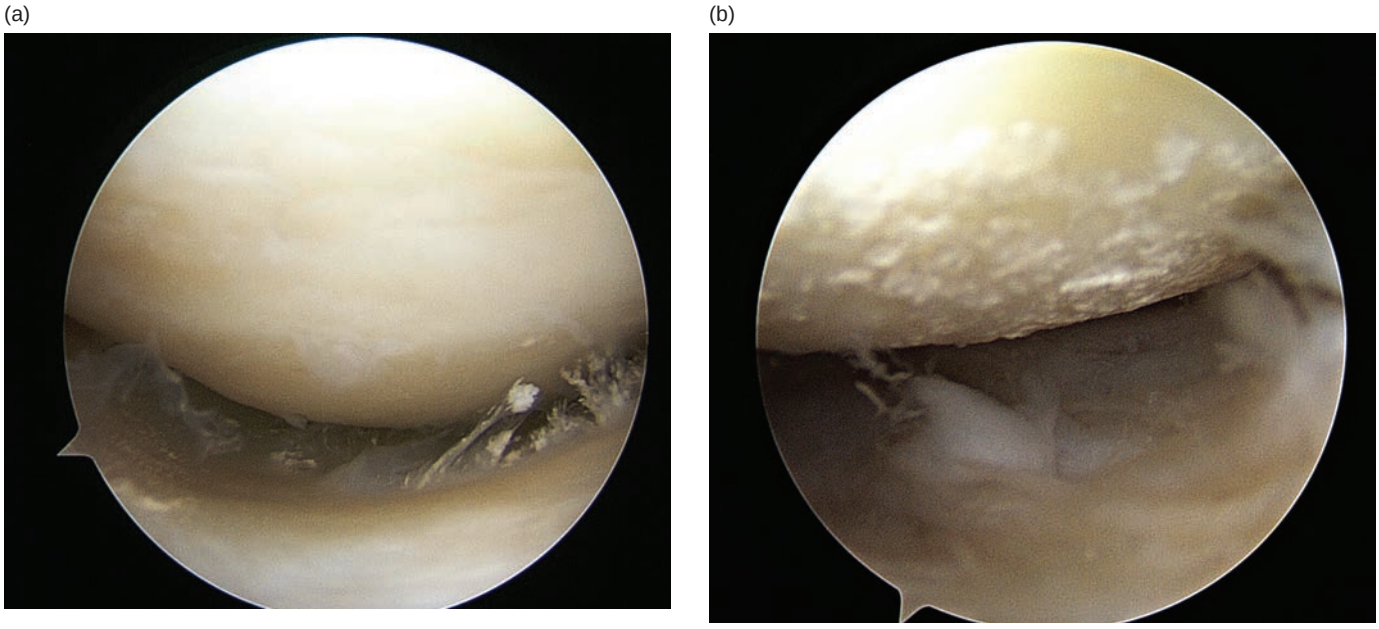


Figure 31.8 (a,b) Athroscopic picture of the knee demonstrating chondrocalcinosis

CPPD crystals weakly positively birefringegent rhomboid-shaped crystals. CPPD crystals are extruded into the joint where they can excite an inflammatory reaction similar to gout (pseudogout). Needle shaped intracellular and extracellular crystals positively birefringent.

Q. Metabolism of uric acid, DNA, purines and pyrimidines

An unexpected question to catch you off guard, certainly not one we would have expected to be asked.

Basic science oral 3: AP radiograph of the hand

Look for oval periarticular erosions. Multiple punched-out erosions distributed throughout the carpi and phalanges. Erosions have sclerotic borders and often overhanging edges (unlike classic rheumatoid arthritis). In early gout, hand and wrist joints have well-preserved joint spaces and normal mineralisation. Later on there is often overlying soft-tissue swelling which may contain small calcified fragments – Subcortical explosion. In the most advanced stages total joint destruction suggestive of a Charcot joint may be seen.

EXAMINER: When do you start long-term treatment for gout?

CANDIDATE: Less than one attack per year – No indication for prophylaxis. More than five attacks per year – Prophylaxis indicated.

Adult and pathology oral 1: Gout

- Causes
- Diagnosis
- Management

Examination corner

Basic science oral 1: Ankylosing spondylitis

Usually the adult and pathology oral. Watch out for the 'trap!' – A radiograph of the pelvis demonstrating bilateral sacroiliac fusion is shown that the candidate fails to spot as they have been told that the patient has a hip problem.

EXAMINER: This is an x-ray of the pelvis of a 20-year-old male with bilaterally painful hips. What do you see?

CANDIDATE: There is a slight loss of joint space of both hips and possibly some mild subarticular sclerosis of the femoral head. There are no osteophytes present. The overall bony texture suggests an element of osteopenia. The radiograph is suggestive of an inflammatory arthritis, possibly rheumatoid arthritis.

EXAMINER: What about the sacroiliac joints? You have failed to mention them.

CANDIDATE: There is bilateral sacroiliac fusion suggestive of ankylosing spondylitis.

If you initially fail to mention that the sacroiliac joints are fused you have lost marks. No matter what you say about ankylosing spondylitis afterwards, how many clinical features you mention, the latest theories regarding aetiology or the newest treatments you have read, you will at best just scrap through with a bare 6. You have missed an obvious diagnosis that should have been picked up. You may fail to pick up the diagnosis as a day 1 orthopaedic consultant in clinic.

Other radiographic features to mention on a pelvic radiograph would be:

- Owl's eyes of the obturator foramen due to flexion spine
- Sacroiliac joint: Bony ankylosis
- Pubic symphysis: Erosions
- Traction spur of the lesser trochanter
- Fuzziness of the ischial tuberosity

With every radiograph of the pelvis look at:
- Hips
- Spine
- Iliac wings
- Sacroiliac joints
- Pubic symphysis
- Bones
- Soft tissues

Basic science oral 2: Ankylosing spondylitis
Shown radiograph of bamboo spine of ankylosing spondylitis. What is the pathology, what is the clinical symptoms they get? How do you manage a patient with painful bilateral hips with 30° fixed flexion deformity? (See Hip clinical cases)

Examination corner

Adult and pathology oral 1
General discussion on the preoperative evaluation of the rheumatoid patient, which lead into a discussion about cervical spinal involvement of rheumatoid arthritis.

COMMENT: This can be a common component of an intermediate case discussion involving a rheumatoid patient that is probably listed for some sort of orthopaedic surgery.

Adult and pathology oral 2: AP radiography of either the pelvis or knee
Long-standing rheumatoid arthritis developing secondary degenerative joint disease.

Tricky one, especially in the heat of the exam, with several candidates assuming the diagnosis was osteoarthritis.

When rheumatoid arthritis is long-standing it is not unusual for secondary degenerative joint disease to superimpose itself on the findings one would expect with rheumatoid arthritis. The key point is that sclerosis and osteophytes indicate secondary degenerative joint disease but are fairly mild compared to the amount of severe joint space narrowing.

Examination corner

Basic science oral 1: Clinical picture of SLE hands
- Rheumatoid pattern arthropathy
- Ulnar drift in the hands (due to ligament laxity)

Basic science oral 2: Radiograph of SLE hands
Radiograph with ulnar deviation of the phalanges. Joint erosions are not typically seen. Joint narrowing and juxta-articular demineralisation. Joint effusion. Joint destruction, even in severe long-standing disease, is unusual.

Basic science oral 3: Clinical picture of typical butterfly rash

Musculoskeletal oncology

Principles of management of patients with metastatic bone disease in terms of investigation, prophylactic and definitive fixation of pathological fractures and oncological management.

Examination corner

Basic science oral 1
Shown radiograph demonstrating lytic lesion in the greater trochanter femur. Asked to describe management, investigations and likely diagnosis. Discussed Merel's scoring system.

Basic science oral 2
Describe this tumour (prop-based question usually using a plain radiograph occasionally other imaging modalities)?

Basic science oral 3
Radiograph of osteosarcoma femur. Describe the x-ray appearance.

Principles of tumour staging and biopsy.

EXAMINER: I am the tumour surgeon and how are you going to stage this tumour and manage it?

Basic science oral 3
Radiograph of a lytic lesion midshaft humerus in an elderly woman. Describe the x-ray, differential diagnosis. Diagnosis was myleloma. Other areas of bony involvement? Where else do you get myeloma deposits? How do you confirm diagnosis? Management of the lytic lesion?

Basic science oral 4
Radiograph of a fibrous cortical defect in distal tibia with pathological fracture. Differential diagnoses and management. How do you manage the fracture? How do you confirm the diagnosis?

Biomechanics and biomaterials

Bone grafts, bone banking and tissue transplantation.

Examination corner

Basic science oral 1
- Types of bone graft: Autograft, allograft, vascular, etc
- Storage
- Sterility
- Antigenicity
- What is bone graft used for?

Basic science oral 2
- Exclusion criteria for femoral head donation
- Method of collection
- Storage including temperature (–70°C ultracold freezer)

- Dose of gamma irradiation for reimplantation (2.5 megarad)

Basic science oral 3

- How are allografts processed?

Biomechanics of musculoskeletal tissues

EXAMINER: What is joint reaction force?

CANDIDATE: This is the force generated within a joint in response to forces acting on the joint.

In the hip, it is the result of the need to balance the moment arms of the body weight and abductor tension

EXAMINER: What do you understand by the term 'free body diagram'?

CANDIDATE: This is a method of determining the forces and moments acting on a body by isolating the body part and ensuring that it is in static equilibrium.

EXAMINER: What assumptions are made while constructing a free body diagram?

CANDIDIATE: The assumptions are as follows:

- Bones are rigid bodies
- Joints are rigid frictionless hinges
- No antagonistic muscle action
- Weight of body is concentrated at the exact centre of body mass
- Internal forces cancel each other out
- Muscles only act in tension (no compressive forces)
- The line of action of a muscle is along the centre of cross-sectional area of muscle mass
- Joint reaction forces are assumed to be compressive only

EXAMINER: Can you draw a free body diagram of a hip joint when a person stands on one leg?

COMMENT: Another diagram that candidates need to practice drawing out beforehand. Be prepared to be challenged/questioned by the examiners who may start asking you unfamiliar questions based on general biomechanical principles. Candidates may find it easier to practice drawing out the hip diagram first until mastered and then concentrating on perfecting the delivery as the second part of the revision process.

During walking, you stand on one leg for a short period of time. The forces acting are body weight minus the weight of one leg(W), the force exerted by the abductor muscles (Ab), and a joint reaction force between the femoral head and the acetabulum (JFR). To have equilibrium all the forces and moments must balance.

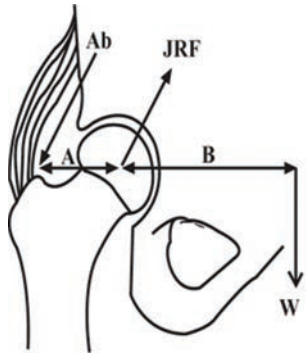
A. Free body diagram of a hip joint (Figure 31.9).

To maintain a stable hip, torques produced by the body weight must be countered by abductor muscle pull.

W = Body weight

This is weight of the body minus weight of ipsilateral leg (or 5/6 body weight)

Ab = abductor muscle force



Ab - Abductor force
A - Abductor moment arm
B - Moment arm of body weight
JRF - Joint reaction force
W - Body weight

Figure 31.9 Free body diagram hip

JRF = joint reaction force

Sum of all moments = 0

Determinants of JRF include:

- Body weight W
- Body weight moment arm B
- Abductor force (muscles) Ab
- Abductor force moment arm A

The JRF is always higher than the body weight

The right lower leg supports the body weight and also the left lower leg i.e. five-sixths total body weight. The effective centre of gravity shifts to the non-suportive leg (left) and produces a downward force to to tilt the pelvis. The right abductors must exert a downward counter-balancing force with the right hip joint acting as a fulcrum, i.e. the body weight acts eccentrically on the hip and tends to tilt the pelvis in adduction – This is balanced by the abductors.

EXAMINER: What happens to the joint reaction force in the hip if the patient has osteoarthritis of the hip and is given a walking stick in the opposite hand?

CANDIDATE: The joint reaction force is reduced by 67% by using a walking stick in the contralateral hand. A cane creates an additional force that keeps the pelvis level in the face of gravities tendency to adduct the leg during unilateral stance. It decreases the momment arm between the centre of gravity and the femoral head. The canes force substitutes for the hip abductors. The longer the distance from the centre of the hip to the contra-lateral hand the better mechanical advantage.

In simple terms the cane transmits part of the body weight to the ground thereby decreasing the muscular force required for balancing.

EXAMINER: Can you draw this out for me?

EXAMINER: What other methods are used to decrease the joint reaction force in the hip joint?

CANDIDATE: Actions that decrease joint reaction force include:

- Increase in ratio of A/B (shifting center of rotation medially)
- Moving the acetabular component as far medial, inferior, and anterior as possible
- Shifting body weight over the affected hip-this occurs with a Trendelenburg gait
- Increasing femoral component offset

- Lateralisation of greater trochanter
- Varus neck-shaft angulation. Increases the abductor lever arm so abductor muscles generate less force and JRF is reduced
- A stick in the contralateral hand reduces abductor muscle pull and decreases the moment arm between the center of gravity and the femoral head
- Carrying load in ipsilateral hand – This produces additional downward moment on same side of rotational point

Candidate: Actions that increase joint reaction force include

- Valgus neck-shaft angulation. Shortens the abductor lever arm and requires the abductor muscles to generate a greater force thereby increasing the JRF across the hip

Biomechanics of fracture fixation

Screw[r]

This is a common A list 5-minute basic science or trauma viva question. Examiners would expect most candidates to have a good knowledge of the topic. Although there are several exam-orientated accounts of the topic circulating the examiners never seem to ask straightforward questions preferring less obvious ones that test a candidates indepth understanding.

Try to work out beforehand which areas the examiners usually hover around. Candidates can read the topic in a textbook but it only really comes to life in a real viva situation with the to and fro questioning and answering between examiner and candidate.

EXAMINER: Can you draw a screw and label various parts?

COMMENT A laminated photograph or photographs showing various screws (canuulated, cancellous, cortical and locking bolt) may be shown to candidates (Figure 31.10). With a standardize viva format the days of just being handed a screw and asked to describe are over. Candidates may be asked to discuss how the various screws differ in design features.

Candidates may also be asked to draw out a screw and so need to practice drawing and describing out loud the various parts of a screw beforehand (Figure 31.11). Candidates may be stopped at any stage to be more closely questioned on a particular aspect of design.

A screw has a commn design consisting of:

- Head
- Shaft. The shaft is comprised of a threaded section and in partially threaded screws there is also a non-threaded section. Shaft diameter is used to describe the diameter of any non-threaded sections. Threaded sections have both a core diameter and a thread diameter

[r] Take hold of a screw and practice out loud describing its various components until flawless.It may take two or three goes to get it right.

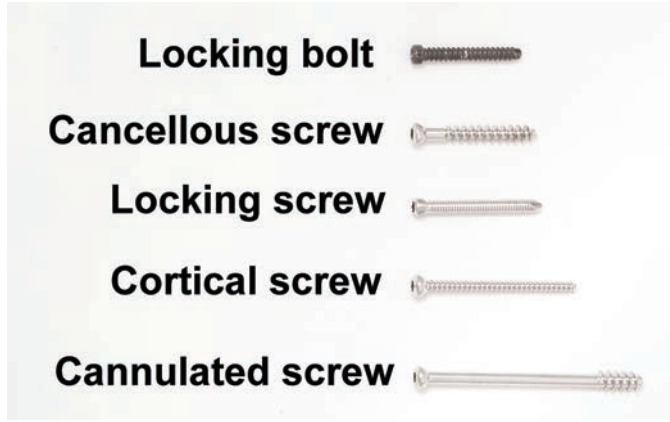


Figure 31.10 Photograph of various screw types: Cortical, locking screw, cancellous and locking bolt

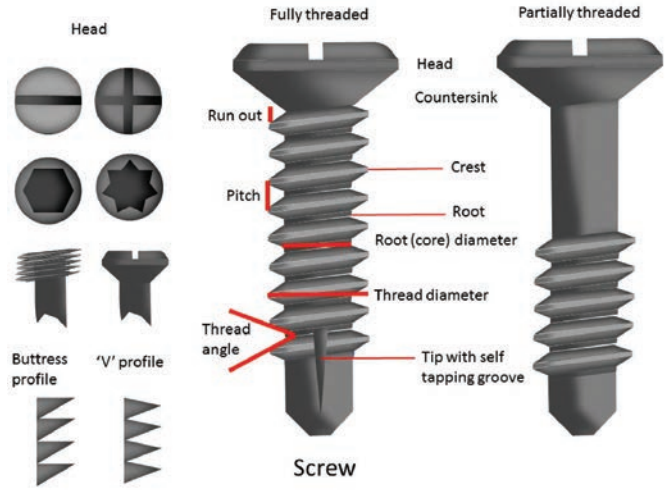


Figure 31.11 Design features of a screw

- Thread
- Tip

EXAMINER: Can you describe the different parts of the screw and their function?

Head:

EXAMINER: Why are there different design features for the head? What is the function of the head?

The head provides a connection for a screwdriver by the recess. The slot for a screwdriver may be star, hexagonal, Philips. A hexagonal head has six points of contact to increase torque, avoid slip and improve directional control. Avoiding slip is important especially for titanium screw heads which can be easily deformed.

A locking screw has threads on the head to allow locking into plates and provide angular stability.

When screws are placed at an angle in cortical bone a counter sink can be used to increase the contact surface area and reduce pressure and the subsequent risk of failure of the underlying bone.

Run out: Transitional area between shaft and thread, where the screw breaks

Shaft: Smooth link between head and thread

Thread geometry

Most bone screws have asymetrical threads i.e. flat on upper surface and a rounded underneath. This provides a wide surface for pulling and little frictional resistance on the underside.

- **Thread pitch**[s]: The distance between the threads. The shorter the distance the 'finer' the pitch, the longer the distance the coarser the pitch. Cortical screws have a fine pitch and, therefore, more number of threads to engage a reduced thickness of cortical bone. Cancellous screws have a coarse pitch
- **Lead:** The linear distance travelled by a screw for one complete (360°) turn of the screw. If a screw has only one thread, the pitch and the lead are identical. If screw has more than one thread, the lead of the screw is increased proportionally to the number of threads. If the screw is double threaded (designed for faster screw insertion), the lead is twice the pitch
- **Thread depth**: Thread depth is half the difference between thread diameter and core diameter. The thread depth determines the amount of contact with bone that in turn determines the resistance to pull out. In cancellous bone (weaker than cortical bone) a deeper thread is needed to increase the surface area to improve the purchase, as the bone is weaker. This increases resistance to screw pull out
- **Thread shape**: The shape of thread may be V-thread, buttress thread, reverse buttress or square thread

Diameters

- **Core diameter**: Smallest diameter in thread section. Solid section from which the threads project outwards The size of drill bit used is equal to the core diameter. Cube of the core diameter is proportional to the tensile strength. Increasing a screws core diameter a factor of two allows it to withstand torques eight times greater
- **Shaft diameter**: Diameter of shaft where no thread
- **Outer or thread diameter**: The maximal thread width. The larger the outer diameter, the greater the resistance to pull out

Flutes: Channels that provide a route for removal swalf (bone debris)

Tip: Several different designs are available. The tip can be:

- Standard non-tapping screw-smooth, conical tip: Needs pre-drilling of a pilot hole and then use of a tap to creat a channel/thread for insertion
- Self-tapping: Needs pre-drilling of a pilot hole but has cutting flutes for creating its own thread/channel in cortical bone that allows bone cuttings to escape. In general inferior bone holding ability
- Self-tapping and self-drilling-tip will make a drill hole and will cut the channel for the thread

EXAMINER: What mechanical properties does a screw have?

What is a screw?

CANDIDATE: Several definitions:

- A screw is a mechanism that produces linear motion as it is rotated
- A screw is a device that converts torsional force into an axial force
- A screw is a mechanical device that converts a rotational movement (torque) into a linear movement (translation)

EXAMINER: How can you maximize pullout strength[t]?

CANDIDATE:

- Increased outer diameter
- Decreased inner diameter
- Increased thread density (reduced pitch)
- Increased thickness of cortex
- Use cortex with more density
- Use locking screw

The 'finer' the pitch and the more turns the surgeon needs to make to insert the screw and the more turns of the spiral thread engage in a given depth of cortex. The more volume of bone caught between threads, the greater the pull out strength. This can be achieved by increasing the thread width of the screw, or increasing the number of threads in contact with the bone. The more threads engaged in the cortex, the greater the pull out resistance.

EXAMINER: Describe the different types of screw?

CANDIDATE:

- Cortical and cancellous. The main differences relate to:
  - Pitch. With cancellous screws larger pitch, greater thread depth and less number of threads. Cortical screws have smaller pitch, greater

[s] Thread pitch is incorrectly described in some textbook as the distance through which a screw advances with one complete turn. This is lead. However, lead and pitch are identical if a screw has only one thread. On a double-threaded screw the lead is two times the pitch. This allows faster screw insertion.

[t] The examiners may stop a candidate halfway through their description of a screw and ask this question.

Figure 31.12 Locking screw

number of threads and are designed to have better purchase in cortical bone

- Tip. Cancellous screw tips are designed as a tapering spiral. These tips create their own threads in cancellous bone, as the tapered spiral advances, it pushes the spongy cancellous bone aside to thread its way into the bone. Cortical screws are usually blunt ended
- Core to outer diameter. Higher ratio in cortical bone

- Fully or partially threaded. Fully or partial threaded with cancellous. Fully threaded with cortical
- Locking (Figure 31.12) and non-locking

Cannulated and non-cannulated screws. Canulated screws have a hollow core to allow placement over a guide wire. The hollow core weakens the screw although clinically this is not often a problem. They are useful for accurate positioning near articular surfaces. Guide wire allows radiological check prior to screw insertion

COMMENT: The locking screw design features may lead onto a general discussion of locking plates.

EXAMINER: What are the principles of lag screw fixation across a fracture site?

CANDIDATE: This involves over drilling the near side object to a diameter slightly larger than the thread diameter, creating a gliding hole. The far object is drilled as normal to core diameter and tapped to the thread diameter. The screw thread only gains purchase in the far object so when the head comes into contact with the nearside object it allows compression of the two objects.

- Drilling large lag hole (near cortex) = 3.5 mm drill and small hole (far cortex) 2.5 mm
- Countersinking
- Measuring
- Taping the far cortex
- Insertion of screw

EXAMINER: What are the functions of a screw[u]?

CANDIDATE:

- To produce interfragmentary compression
- To attach implants to bone by compressing them onto the bone surface
- To attach implants to bone producing angular stability
- To hold two screws in correct relationship

Figure 31.13 IM locking bolt

- To lock an intramedullary nail to the cortices
- To block movement of a main fragment around an IM nail – Poller screws

EXAMINER: What are the design features of an IM locking bolt (Figure 31.13)?

CANDIDATE: A locking bolt has a wide core diameter and small outer thread diameter. It functions to create a rotationally stable construct with a large tensile strength and small thread diameter as it does not require a large amount of pull out strength.

Locking plate

Laminated photograph shown of volar wrist locking plate (Figure 31.14). A 5-minute viva on volar locking plates in which a candidate will need to keep talking[v] and not dry out with nothing to say after a minute. This viva will not go onto other types of plates but sticks with the locking plate construct. With the new viva style you are unlikely to be handed a locking plate by the examiners. This viva topic is equally applicable to the trauma viva.

EXAMINER: Do you recognise this plate?

CANDIDATE: Yes it is a volar wrist locking plate

EXAMINER: How does a locking plate work?

CANDIDATE: A locking plate is a fracture fixation device with threaded screw holes that allow the screw to lock into the plate eliminating screw toggle and creating a fixed angled device that maintains reduction.

A non-locking plate when used in a buttress mode can resist only moderate axial and bending forces, thus, potentially allowing angular motion at the plate screw interface.

Fixed-angle implants transfer load stress from the fixed distal fragment to the intact radial shaft, thus, enhancing peg/plate/bone construct stability, unlike rigid internal fixation devices that rely mainly on the frictional force between plate and bone to achieve fixation.

Most distal volar locking plates have two parallel rows and the orientation planes of their respective pegs specifically

[u] The examiners may drill down for more detail onto a particular fuction. Please see website for further detail: www.postgraduateorthopaedics.com.

[v] Provided what you talk about is relevant and sensible and not waffle. The other irritating habit to examiners is talking 10 sentences but only saying 1 sentence of importance.

Figure 31.14 Volar distal radius locking plate

match the complex three-dimensional shape of the radial articular surface.

The primary row pegs are directed obliquely from proximal to distal to support the dorsal aspect of the aticular surface. They are angled accurately to provide support for the radial styloid and dorsal ulnar fragments. These pegs are most effective in supporting the dorsal aspect of the subchondral plate and, hence, avoid the redisplacement of the dorsally displaced fractures. Concurrently, their action induces a volar force that tends to displace the fragments in a volar direction, an effect that must be opposed by a properly configured volar buttressing surface.

To help fracture fixation in cases of severe comminution or osteoporosis, an additional row of pegs originating from a more distal position on the plate and having an opposite inclination to the proximal row was created. The distal row is directed in a relatively proximal direction and crosses the proximal row at its midline and is intended to support the more volar and central part of the subchondral bone. It prevents the dorsal rotation of a volar and marginal fragment and volar rotation of severely osteoporotic or unstable distal fragments with central articular comminution, thus, neutralizing volar displacing forces of the pegs in the proximal row.

The locking screw design is different

Threaded underside of head

- To thread (lock) into plate hole

Larger core diameter

- Increases strength
- Dissipates load over larger area of bone

Smaller thread pitch

- Threads not used to generate compression between plate and bone

EXAMINER: What are the advantages of using a locking plate?

- Strength of fixation relies on the fixed angle construct of screws to plate
- As the locking plate is slightly off bone there is a preservation of periosteal blood supply compared to conventional plates that compress the undersurface of the plate to cortical bone
- Conventional screws use most of the pullout force for plate compression; little pullout force remains to resist physiologic forces. Locking screws do not compress the plate to bone and use the entire pullout force (pullout strength) to resist physiological forces. Conventional screw constructs usually fail because individual screws toggle, loosen, and pull out. As locking screws are fixed to the plate, the screws must all fail or pull out simultaneously
- The plate sits slightly off bone and acts as an 'internal' external fixator
- It is a precontoured plate that allows anatomic fit. They do not require precise adaption of the plate to the contours of the underlying bone
- Some locking plates allow percutaneous insertion in contrast to a single large incision(minimally invasive plate osteosynthesis –MIPO). Generally best to use a long plate in this situation.

EXAMINER: What are the disadvantages of using a locking plate?

- They should not be used in helping to achieve a fracture reduction. Many locking plates may have combination holes to allow insertion of either a locking or conventional screw. Non-locking screws should be inserted first lagged through the plate to achieve compression. This reduces the possibility of fracture distraction occurring that then is held rigid which predisposes to delayed or non-union
- It can be difficult to remove screws due to cold welding, osseointegration or over-tightening and stripping of screw heads
- Expensive. Both the plate and locking screws
- Any attempt to contour locked plates could potentially distort the screw holes and adversely affect screw purchase
- Pre-determined screw direction may result in improper seating of the screw head
- There is a danger of producing too rigid a construct in situations where relative stability is preferred leading to delayed and non union
- A surgeon may lose the feel for bone quality during screw insertion and tightening, when the screw head engages in the conically threaded plate hole
- Insufficient working length of locking plates when bridging fractures may lead to premature plate fracture. Plate length should be a minimum of 2–3 times the length of the zone of comminution.

EXAMINER: What are the indications for using locking plates?

When conventional screw purchase may be poor

- Fractures occurring in poor quality bone
- Comminuted metaphyseal or diaphyseal fractures

- Periprosthetic fractures
- Failed fixation (non-union)
- Complex periarticular fractures

Can use either unicortical or bicortical locking screws. Bicortical screw fixation is generally preferred as theoretical concerns that unicortical locking screws may have suboptimal torsional fixation strength. A minimum of 2 bicortical or three unicortical screws on either side of the fracture should be used.

To maximize locking plate stability use bicortical locking screws, use a large number of screws, minimal screw divergence from the screw hole(<5°) and use a long plate.

Types of plates[w]

Plates are classified according to their design (compression, one-third tubular, locking, reconstruction) or function (compression, neutralisation, buttress, bridging, antiglide)

If shown a plate begin by describing design, material, shape of hole, shape and any biological factors (LCP)

- One-third tubular plate

  This is primarily used as a neutralisation plate, when a lag screw has already provided fracture compression. The plate is thin and allows for easy contouring. The plate 'neutralizes' forces and protects the lag screw from failure
- Compression plate

  Compression plates can be used in either static or dynamic compression mode

  Dynamic compression (DCP) dynamic. The plate holes are oval designed with a sloping edge at the end of the hole farthest from the fracture. The plate hole allows a screw to be inserted eccentrically so that as it is tightened, the underside of the screw head glides down the slope and moves the plate in relation to the bone creating compression at the fracture site. The first screw is applied in a neutral position in a plate hole on one side of the fracture and then a second screw is applied in an eccentric position in a plate hole on the other side of the fracture

  Static compression. The plate hole design allows a screw to be inserted in a neutral position, so that when the screw is tightened, relative movement occurs between the plate and bone. The plate can produce fracture distraction at the cortex opposite the plate and to reduce this fracture distraction the plate should be pre contoured to provide a concave bend that will create compressive forces on both the far and near cortices of the fracture

Low-contact dynamic compression plates (LCDCP)

The plate bone interface can result in periosteal compromise and necrosis. Limited contact DCPs are designed to limit stress shielding and vascular compromise by decreasing plate to bone contact

- Less invasive stabilisation system (LISS).

  LISS was originally designed as a device that would provide angular stability and accommodate only locking head screws; all of the plate holes are threaded. However, surgeons found that this technology was too restrictive in some cases. This led to the concept of a combination hole, which has been incorporated into the locking compression plate
- Locking compression plates – LCP

  The holes in a locking screw plate are threaded as are the heads of the corresponding screws so that a screw lock into the plate when tightened. This configuration provides a rigid construct for fracture fixation and acts similar to an external fixator. This system is sometimes referred to as an internal external fixator
- Reconstruction plates

  These plates are thinner than DCPs but thicker than one-third tubular plates. Deep notches in the side of the plate allow contouring in three planes. Bending of more than 15° at any one site should be avoided. Their main use is in pelvic and acetabular fractures
- Precontoured plate

  A precontoured plate provides rigid fixation without compromising plate stiffness and fatigue strength

Principles of plating

- Fixation of plates, screws and bone should form as stable a construct as possible
- Preserve soft tissue and blood supply to the bone
- Place plate in position relative to bone to minimize stress
- Plate materials should be sufficiently strong to resist fatigue failure

Modes of plating[x]

- **Neutralisation plate** – This protects lag screw fixation from bending, torsional and shearing forces
- **Compression plate** – Plate provides compression of fragments at the fracture site
- **Bridge plate** – In multi-fragmentary fractures occasionally the only option is to bridge the fracture fragments. No screws are used at the level of the fracture. The plate provides for relative stability, length and alignment in heavily comminuted fractures with possible bone loss. This reduces soft-tissue dissection at the fracture site and is, therefore, less disruptive to the blood supply of the fracture. A plate applied in bridging mode is at increased risk of fatigue failure

[w] This is another 5-minute viva topic equally at home in either the basic science or trauma viva. There are a lot of biomechanical principles than can be discussed that may be focused down into narrow discrete areas.

[x] This may be asked in a fairly generic discussion of plates and can take up 2–3 minutes of a viva question.If gone through quickly then the examiners may move on to the mechanical strength of a plate and bending rigidity.

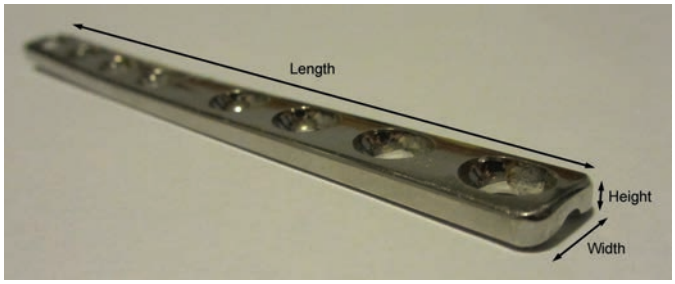


Figure 31.15 The stiffness of a plate is proportional to its width, and to its thickness to the third power. A small increase in the thickness of a plate greatly increases its stffness

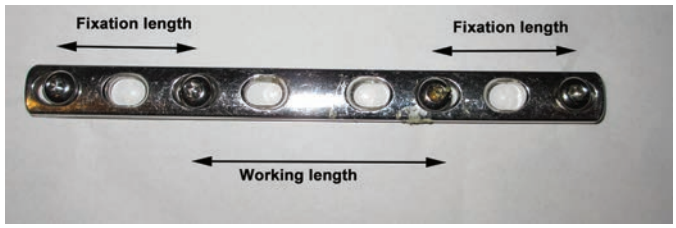


Figure 31.17 Increased working length of a plate decreases the stress and strain concentration on the plate and allows the plate to withstand more cycles before failure

- **Buttress** – The plate is used to support or reinforce (buttress up) articular surfaces. The plate counteracts compression and shear forces that often occur with periarticular fractures. A plate is applied in buttress mode when a fracture has an apex/axilla, a plate applied to this surface prevents sliding motion between fragments
- **Tension-band plate** – Bones are not always loaded evenly along all axes. If the fracture is fixed on the side tending to open (tension side) then the tension forces on one side are converted to compression forces on the opposite cortex
- **Antiglide plate** – The plate is placed at the apex of an oblique fracture that prevents shortening or angular displacement

Mechanical strength of a plate

The moment of inertia is a term used to describe the spatial distribution of a material within an object. For a plate the moment of inertia increases directly with the width of the plate and the cube of its height (Figure 31.15).

For a structure with a quadrilateral cross-section (plate) the secondary moment area (SMA) = $WH^3/12$

where H is the smaller dimension and W is the larger dimension of the cross-section.

Rigidity in bending is an important biomechanical issue of a metal plate

Bending rigidity = SMA × Young's modulus

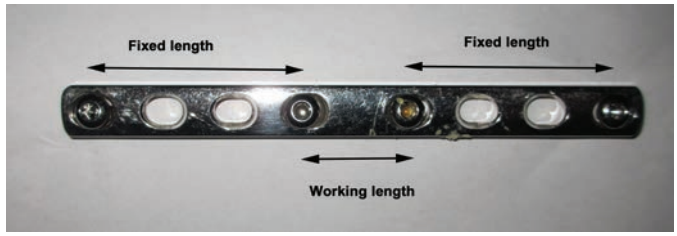


Figure 31.16 The working length of a plate can be altered based on screw position. Screws placed close to the fracture create a short working length, which increases the plate construct stiffness

Therefore, using a material which is twice as stiff results in makes a given plate twice as rigid. Doubling the thickness of a plate results in an increased rigidity by $2^3 = 8$.

The working length of a plate is defined as the distance across a fracture site between the 2 nearest points where the bone is fixed to the plate, e.g. the distance between the 2 screws closest to the fracture (Figure 31.16). Placing screws close to the fracture site has the potential to create stress concentration on the plate and fatigue failure. When the inner most screws are further away from the fracture site, the working length of the plate is greater, allowing bone deformation and gap opening, but there is better stress distribution in the plate.

With the length of the plate constant, an increase in number of screws used results in a decreased load on each screw. With more screws, the fixation is more rigid and there is less tendency of failure due to screw pull-out. However, more screws weaken the bone. Hence, there is a need to strike a balance with the appropriate number of screws used

Examination corner

You have been asked to design a new plate for fracture fixation. What features in the plate will you incorporate into your design[y]?

Chose a plate to discuss. If you are familiar with the locking plate concept it could be relatively straightforward to discuss the features of a anatomical preshaped locked compression plate with the additional new feature of variable angle locking. This allows for a customised, multi-directional locking plate construct that permits locked screws to be inserted at varying angles, to avoid existing hardware or to capture specific fracture fragments

An easier option would be to discuss the design of a new compression plate

Ideally you would want to accelerate fracture healing whilst not interfering with bone physiology

It is important to minimize bone–plate contact interfering with cortical perfusion as this may lead to cortical porosis, delayed healing and refractures after plate removal.

[y] Never straightforward questions, always a twist in what gets asked.

Sufficient strength, corrosion resistance and minimizing any stress shielding
Material (stainless steel, titanium, cobalt chromium)
Screw hole (eccentric for dynamic compression)
Plate shape (anatomic)
Biological factors (LCP)
Locking or non-locking

The ideal biomaterial for a bone plate should have good corrosion resistance, wear resistance, strength and biocompatibility. It should also have close mechanical properties, especially Young's modulus, to bone. Furthermore, it should not cause toxicity, allergy or sensitivity

COMMENT: This question tests higher-order thinking. Candidates have to apply principles and knowledge they have learnt into a question they probably were not expecting. The basic science viva is never too straightforward.

Examination corner

The principles of tension-band wiring is a fairly common viva question either trauma or basic science. Candidates may be shown different props (Figures 31.18 and 31.19) to lead into the question. Equally candidates may be asked to draw out and explain how the tension-band principle works. Rehearse well beforehand and sound convincing in your answer. Do not run out of steam after 30 seconds or become blank if probed in more detail beyond the basic principles.

The candidate was shown Figure 31.19 and asked to explain the diagram.

Forces are distributed in an eccentrically loaded column so that compression forces occur on the compression side of the column, the side closest to the load, and tension forces open the column on the side away from the load.

The eccentrically placed load can be balanced by an equal and opposite tension force eccentrically placed on the opposite or tension side of the column. In this figure, the mechanical device creating the tension force is a chain. In fracture treatment, the chain is replaced by a tension-band wire system. The tension-band wire system absorb the tension forces of an eccentrically and oppositely placed load so that the column, or in the case of a fracture, the bone, receives only compression forces[z].

A curved, tubular structure under axial load always has a compression side as well as a tension side. A tension-band converts tensile force into compression force at the opposite cortex. This is achieved by applying a device eccentrically, on the convex side of a curved bone. In fractures where muscle forces distract the fragments, such as an olecranon fracture, the application of a tension-band will neutralise these forces and convert them into compression when the joint is flexed.

EXAMINER: What are the prerequisities of tension-band fixation?

[z] Candidates can sometimes give a perfectly reasonable answer but not sound convincing or confident to the examiners. If this occurs a candidate may need to track in a slightly different direction with their answer.

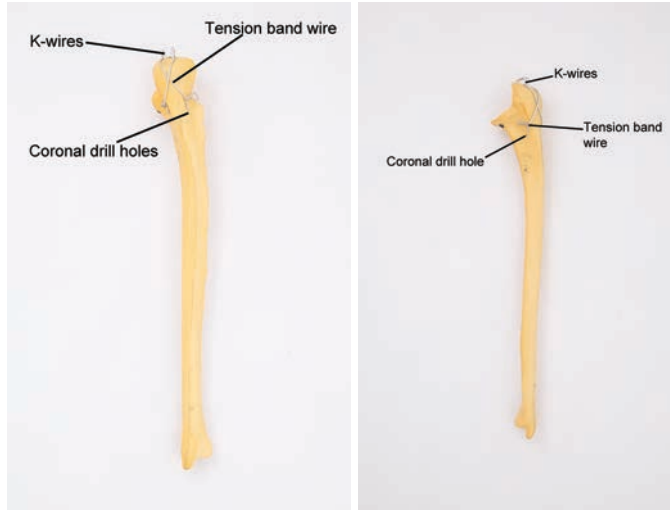


Figure 31.18 Bone model illustration of tension-band fixation of olecranon. Two parallel K-wires are used and the tension-band wire is placed through a drill hole in the distal fragment. Before tightening the figure-of-8 wire, 2 loops should be made, which allows more even tightening on both sides of fracture

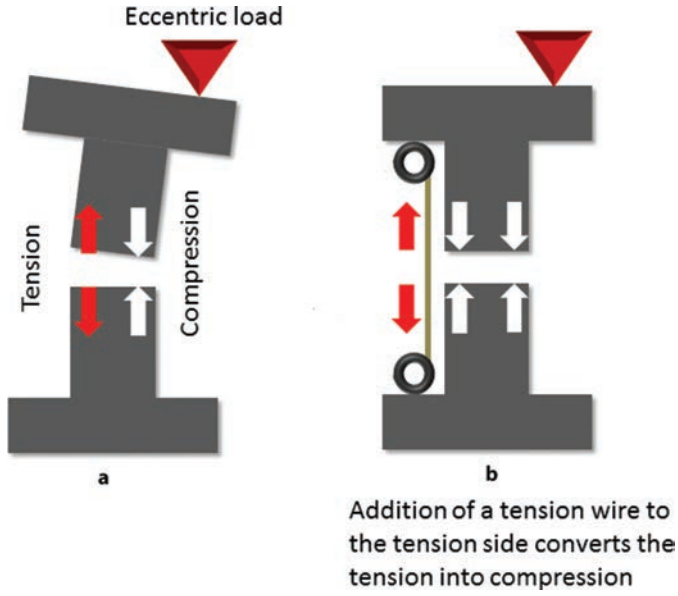


Figure 31.19 The tension-band principle. In (a) an eccentrically loaded fractured bone has tension and compression sides. The application of a tension-band in (b) prevents distraction and converts tension into compression at the opposite cortex

CANDIDATE: Prerequisties of tension-band wiring are: (1) A wire that is able to withstand the tensile forces; (2) bone that is able to withstand a compressive force; (3) an intact buttress of the opposite cortex.

EXAMINER: So how is this relevant to the TBW fixation of the patella?

CANDIDATE: The anterior tension-band around the patella converts tensile forces on the anterior aspect of the knee joint into compressive forces at the joint line. Multiple fragmented patella

fractures can not be fixed with a tension-band as the posterior articular cortex cannot be communited as it must provide a buttress to allow compression.

Advantages are that this system allows early range of movement but disadvantages include metalworking backing out requiring removal.

Tribology of natural and artificial joints

Examination corner

Basic science oral 1

EXAMINER: What types of lubrication exist?

CANDIDATE: There are three basic types of lubrication: Boundary, mixed and fluid film lubrication. Fluid film lubrication includes hydrodynamic lubrication, elastohydrodynamic lubrication, and microelastohydrodynamic lubrication. With synovial joints the cartilage on cartilage interface has additional mechanisms for achieving fluid film lubrication under different conditions and include weeping lubrication, squeeze film and boosted lubrication.

Hydrodynamic lubrication is the most basic form of lubrication. Rigid bearing surfaces that not parallel and are separated by a fluid film slide tangentially in relation to each other.

EXAMINER: That's fine you don't need to go into any more detail. What type of lubrication exists in artificial joints?

CANDIDATE: It is thought that boundary lubrication predominates. In this regime, the synovial fluid (lubricant) is not thick enough to prevent contact between the asperities of the two opposing surfaces. However, the boundary lubricant can separate the two surfaces enough to prevent severe wear

(Pass)

Basic science oral 2

Q. What are the various types of lubrication in synovial and artificial joints?

Design of implants and factors associated with implant failure (wear, loosening)

Cemented femoral stem

Introduction

This viva question should be relatively straightforward. Ten years ago when candidates were not very familiar with exam viva material it was definitely an awkward topic especially if complicated biomechanical concepts had to be worked out from first principles. With the informal development of a known exam viva question bank for candidates there are really no excuses now for botching this question up.

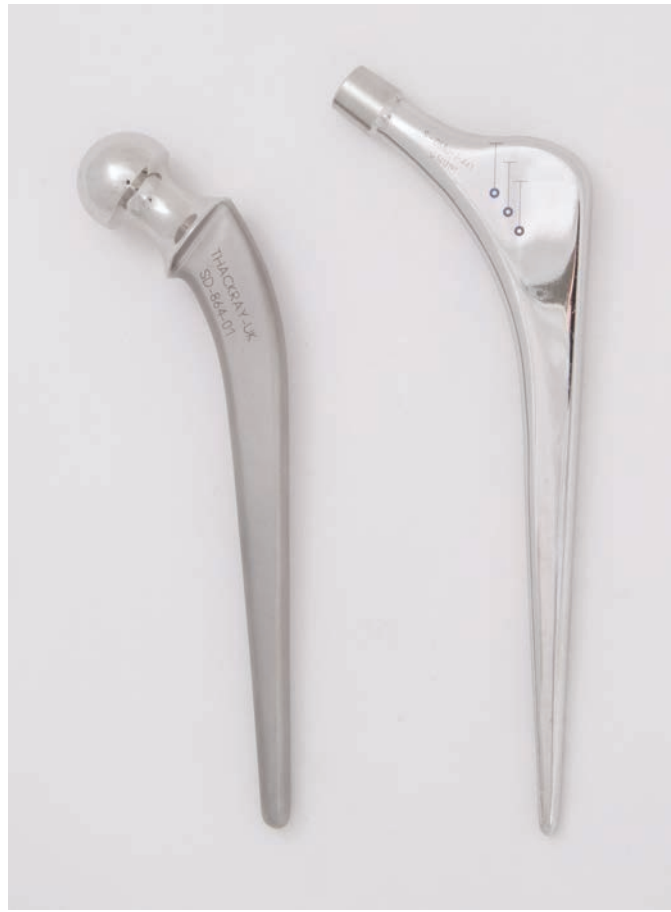


Figure 31.20 Exeter and Charnley femoral stem

It is such a well-known viva question that we know at least three model answers in various FRCS (Tr & Orth) revision books. The same (almost) identical pictures of a Charnley and Exeter femoral stem are shown (Figure 31.20), but the answers vary in detail from bullet-point headings to complicated biomechanical accounts.

That all said it is surprising how candidates can still mess the question up. Candidates should be aiming for a 7 not a 6, so be pro-active and rehearse your answer to your study group rather than passively reading the topic from a book. This tactic may make the difference between scoring a 6 or 7 and ultimately between passing and failing the exam.

Usually a laminated clinical photograph is shown of an Exeter and Charnley femoral stem (Figure 31.20). Previously an examiner may have brought the stems along to hand to candidates but for exam consistency all examiners now have the same standard question and clinical photographs to show to candidates

EXAMINER: What are these components?

CANDIDATE: This is an Exeter and a Charnley femoral stem. Both are cemented stems but have a different design philosophy.

EXAMINER: What are the characteristic features of these designs?

CANDIDATE: The Charnley stem is a matt finished monobloc, round back stem with a 22.25 mm femoral head. It is an example of a composite beam design that has a collar or flange and a rough surface finish.

The Exeter stem is a collarless, highly polished, double tapered stem.

EXAMINER: Which material are they composed of and why?

CANDIDATE: The Exeter femoral stem is manufactured from a stainless steel alloy (Orthinox®). This has high strength with ductility and a high corrosion resistance. The Charnley stem is also manufactured from a stainless steel alloy (Ortron®) for the same reasons.

EXAMINER: What about their design philosophy. How do they work?

CANDIDATE: In the composite beam model, the stem is considered a rod within two tubes, an inner cement tube and an outer bone tube. The composite beam depends on strong/perfect bonding at both the prosthesis-cement interface and the bone cement interface to form a stable construct from three materials with different mechanical properties: Metal, cement and bone

Composite beam implants have rough surfaces to promote bonding with the cement and usually have a collar designed to load the calcar.

Once inserted the stem should not move and any debonding or subsidence, at the implant–cement or bone–cement interface represents loosening and failure.

The load is transmitted from the prosthetic femoral head via the stem to its tip, by passing the proximal femur. From the stem tip, the load is transferred to the distal bone cement and subsequently to the host bone.

Composite beam fixation relies on perfect bonding between the stem and cement at all times and for all loading conditions.

Charnley pioneered the concept of low frictional torque. It is based on the largest possible difference between the radius of the femoral head and that of the outer aspect of the acetabular component. As the movement takes place at the smaller radius (the articulation) the bone–cement interface is protected. This is achieved in clinical practice with the use of a 22.25-mm diameter head as part of a monobloc stem. Charnley's original flat-back design was polished and tapered in the anteroposterior dimension. Charnley perceived the subsidence he observed in his early series as undesirable, despite its low rates of loosening. Therefore, he introduced design features such as the Cobra flange and surface texturing to resist subsidence. This heralded the composite beam philosophy of cemented stems, which ultimately led to stems with design features and surface texturing specifically intended to not only resist subsidence, but to achieve rigid fixation of the implant to the cement. To improve the fatigue strength Charnley increased the cross-sectional area of the stem from a flat back to a round-back stem. In 1975, the addition of anteroposterior flanges aimed to enhance stability of the stem and to increase the interlock between the stem and the cement. The strength of this implant-cement interface became critical, as it needed to resist the shear forces generated by weight-bearing and loading of the prosthesis. The survival rates of the flanged grit blasted cobra shaped Charnley stem at 15–25 years were less satisfactory than those of the non-flanged polished version.

Taper-slip components are polished, collarless and taper in 2 or 3 planes. There is no bonding with cement and the stems are designed to subside within the cement mantle, which, therefore, must be regular and complete.

Stems are collarless to permit subsidence and polished to avoid abrasions of the inner surface of the cement mantle and the generation of debris.

The Exeter stem depends on the taper slip (or forced closed) engineering principle. Hoop stresses are transmitted to the femoral bone, which expands during loading of the component. The polished stem allows for subsidence within the cement mantle, which maintains stability of the implant and protects against loosening. The philosophy behind the design is that it anticipates stem-cement debonding, distributes the stresses evenly in the cement mantle and accommodates creep and stress relaxation in the cement mantle.

EXAMINER: What is bone cement[aa]?

CANDIDATE: Bone cement is polymethylmethacrylate.

In arthroplasty cement allows secure fixation of implants to bone. It is not a glue it has no adhesive properties.

EXAMINER: What is creep?

CANDIDATE: Creep is time-dependent deformation of a material in response to a constant load. Creep rate reduces with time. Cements with higher porosity and viscosity are less resistant to creep deformation. Creep is most prevalent under high stresses (and temperatures). The creep resistance of bone cement results in reduction of the prosthesis subsidence in the cement mantle.

EXAMINER: What is the function of the centralizer?

CANDIDATE: The Exeter centralizer allows the stem to move distally within the cement mantle, whilst at the same time preventing end bearing of the stem directly onto the cement.

The stem is known to subside into an air-filled centralizer, which leads to low shear stresses, high compressive stresses and almost no tensile stress. This in turn increases axial and torsional stability. This subsidence also seals off the stem-cement interface to prevent any fluid flow, which may lead to loosening (effective joint space). A centraliser also improves stem position and the quality of the distal cement mantle.

The load is transmitted from the prosthetic femoral head and forces the taper to subside within the cement mantle. This subsidence creates radial compressive forces within the cement and subsequent hoop stresses within the bone. This stabilises the bone–cement–prosthesis composite. The visco-elastic properties of cement (creep and stress relaxation) are important determinants to the process.

EXAMINER: What balances the hoop stresses generated within the bone?

CANDIDATE: The fibers arrangement within bone counterbalances the hoop stresses generated within the cement. A similar effect can be seen in the meniscus or as metal hoops around a barrel.

[aa] We were surprised candidates were then asked about cement and creep with this question, but it relates to the properties of cement allowing subsidence.

EXAMINER: How do they fail?

CANDIDATE: Composite beam stems fail when movement occurs at the prosthesis-cement interface. A rough surface finish will abrade the cement mantle once micromovement is established. This most likely leads to gap formation that in turn further increases micromovement and also allows the circulation of wear debris.

Composite beam fixation relies on perfect bonding between the stem and cement at all times and for all loading conditions. Evidence suggests that this bond does not exist even immediately following stem insertion and that a gap exists between stem and cement that allows fluid migration and the formation of a membrane at this interface. Such a gap may exist due to thermal shrinkage of cement or imperfect cement interdigitation into the stem surface features. Further studies have shown that during the lifetime of a hip replacement the stem further debonds from the cement opening up a gap between the cement and stem.

A composite beam stem mode of fixation applies shear stresses on the cement implant interface in which the loading cement mode is weak and is, therefore, prone to failure and debonding

Polished, tapered stems are inherently unstable implants and their mechanism of failure is not well established. RSA studies suggest that these devices fail when they rotate in the axial plane.

EXAMINER: Do you know of any material changes that occurred with the Exeter stem?

CANDIDATE: When the Exeter stem was changed to a matte finish but this had a higher loosening rate. Polishing is labour intensive and expensive but the revision rate for the matt finish was found to be 10% at 10 years compared to 2.5% for a polished surface.

EXAMINER: Why the higher loosening rate with a matt surface finish?

CANDIDATE: The matte surface led to the stem bonding with cement although biomechanically it is not designed for this. The matte taper slip is, therefore, unable to subside in cement and convert the shear forces to compressive forces.

Although there are many potential routes of questioning two routes come to mind:

The candidate's answer has hinted at the concept of effective joint space. The examiners may chose to go down this path or they could just move onto the long-term clinical results of these hips.

EXAMINER: What are their long-term clinical results?

CANDIDATE: The results of both stems are excellent. The Exeter femoral stem has a 13-year survival of 94.4%, while the Charnley femoral stem has a 25-year survival of 86.5%. Both the Exeter and the Charnley stems have a 10A ODEP rating.

COMMENT: The viva would usually have run out of time at this stage but if a candidate is doing extremely well and covers the ground fast the discussion may lead onto a number of more complicated higher-order thinking areas.

EXAMINER: What do we mean by the term effective joint space?

CANDIDATE: Schmalzried et al. coined the term 'effective joint space' to describe areas of localized bone loss containing intracellular polyethylene debris remote from the actual joint space.

According to their hypothesis, debris can migrate, along a pressure gradient following the path of least resistance and driven by intracapsular pressure, to all areas that are accessible to joint fluid.

With increasing flow, particles accumulate locally and as the concentration of particles rises, the process of osteolysis is initiated. The local collection of particles encourages further particle accumulation and an expansile lesion is, thus, created. In situations where the flow of particles is less and an adequate fibroblastic response is mounted, the resorption progresses at a lower rate and a more linear pattern of osteolysis along the interface is evident

Schmalzried postulated that a barrier would block or delay this flow of debris, thereby protecting the bone–implant interface.

Classic reference

Schmalzried TP, Jasty M, Harris WH. Periprosthetic bone loss in total hip arthroplasty: Polyethylene wear debris and the concept of the effective joint space. *J Bone Joint Surg Am.* 1992;74:849–63.

Massachusetts General Hospital Boston

Schmalzried et al. introduced the concept of effective joint space (EJS) in total hip arthroplasty (THA), which refers to a path in the periprosthetic region for the passage of the particulate debris away from the articulating surfaces. It includes all periprosthetic regions that are accessible to joint fluid and wear debris. Wear particles are not only found in the joint, but also in the implant–bone interface suggesting that the synovial cavity is in continuity with this interface.
An understanding of this concept is important as any reduction in the EJS could, in theory, reduce the area that can potentially undergo osteolysis.

Broken femoral stem

It is quite possible candidates may be shown a radiograph or photograph (Figure 31.21) of a broken stem in a basic science or adult pathology viva exam.

Candidates would first need to describe the radiograph. The stem may be either a loose broken cemented stem or an uncemented stem.

If cemented we suggest to proceed with describing the Gruen mode of femoral stem failure (almost invariably four) and for completeness sake continue on to describe the other three Gruen modes of failure.

The examiners may just want to concentrate on the basic science of metal fatigue properties. The other route if in the adult pathology viva is to follow on and discuss revision options and preoperative preparation for surgery. Mentioning specific practical technical difficulties encountered

Figure 31.21 Photograph of stem retrevial due to stem fracture

at surgery and how to overcome them would score candidates a 7 or 8[bb].

Uncommon with a reported incidence of 0.27%. Femoral stem fractures are much less common now with stronger alloys. Cracks commonly begin on anterolateral surface of femoral component where the stems taper and become thin. The fracture usually begins in the middle third of the anterolateral aspect of the stem and progresses medially.

EXAMINER: What causes femoral stem fractures?

CANDIDATE: Predisposing factors to stem fracture include:

- Stems with decreased cross-sectional area and long necks
- Increased incidence with heavy (obese), active patients, especially if the stem is undersized
- Inadequate calcar cancellous bone removal (which leads to undersizing of the femoral stem)
- Poor cement mantle (mention Barracks cement grading)
- Varus positioning of stem
- Stainless steel components
- Poor support in the proximal one-third(cantilever bending)
  - When a cemented stem is well-fixed distally but loose proximally
  - Cement debonding of the femoral stem along the proximal-lateral aspect is often a precursor to fracture
  - Osteolysis underneath the cement collar
- Lateral stem nicks produced by drilling for greater trochanteric wires may lead to fatigue fracture

EXAMINER: What do we mean by fatigue failure?

CANDIDATE: Fatigue failure refers to failure of a material with repetitive loading at stress levels below the ultimate tensile strength.

EXAMINER: Can you draw me a S–N curve?

COMMENT: Candidates should be about to draw out an S–N curve explaining various terms as they go along.

The endurance limit is defined as the stress at which the material can withstand 10 million cycles without experiencing fatigue failure.

EXAMINER: How does a stem fracture occur?

CANDIDATE: The material has notch sensitivity. This is the extent to which the sensitivity of the material to fracture is increased by the presence of surface inhomogeneity(scratches or cracks).

COMMENT: This is a score 7 question and is getting beyond the basics of a pass.

EXAMINER: What happens to the metal to cause failure of the implant?

CANDIDATE: Fatigue fracture occurs in three steps:

1. Nucleation of a crack. This occurs at locations of highest stress and lowest local strength. These are usually at or near the implant surface and include surface defects, such as scratches or pits, sharp corners, inclusions, grain boundaries or dislocation (linear defect) concentrations
2. Propagation of a crack. Towards the lower stress regions. The crack propagates a little bit further with each cycle, until the load-carrying capacity of the metal is approached
3. Catastrophic failure. This occurs in a brittle manner. Often the fatigue crack region can be distinguished from the final fracture region by beach marks. Beach marks are macroscopically visible semielliptical lines running perpendicularly to the direction of fatigue fracture propagation

The entire fatigue process involves the nucleation and growth of a crack or cracks to final fracture. The fatigue crack size at fracture can be very small or very large, occupying <1% of the fracture surface up to almost 100%.

EXAMINER: How do you revise a broken stem[cc]?

CANDIDATE: Extraction of a broken femoral stem is problematic because the remaining distal portion of the stem may be held rigidly in its cement bed. The proximal stem fragment is removed in the usual manner. The distal stem fragment is removed by creating a window in the anterior cortex just below the level of stem fracture. After making a window down to the metal surface, a sharp-tipped punch is used to divot into the metal and push the distal metal fragment proximally. As the stem moves proximally, additional divots are then made.

EXAMINER: What about uncemented stems?

CANDIDATE: This can be very difficult.

Removal of an extensively porous-coated femoral stem can be problematic because bone ingrowth occurs along the entire length of the coating[8]. If the stem is fractured, gaining access to

[bb] Demonstrates candidates have been 'on the job' in theatre rather than just reading the material from a book.

[cc] This is a clinical question that the examiners may avoid asking as they may well prefer to stick to basic science theory.

Figure 31.22 Photograph of the three main types of uncemented femoral stem

the distal piece presents an additional challenge. Removal of a well-fixed component requires disruption of the bone-prosthesis interface circumferentially, and concerns have been raised about weakening of the bone due to the 'gutters' which are cut into the surrounding cortex. Hollow trephines have been developed which are sized to allow over drilling of the distal femoral stem while minimizing cortical bone loss. However, successful use of these devices depends upon the fracture being through the cylindrical portion of the stem. If the fracture is more proximal, a larger trephine would be required, with an increased risk of eccentric reaming and perforation of the cortex.

Alternatively, direct access to the femoral stem can be gained via a cortical window (which provides only limited access) or a femoral osteotomy. Paprosky[9] has described an 'extended trochanteric osteotomy' which allows wide access to the femoral canal for removal of an intact or broken stem. With this technique, a tongue of bone comprising one-third of the femoral circumference is elevated, with the soft-tissue attachments of the gluteus medius and minimus and vastus lateralis left intact. At the end of the case, the osteotomy is reattached using multiple cables or cerclage wires. Excellent outcomes have been reported using this technique, with a non-union rate of only 1% at nearly 4-year follow-up.

Rather than the Exeter/Charnley hip question the examiners may decide to ask instead on uncemented femoral designs. The three main uncemented femoral stem designs are: (1) grit-blasted on-growth; (2) porous-coated in-growth; and (3) hydroxyapetite coated. Candidates may be questioned on one or all of the stem feature designs[dd]. The classic hydroxyapatite coated stem candidates may be shown is the Corail stem. Hydroxyapatite is a calcium phosphate compound that is used as an adjuvant surface coating on porous-coated and grit-blasted surfaces. HA is an osteoconductive agent and enhances

[dd] This topic has been dealt with in the hip core topic chapter

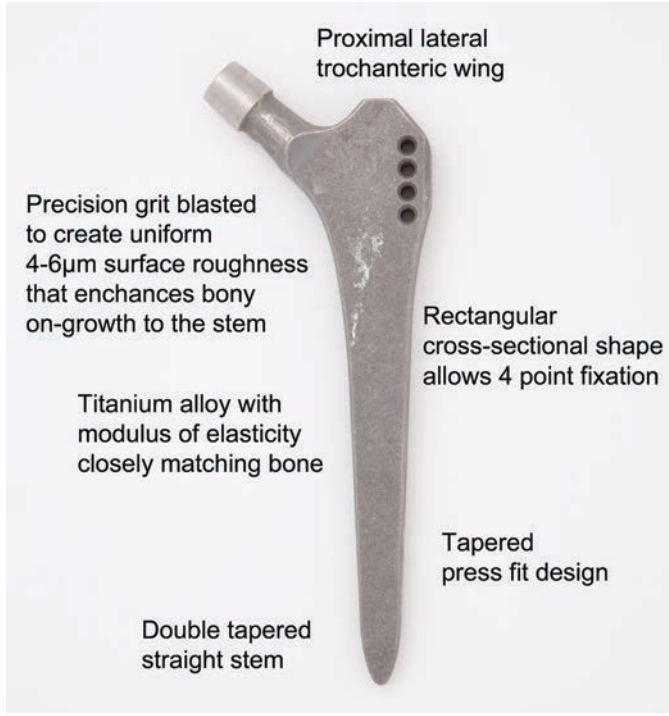


Figure 31.23 Photograph of a Zweymüller® uncemented femoral stem retrevial. This relies on bone ongrowth. Note the rather sparse amount of bone on the surface of this prosthesis

growth of mineralized bone onto the implant by providing bone with its mineral phase substrate. HA allows for more rapid closure of gaps its surface readily receives osteoblasts and, thus, provides a bi-directional closure of gaps (i.e. bone to prosthesis and prosthesis to bone), which clinically shortens the time to biological fixation. The optimal thickness of hydroxyapetite is 50–75 μm. Thicker coatings have been reported to delaminate off the prosthetic interface. The Zweymüller® uncemented femoral stem is the classic grit-blasted femoral stem that may be shown (Figure 31.23). It has a rectangular press-fit design, is made of titanium-aluminum-niobium alloy (Ti-6Al-7Nb), and has a modulus of elasticity closely matching bone that reduces both thigh pain and stress shielding

The rectangular cross-sectional shape allows 4-point fixation along the 4 corners within the femoral canal

The dual wedge taper (wedge in both coronal and sagittal planes) is tightly fitted through the metadiaphyseal portion of the proximal femur locking the stem in a secure axial position, while the rectangular shape anchors the stem rotationally

The stem works on a fit-without-fill principle that provides axial and rotational stability

The entire stem is precision-grit-blasted to create a uniform 4-6μm surface roughness that enhances bony on-growth to the stem. Any number of femoral uncemented porous-coated stems may be shown (Figure 31.24). Start by describing general features of porous coated stems. If you are lucky you may recognise the prosthesis and be able to be more specific with your description.

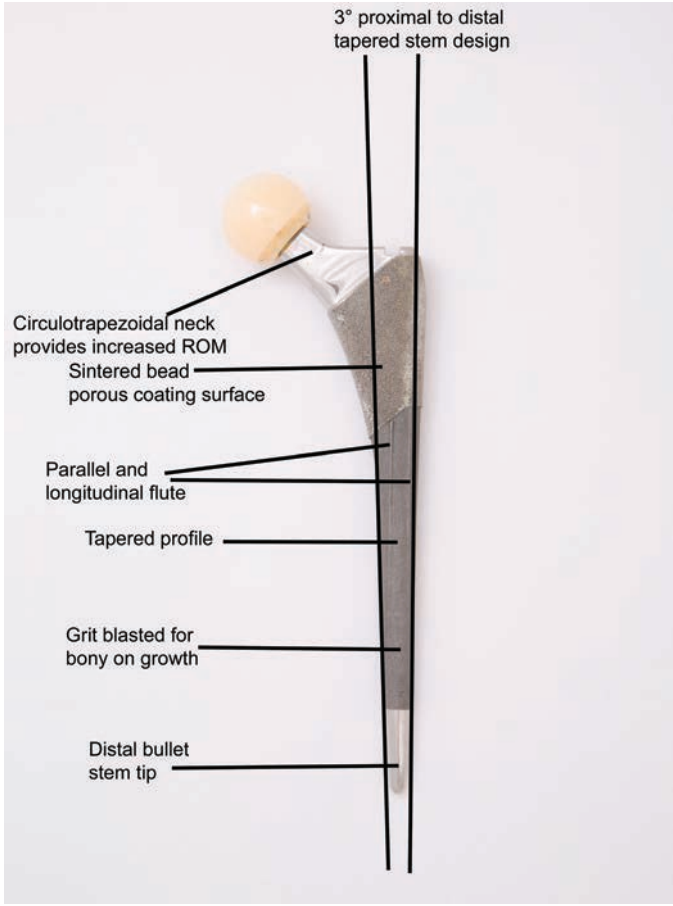


Figure 31.24 Photograph of the design features of a porus-coated uncemented femoral stem retrevial. The synergy stem is made of high-strength, forged titanium with a 3° proximal to distal tapered stem design. Parallel and longitudinally arranged flutes provide axial and rotational stability. Below the proximal third, the stem has been grit blasted for bony ongrowth. The distal bullet stem tip is polished to minimize distal endosteal irritation and thigh pain.

The synergy stem achieves initial stability through three-point fixation. Roughcoat sintered bead porous coating promotes bone ingrowth and a 3-dimensional interlocking at the bone implant surface. The circulotrapezoidal neck provides increased ROM compared to more traditional designs with less risk of dislocation. Be careful as below the proximal third, the stem has been grit blasted for bony ongrowth so it is not strictly speaking just a porous coated stem. This is true of a number of other porous stem designs.

Examination corner

Basic science oral 1

- Handed two uncemented femoral prostheses and asked to describe
- Discussion about the mechanisms of component wear

Basic science oral 2

- Aseptic loosening of hips
- Wear particles
- Sources
- Third body wear
- Measurement of linear and volumetric wear

Basic science oral 3

Airport metal detectors and various prostheses
Metals/alloys in prostheses
Statistics on new THA and mechanisms of testing wear
Fatigue failure

Basic science oral 4

'I was shown a clinical photograph of a failed tibial knee prosthesis that had been removed from a patient and asked to describe it (Figure 31.25)'
General discussion on wear
PE tibial insert – How can you improve its properties?
Cross-linking of polymer in PE
Discussion on strength, stiffness, etc

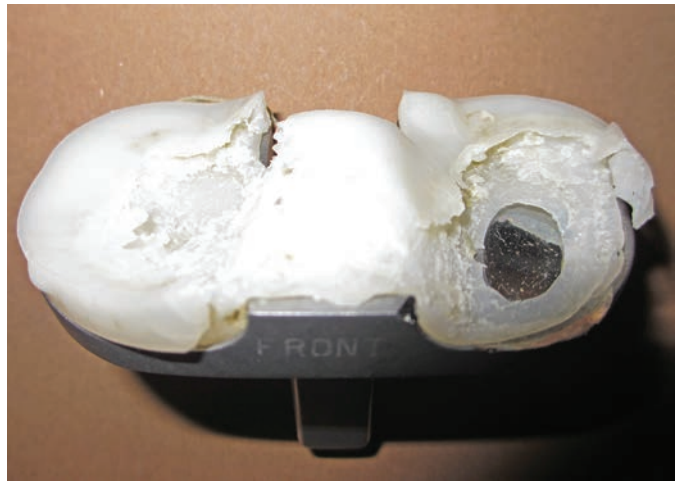


Figure 31.25 Photograph of retrieval tibial knee implant demonstrating catastrophic PE wear

Catastrophic PE failure

This topic is well covered in most textbooks each with a slightly different focus. It is important to dry run your answer beforehand to iron out any mistakes. The question is better suited to PE tibial knee wear rather than the hip although PE failure could be discussed as part of hip implant design or retrieval of a failed hip implant (Figure 31.27).

EXAMINER: What is going on here? (Figure 31.25)

CANDIDATE: This is a picture demonstrating catastrophic PE wear.

EXAMINER: What do we mean by catastrophic PE failure?

CANDIDATE: This refers to macroscopic premature failure of polyethylene.

EXAMINER: What are the causes for this?

CANDIDATE: It has been attributed to numerous factors including poor surgical technique, reduced polyethylene thickness, poor locking mechanisms of modular fixed-bearing tibial components, gamma

Figure 31.26 Anterior photograph of retrieval cruciate retaining (CR) total knee arthroplasty implant demonstrating catastrophic PE wear. The PE insert is generally more flat to allow femoral rollback onto the posterior part of the bearing

Figure 31.27 Photograph of retrieval acetabular cup implant demonstrating cracking and delamination of its edge from neck impingement

irradiation sterilisation techniques of PE in the presence of oxygen, knee kinematics and low conformity implant designs.

- COMMENT: The answer whilst correct is a bit haphazard and incomplete lacking the structure needed to maximize marks:
- PE thickness
- Articular surface design
- Knee kinematics
- PE sterilisation
- PE machining
- Surgical technique

It is likely the examiners will want to know more details for each factor.

EXAMINER: What surgical factors predispose to catastrophic failure?

CANDIDATE: Significant varus alignment of the knee, excessive femoral rollback.

COMMENT: A tight flexion gap accelerates slide wear. This can occur secondary to a tight PCL or anterior tibial slope.

EXAMINER: What do we mean by excessive femoral rollback?

CANDIDATE: Femoral rollback is the posterior translation of the femoral–tibial contact with progressive flexion. Femoral rollback optimizes flexion at the cost of increasing contact stress and catastrophic failure.

EXAMINER: So how does this cause catastrophic failure of a TKA?

CANDIDATE: There is dyskinetic sliding movement on the femur on the tibia causing increased contact stress and increasing the risk of catastrophic failure

EXAMINER: How do we reduce femoral rollback?

CANDIDATE: There are several strategies available. These include increasing the posterior cutting slope of the tibia, using a more congruous knee design or using a PCL substituting knee replacement for an incompetent PCL.

EXAMINER: How do we improve surgical technique?

CANDIDATE: This can be achieved by improvements in surgical instrumentation such as better cutting jigs, intramedullary alignment guides, blocks to measure ligamentous balancing. The surgeon should be ideally fellowship trained in arthroplasty surgery, perform a minimum number of knee replacements per year, have attended various knee arthroplasty training courses, kept themselves up to date.

PE thickness

PE thickness should be at least 8 mm. The classic paper to quote is from Bartel et al.[10]

Classic reference

Bartel DL, Bicknell VL, Wright TM. The effect of conformity, thickness, and material on stresses in ultra-high molecular weight components for total joint replacement. *J Bone Joint Surg Am.* 1986;68:1041–51.

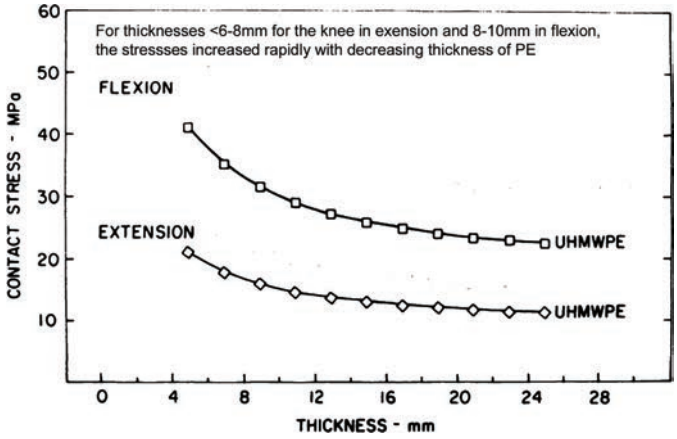


Figure 31.28 Variation of the maximum (compressive) contact stress on the surface of the tibial component with varying thicknesses of the polyethylene layer

Early failure of polyethylene knee components is a major concern to orthopaedic surgeons.
Studies to determine the source of failure began with the investigation of the contact stresses, which revealed that designs with the highest contact stresses often had higher rates of damage than more congruent designs. Design improvements led to increased contact areas between metal and polyethylene components, resulting in reduced contact stresses.
Bartel et al. demonstrated the importance of conformity in prosthetic TKA design to increase contact area and to decrease contact stress. The greater the conformity, the greater the articular contact area with resulting reduced subsurface polyethylene contact stress per unit of area and, therefor,e less polyethylene wear.
The authors demonstrated that the contact stresses in polyethylene inserts <6 mm thick increase exponentially as the thickness of the implant decreases (Figure 31.28). For example, in very thin components, a small reduction in thickness generates a large increase in contact stress and less favourable wear characteristics.
Bartel et al. used finite element techniques to analyze the role of PE thickness on wear. These analyses indicated that bearing thicknesses <6–8 mm would result in increased maximum shear stresses occurring 1–2 mm below the surface. This depth corresponds to the location of cracking and delamination, also referred to as fatigue failure, seen in retrieved knee components.

Articular surface design

In general there are two designs in total knee prosthesis:

1. A deeper congruous shape without rollback. This is less anatomic, mimimizes contact loads (i.e. force/area) and decreases contact stresses
2. A flat tibial PE. This improves femoral rollback and optimizes flexion. It is more anatomic, PCL sparing but increases contact stresses and the risk of catastrophic failure (Figure 31.26)

Avoid flat PE tibial designs as the low contact surface area leads to high contact stresses in areas of contact. The newer prosthesis design have a more congruent design in both coronal and sagital plane in order to decrease contact stresses. Goal is to maximize contact area and minimize contact loads.

Knee kinematics

Two variables that affect knee kinetics are: (1) femoral rollback; and (2) knee alignment.

Sliding wear is deleterious for PE. Sliding wear occurs when the ACL is sacrificed. When the ACL is removed and the PCL retained, the femur slides across the tibial PE during flexion and extension. Sliding movements are most marked in a CR knee with a flat PE insert. Rollback can be reduced by using a more congruous joint design, increasing the posterior slope of the tibia and inserting a CS knee. It is important to minimise dyskinetic femoral rollback by avoiding use of a CR knee with an incompetent PCL.

Avoid varus knee by an accurate tibial cut and medial release of soft tissue.

EXAMINER: How do you avoid a varus slope of the knee?

CANDIDATE: Most surgeons follow Install et al.'s teaching that the tibial resection should be at 90° to the tibial axis in the coronal plane.

EXAMINER: How do you avoid a varus slope in a knee replacement?

CANDIDATE: By accurate placement of the extramedullary tibial alignment jig. Palpable landmarks include tibial tubercle, tibial spines, medial and lateral malleolus. The jig should be positioned over the tibial tubercle and aligned parallel with the tibial spines, whilst intersecting the intermalleolar distance. The jig should line up with the second ray of the foot. Just before cutting with a saw the surgeon should also double check by using an angel wing inserted into the saw slot wrapped around bone to visualize the cut before it is made and lastly also eyeballing the tibial cut.

Surgical technique

Avoid a tight flexion gap and as mentioned an excessive varus tibial slope. A tight flexion gap accelerates sliding PE wear compounded by either a tight PCL or anterior tibial slope.

PE manufacture

Avoid sterilization in an oxygen-rich environment as this allows free radical formation as this diminishes resistance to adhesive and abrasive wear.

COMMENT: PE manufacture is easily a 5-minute viva topic in itself and involves discussion of processing, machining and sterilization.

Basic science oral 4

Exhibit – A stainless steel femoral component was presented and the candidate was asked to comment.

CANDIDATE: It's worth taking a second or two to look carefully at the prosthesis before launching in. The trick here was to recognise that it had been used and retrieved, but avoid using the word 'failed' in your assessment unless pressed as the examiner might feel piqued by your judgement, and it could be that it was retrieved from a cadaver and did not fail!

Apart from noting the general features of a hip femoral prosthetic component, look for wear on the shaft and comment on it. The wear pattern may well give away the side of

the patient on which it was used (think about rotational forces at the femur/cement interface with the prosthesis) and you will get 'gold stars' for this. Avoid saying the specific type of prosthesis unless you are certain – You are safe saying collarless/collared, etc.

If it's going well you might be led off into a discussion about offset, cement technique, and the place of cementless stems.

Examination corner

Basic science oral 1

What is corrosion?

What are the different types of corrosion?

Total reverse shoulder arthroplasty[11]

Lead-in prop would be either a picture (Figure 31.29) or radiograph shown of total reverse shoulder arthroplasty (RTSA). This topic can cross several viva boundaries including basic science of implants, trauma or adult and pathology.

EXAMINER: What are the indications for reverse shoulder replacement?

CANDIDATE: Total reverse shoulder arthroplasty is used in patients with a non-functional rotator cuff muscles. The two main indications are rotator cuff tear arthropathy with osteoarthritis and massive, irreparable rotator cuff tear(without established osteoarthritis). Cuff tear arthropathy usually affects elderly patients over 80 years of age with symptoms of night pain and pseudoparalytic arm. Many patients are unable to live independently and there was no effective treatment for these patients until the development of RTSA. With massive irreparable rotator cuff tear a patient should have significant pain and <90° of active forward flexion. Much better functional outcome scores and better range of motion reported than those patients who had >90° forward flexion prior to surgery.

Other indications include rheumatoid arthritis, revision of failed hemiarthroplasty and displaced proximal commuinuted humeral fractures in the elderly with poor quality rotator cuff muscles and healing of the tuberosities .

EXAMINER: What is the functional deficit created by a massive rotator cuff tear?

CANDIDATE: The rotator cuff stabilises the humeral head preventing it from translating superiorly during abduction. With a massive cuff tear the superiorly acting deltoid force is unapposed. The biceps is also usually torn and its depressing affect is also lost. The humeral head migrates superiorly and wears against the superior aspect of the glenoid and the undersurface of the acromium.

EXAMINER: What are the design features of the RTSA?

CANDIDATE: The humeral component is a stemmed implant with a polyethylene cup. The stem is either cemented or uncemented depending on bone quality. The humeral component has a non-anatomic head/shaft angle of 155°. A humeral PE socket is assembled to the humeral stem via a Morse taper. The glenoid component consists of a porous-coated baseplate that is secured to the glenoid with a central screw and multiple peripheral screws (cementless fixation). A large metal hemispherical ball (glenosphere) is attached to the metal base plate. The humeral cup covers less than half of the glenoshere. The humeral stem is positioned in 25–40° retroversion and the glenoid baseplate tilted inferiorly 10–15°.

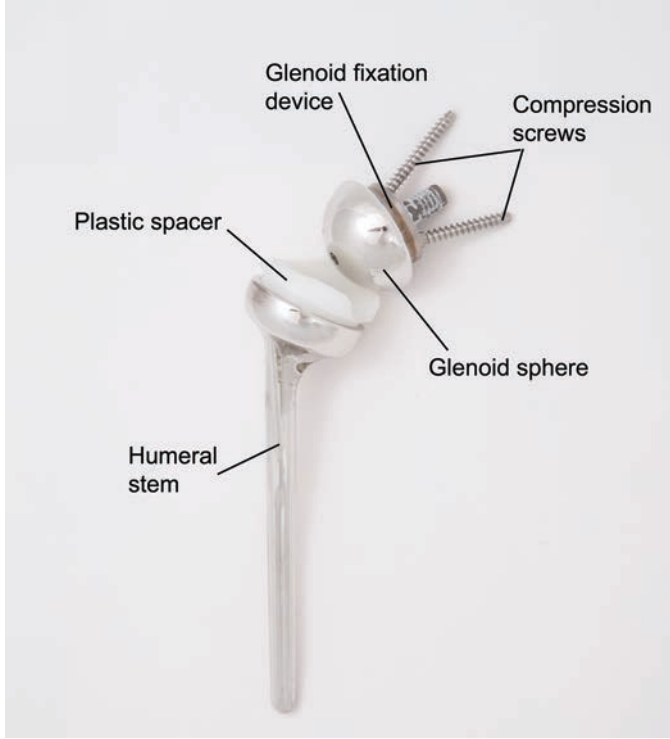


Figure 31.29 Reverse shoulder replacement with scapula notching shown

EXAMINER: How does the RTSA function?

CANDIDATE: The RTSA translates the centre of rotation medially and distally. Biomechanically this has four main effects: (1) lengthens the lever arm of deltoid so that it works more effectively in the absence of supraspinatous; (2) allows the recruitment of more deltoid muscle fibers for elevation and abduction; (3) reduces the torque on the glenoid component and; (4) lowers the humerus compared to the glenoid which increases deltoid tension.

EXAMINER: What factors affect RTSA stability?

CANDIDATE: The stability of RTSA is affected by head diameter, component positioning and deltoid tension. Head size is constrained by the size of the metaphysis.

EXAMINER: What position typically causes dislocation?

CANDIDATE: Dislocation typically occurs with hyperextension and external rotation and is anterior.

COMMENT: The key to stability is the correct tension in the deltoid and correct version of the components. The tension depends upon implanting the correct thickness of polyethylene spacer.

EXAMINER: What are the criteria for RTSA?

CANDIDATE: Criteria include: (1) full function of deltoid; (2) intact axillary nerve; (3) no osteoporosis; (4) adequate glenoid bone stock; (5) low patient activity demands; (6) limited overhead use.

EXAMINER: If you look carefully at the plastic PE cup at the bottom, what is going on?

CANDIDATE: There is wear of the PE.

EXAMINER: What causes this?

CANDIDATE: I am not sure.

COMMENT: This is for a 7 or 8 score. The question is the sting in the tail for the basic science and depends on on how far the examiner pushes things.

EXAMINER: Have you heard of notching?

CANDIDATE: Notching is due to repetitive contact between the polyethylene of the humeral component and the inferior scapular neck during adduction. It is a complication specific to TRSA and is defined as resorption of the lateral pillar of the scapula.

EXAMINER: So this is a notch in the PE not bone?

CANDIDATE: I'm not sure.

COMMENT: Notching can cause pain and lead to either fracture or dislocation. What the examiner was trying to draw out of the candidate was that notching leads not only to scapular neck erosion but also polyethylene wear, possible joint inflammation, and potential implant loosening.

EXAMINER: What causes notching?

CANDIDATE: Factors associated with the development of scapular notching include prosthetic design, surgical approach, positioning of the glenosphere, preoperative diagnosis, and pattern of glenoid wear occurring during the degenerative process.

EXAMINER: How do we minimize the risk of notching?

CANDIDATE: Notching is reduced by: (1) positioning the glenoid base plate (and glenosphere) as low as possible on glenoid bone; however, the deltoid is lengthened, which may lead to a higher incidence of acromial fracture; (2) optimizing glenosphere head diameter. This pushes the humeral socket away from the scapula; (3) using an eccentric glenosphere, but this could lead to an increased incidence of glenoid loosening; (4) using a shallow PE spacer, but this could lead to a higher incidence of instability.

EXAMINER: What are the complications of RTSA?

CANDIDATE: Complications include nerve palsy, acromial fractures, dislocation, glenoid loosening, infection and notching

COMMENT: Score 8 candidates may be asked about the Sirveaux classification of scapula notching (Figure 31.30).

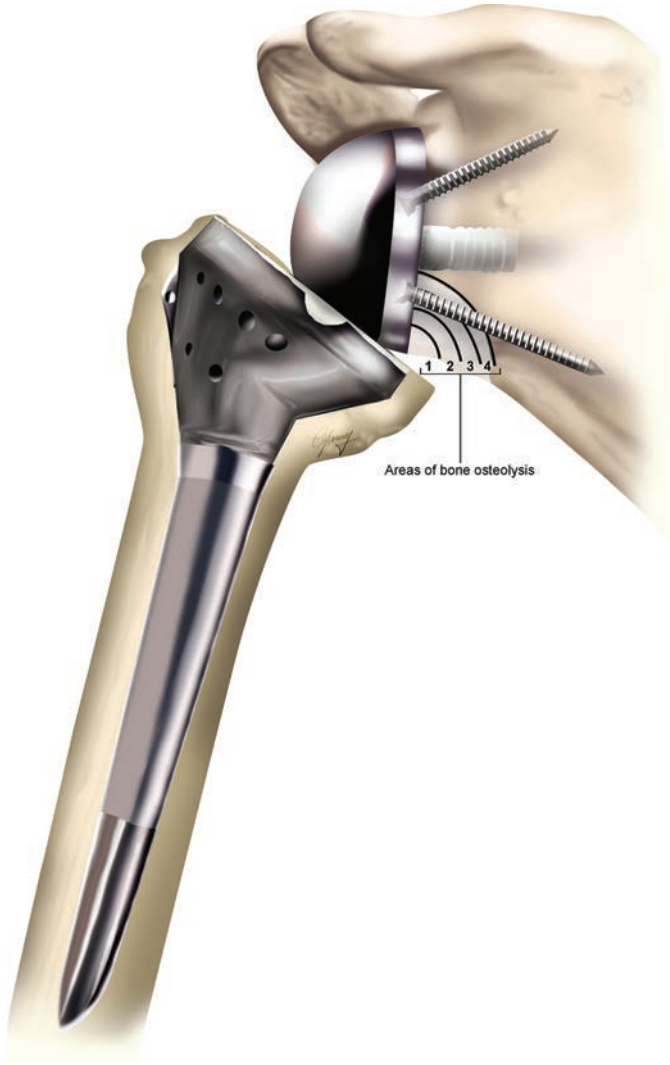


Figure 31.30 The Nerot–Sirveaux grading system for postoperative scapular notching after reverse TSA is illustrated. A grade 1 notch is a defect contained within the inferior pillar of the scapular neck. A grade 2 notch is erosion of the scapular neck to the level of the inferior fixation screw of the glenosphere baseplate. A grade 3 notch is extension of the bone loss over the lower fixation screw. A grade 4 notch is progression to the undersurface of the baseplate

Biomaterials

Cement

This is a straightforward topic with no real tricks. A good first question. There are no excuses for not working through an answer in a study group or viva practice session. Be slick with your answer. The only potential difficulties that may occur are if the examiners ask obscure non-textbook questions.

EXAMINER: What is cement?

CANDIDATE: Bone cement is polymethylmethacrylate (PMMA).

EXAMINER: How does it work?

CANDIDATE: It allows secure fixation of an implant to bone. It acts as a grout not a glue, it has no adhesive properties. It is a space-filling load transferring material. There is no chemical bond between the PMMA and the bone or implant surface.

EXAMINER: What do we mean by curing (polymerisation) of PMMA?

CANDIDATE: Carbon to carbon double bonds are broken down and new single bonds are formed to give long chain polymers that are largely linear and free of cross-linking.

EXAMINER: What are the phases of cement setting?

CANDIDATE:

Dough time: Starts from the beginning of mixing and ends when the cement will not stick to an unpowdered surgical glove (2–3 min).

Setting time: The time from the beginning of mixing until the surface temperature is half maximum (8–12 minutes).

Working time: The difference between the dough time and the setting time.

EXAMINER: What are the phases of the polymerisation process?

CANDIDATE: When the polymer powder and monomer liquid meet, the polymerisation process starts. During polymerisation of the monomer, the original polymer beads of the powder are bonded into a dough-like mass.

The polymerisation process can be divided into four different phases: Mixing, waiting, working and setting.

Mixing phase: In the mixing phase, the cement should be mixed homogeneously, minimizing the number of pores. Vacuum mixing has been shown to reduce the cements porosity and increase its mechanical strength

Waiting phase: During this phase, the cements achieve a suitable viscosity for delivery of bone cement. The cement is still a sticky dough

Working phase: The working phase is the period during which the cement and the implant can be introduced with ease. The cement must not be sticky, and its viscosity should be suitable for application. If viscosity is too low, the cement may not be able to withstand the bleeding pressure and prevent blood from entering the cement

Setting phase: During this phase, the cement hardens and sets completely. Hardening is influenced by the cement temperature, the OR temperature as well as the body temperature

EXAMINER: What factors affect the curing of PMMA (polymerisation–exothermic reaction)?

- Rapid mixing shortens the dough time
- Increases in room temp. shortens both the dough and setting times by 5% per degree °C and decreases have the opposite effect
- Increased humidity decreases setting time

EXAMINER: What are the mechanical properties of cement?

CANDIDATE:

Poor tensile strength

Moderate shear strength

Strong in compression

Weak in shear

Brittle and, therefore, notch sensitive

Young's modulus is between cancellous and cortical bone – 2.2 GPa

Viscoelastic properties -undergoes creep and stress relaxation

EXAMINER: What is the composition of cement?

CANDIDATE: Cement is supplied as a liquid and a powder. After mixing the liquid and powder the final material becomes cement.

Packet contains:

- **Polymer** (PMMA powder). Particles of varying sizes and shapes of (influencing volume and viscosity)
- **Starter** (1% di-benzoyl peroxide) BPO
- **Radiopacifier**. Either zirconium dioxide (ZrO_2) or barium sulphate ($BaSO_4$). Zirconium dioxide is one hundred times less soluble than barium sulphate and has less effect on the mechanical properties of the cement
- **Colourant**. Chlorophyll is added, the colour makes the cement more easily visible in the operating room, especially during revision procedures
- **Antibiotics.** Usually gentamycin or a combination of antibiotics (such as gentamycin and clindamycin)

Vial of liquid contains:

- **Monomer.** Methyl methacrylate (MMA)
- **Initiator**/activator (accelerator)(3% DMP toludine). This encourages the polymer and monomer to polymerise at room temperature
- **Inhibitor/stabiliser** (hydroquinone). A trace amount is added to minimize monomer polymerisation during storage

EXAMINER: What are the factors affecting bone cement strength? (Table 31.6)

COMMENT: This question has caught out a large number of candidates even though it is in the textbooks.

EXAMINER: What antibiotics would you use in cement?

CANDIDATE: There are two main choices of antibiotics. Gentamicin is an aminoglycoside antibiotic. It is bactericidal, has a dose-dependent killing curve, remains stable when exposed to heat and is soluble in water.

Vancomycin belongs to the glycopeptides group of antibiotics. It is viewed as the antibiotic of choice in cases of periprosthetic infection caused by MRSA.

EXAMINER: What are the complications with using cement?

CANDIDATE:

- Hypotension
- Fat embolism
- Thermal necrosis of bone, impaired local blood circulation and predisposition to membrane formation
- Chemical necrosis of bone, due to the release or leakage of unreacted monomer liquid before polymerisation of the cement in the bone bed
- Third body wear from retained loose fragments
- Cement mantle defect
- Cement reaction

EXAMINER: What are the local tissue effects of PMMA?

CANDIDATE: Occlusion of the nutrient artery may lead to bone necrosis.

EXAMINER: Why do we vacuum mix cement?

CANDIDATE: Compared to hand mixing vacuum mixing prevents air entrapment in cement, reduces cement porosity, decreases the number of unbounded particles in cement and increases cement's mechanical strength. In addition vacuum mixing reduces monomer evaporation and exposure in the operating room.

EXAMINER: What are the dangers to the nursing staff mixing the cement? How do we create a safe working environment?

CANDIDATE: When mixing cement, avoid excessive exposure to monomer vapours, as they may irritate the respiratory tract and the eyes. Monomer exposure is regulated by law in many countries.

Liquid monomer is a potent solvent that is highly volatile and flammable. The monomer should not be allowed to come into direct contact with the skin or with rubber gloves.

Conventional surgical gloves (latex) provide only limited protection against monomer (up to 1 min). As an additional precaution when

Table 31.6 Factors affecting bone cement strength

Factors that can be controlled by the surgeon	
Antibiotic inclusion	Can lead to 5–10% loss of strength
Mixing speed	If mixing too fast or slow, strength loss can be up to 20%, frequency of beating and duration of mixing govern monomer loss from evaporation and porosity
Radioopaque fillers	Can reduce strength by 5%
Insertion pressure	Laminations harder to obliterate with increasing viscosity, pressurisation reduces laminations porosity, therefore, increases strength
Cement insertion	Laminations more difficult to obliterate with increasing velocity, pressurisation to reduce laminations and porosity
Centrifugation/vacuum mixing	Leads to increased strength by 10–25%
Factors that can be partially controlled by the surgeon	
Cement thickness	Optimal thickness is 2–5 mm. If too thick, stress shielding can occur in the surrounding bone. If too thin the cement can fracture
Constraint	The more constrained the cement is the more likely it is that compression is the resultant force rather than tension or shear
Inclusion of blood or tissue	Can decrease strength by up to 70% depending on the amount
Stress risers (sharp edges in implant)	Cement is notch sensitive
Factors that can't be altered by the surgeon	
Equilibrium moisture content	Weaker by 5–10% than dry cement
Age of the cement after implantation	Gradual 10% loss of strength due to chemical changes
Strain rate	Stronger at higher strain rates (viscoelastic)
Environmental temperature	10% weaker at body temp than at room temp/

handling MMA, an extra pair of polyethylene (PE) gloves should be worn. The PE gloves provide 5 minutes extra protection

Modern vacuum mixing systems remain closed up until cement delivery. All ingredients are added and mixed while the system is closed. The mixing and delivery of the cement should be performed with the same cartridge.

EXAMINER: How do we reduce the risk of cement reaction?

CANDIDATE:

Adequately pre-oxygenate the patient

Ensure no hypotension is present prior to cementing

Thorough pulsed lavage to remove fat and blood contaminates

Reduce pressurisation

EXAMINER: What are the grades of cementing?

CANDIDATE:

First generation:

- Hand mixing, finger packing

Second generation:

- Use of a medullary plug, delivery of a doughy mix of cement in retrograde fashion by a cement gun, use of a collared cobalt-chrome femoral stem with a rectangular cross-section and rounded corners. The medullary canal was irrigated and packed with sponges until immediately before cementing

Third generation:

- Vacuum mixing of cement to reduce porosity, pulsed lavage and brushing, cement pressurisation with air vent to improve cement interdigitation.

Fourth generation:

- Fourth-generation cementation techniques have added distal and proximal prosthesis centralizers to improve the stem position allowing for an optimal and even cement mantle.

EXAMINER: Why do we need a cement restrictor?

CANDIDATE: Distal intra-medullary plug will allow for cement containment and ensure adequate cement pressurisation.

It allows for improved cement penetration, shear strength and clinical outcome.

Cement restrictor failure will result in a poorer cement mantle and long distal cement plugs, which are difficult to remove in revision cases.

COMMENT: This question can faze a number of candidates who haven't really thought this through and are, therefore, caught off guard.

EXAMINER: How do we reduce the incidence of inclusions of blood and cement voids?

CANDIDATE: There needs to be an appropriate lavage and suction off all fluid in the femur and the surgeon needs to notice any pockets of blood and air remaining in a cavity.

A vent tube placed at the restrictor will remove trapped air and blood as cement is extruded from the femur.

Inserting cement at the correct viscosity.

Have the operative field clean of blood and tissue contaminants.

Centrifugation eliminates large cement voids.

EXAMINER: How do we grade the cement mantle in hip arthroplasty surgery?

CANDIDATE: Barracks grading.

An 'A' grade represents complete filling of the medullary cavity by cement, a so-called 'white-out' at the cement–bone interface

In grade 'B' the distribution of cement is nearly complete but it is possible to distinguish cortical bone from cement in some areas.

Grade 'C' presents radiolucencies involving 50–99% of the cement–bone interface or a void or deficiency is present in the cement mantle.

A grade of D is given if there are gross deficiencies in the cement mantle, such as no cement distal to the tip of the stem, major defects in the cement mantle or multiple large voids.

This classification was subsequently modified by Mulroy and Harris with C being divided into C1 and C2.

C1 occurs if there is an extensive radiolucent line (along more than 50% of the cement–bone interface) or voids in the cement

C2 occurs if there is either a thin (<1 mm) cement mantle at any site or a defect in the cement mantle, with the metal in direct contact with cortical bone

Examination corner

Basic science oral 1

You may be asked a straight question such as 'What is bone cement?' or shown a clinical photograph of a scrub nurse mixing cement (Figure 31.31).

The examiners started by asking me what the scrub nurse was doing.

CANDIDATE: I began by saying that the scrub nurse was mixing cement and I then just kept on talking about cement. I mentioned everything possible that I knew. My answer just seemed to come together very well and the examiners were very happy with my discussion.

I started by saying that cement functioned as a 'grout', a space-filling load-transferring material, that it was not glue, and that it had no adhesive properties. I then continued to discuss dough times, setting times and working times. It was a very detailed discussion and we covered just about everything possible you could talk about.

EXAMINER: This candidate was always a little self-depreciating about their oral performances but had prepared well for the topic and had obviously rehearsed the answer beforehand.

PE manufacture[12]

This is a big topic that the examiners may peripherally touch on if discussing implant design or wear. Some of this material is complicated so try and have a simplified structured rehearsed answer to make sure you score a 6 pass.

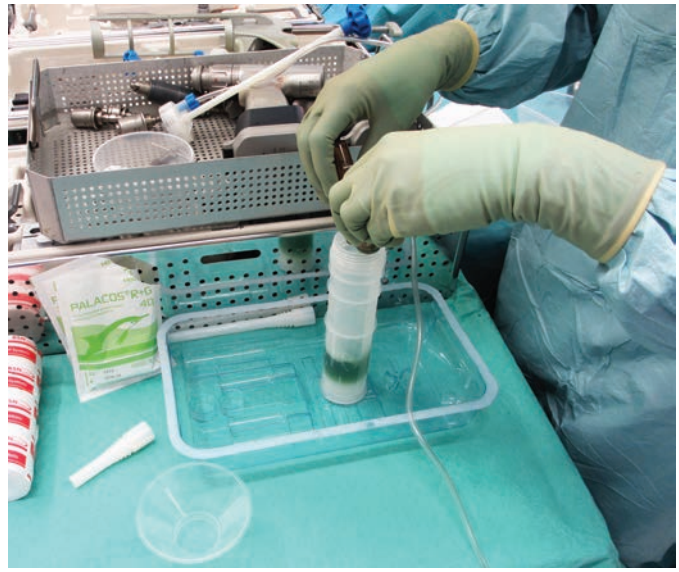


Figure 31.31 Mixing of bone cement

EXAMINER: What is this (Figure 31.32)?

CANDIDATE: This is UHMWPE from a total hip replacement.

Polyethylene$(C_2H_4)_n$ is a linear homopolymer of ethylene gas (C_2H_4) in which the double carbon bond is reduced to a single bond and ethylene molecules link up to one another forming a long chain.

COMMENT: Candidates may also be shown a picture of tibial UHMWPE from either a uni- or total knee replacement.

EXAMINER: How is ultra-high molecular weight polyethylene manufactured?

CANDIDATE: UHMWPE is manufactured by the Ziegler process involving ethylene gas, hydrogen, titanium tetrachloride (catalyst) and a solvent conducted at pressures between 4 – 6 bar at a temp of 66–80°C. Ethylene gas is polymerized in a low-temperature, low-pressure enviroment. This produces a fine UHMWPE powder that can be processed by one of several methods:

1. Ram extrusion

Produces bar stock, lowest quality, significant variation in PE quality within the bar, cheapest processing method

In ram extrusion, the UHMWPE powder is extruded into a cylindrical bar stock ranging from 2 inches to 6 inches in diameter. The powder is introduced into a heated cylindrical barrel by a ram and as the ram retracts, the chamber is refilled with UHMWPE powder. Due to the heat and pressure the powder is consolidated into a continuous bar that is subsequently machined into the final product. The applied pressure is not constant but varies due to the oscillations of the ram(on/off pressure) as well as the differences between dynamic and static friction as the bar is extruded resulting in variations in the end product

EXAMINER . What are the affects of adding calcium stearate to the process?

CANDIDATE: With the ram bar extrusion process, calcium stearate was once added to the raw powder to act as a residue catalyst scavenger, lubricant and also prevent corrosion on PE machine parts but was later abandonded

The addition of calcium stearate adversely affects PE wear rates causing the following problems:

- Inconsistent PE consolidation
- Unfused PE particles(fusion defects)
- Increased PE oxidation potential
- Reduced mechanical properties of PE

2. Sheet compression molding

In sheet compression molding the powder is molded into large sheets of PE. The resin is poured onto a platen and these platens are brought together and heat is applied

After a specific heating cycle has been completed the pressure is increased to a desired set point and the material is allowed to cool under pressure. The sheets are then sectioned, cut into bars, annealed and sent for machining.

One disadvantage of sheet compression molding is that variance in bulk density of the resin can lead to inconsistency in the amount of powder in one region of the plate. This results in pressure differentials during consolidation from one area of the block to another resulting in different mechanical properties.

3. Hot isostatic pressing

This method was developed in an attempt to improve consolidation and reduce residue oxygen in the material. The process consists of cold compaction of the resin into a shape, followed by vacumn sealing in a non-gas permeable container, then hot isostatically pressing the cold compact to fuse the resin.

4. Direct compression moulding

The most advanced form of PE manufacture involves direct compression molding from PE powder. The resin is directly molded into the finished implant. There is usually no secondary machining of the bearing surface. The bearing surface is, therefore, less rough. Direct compression molding has a lower susceptibility to fatigue crack formation. The process is relatively slow and expensive but has the best wear characteristics of the various manufacturing processors described.

Machining

UHMWPE bar, sheet or compression moulded are shaped into their final form. The cutting tools used to machine PE may microscopically stretch PE chains. Stretched PE chains are more susceptible to radiation resulting in greater oxidation in this region.The classic finding of the PE stretch/oxidation effect is a white band of oxidation in the subsurface of the PE

Sterisation

Three methods of sterisation exist: Ethylene oxide gas, gas plasma treatment or gamma irradiation.

Ethylene oxide (EtO) sterisation is a lengthy process of about 40 hours and requires preconditioning/exposure/forced aeration. It involves no oxidation or crosslinking. This process may leave toxic residues.

In **gas plasma** technique, radiofrequency (RF) energy is used to generate plasma from vaporized hydrogen peroxide/paracetic acid. It is expected not to leave any toxic residue. It takes less time, about 4 hours with no lengthy aeration requirements and has no oxidation/crosslinking effects.

The most common method of sterilizing of conventional UHMWPE is **gamma sterilisation**. The gold standard dosage is about 2.5–4 Mrad from a cobalt-60 source. The high energy generated during gamma radiation breaks some of the C-bonds in the PE chain and generates free radicals. These free radicals can react in one of three ways a) Recombination (b) Oxidative Chain Scission or (c) Cross-linking.

Each of these reactions has different effects on the molecular weight and mechanical properties of the UHMWPE.

Chain scission decreases the molecular weight of the polyethylene resulting in the degradation of mechanical properties.

During shelf storage, UHMWPE components that are gamma sterilized in air permeable packaging undergo **oxidative degradation**, resulting in increase in density and crystallinity and subsequent loss in mechanical properties. Such products become more susceptible to delamination, fracture, decrease in molecular weight, creep deformation, fatigue strength and potential to undergo abrasive wear.

Based on the deleterious effects of oxygen on the longevity of the implant, manufacturers no longer perform gamma irradiation in air. Sterilisation of UHMWPE components is generally performed under vacuum or in an inert oxygen free environment in the presence of nitrogen or argon. Sterilisation in an oxygen free environment decreases **oxidative chain scission** whereas it facilitates **recombination** and **cross-linking** that decreases component wear in vivo.

Candidates may be asked about the two forms/phases of UHMWPE that exist in manufactured implants: (1) crystaline phase; (2) amorphous phase. As the molecule is cooled down below its melting point, the carbon bonds fold to form ordered sheet like crystalline lamella that are embedded within the amorphous matrix of disorganized chains.

The crystalline phase provides the mechanical strength to PE. The optimal crystalinity is between 45 and 65%. Below 45% decreases the mechanical properties of PE making it prone to cracks and failure. Increasing the crystalline phase means there is only a small amorphous phase for cross-linking, making it more prone to chain scission oxidation which results in an increase in wear particle debris.

EXAMINER: What is highly crossed linked polyethylene (HXPLE)?

CANDIDATE: The manufacture of HXPLE involves bombarding the material with high dose gamma irradiation that causes chain scission of double covalent bonds, followed by rebonding, either by oxidation or cross-linking between adjacent polymer chains. To prevent oxidation due to the formation of free radicals, the process is performed in an inert environment (a vacuum). HXPLE must be either: (1) annealed (heating below the melting point of 137°C); or (2) remelted, to release oxygen-free radicals from the material, as they will cause slow oxidation and reduce shelf life.

Figure 31.32 Clinical photograph of acetabular UHMWPE cup

Annealing eliminates some but not all free radicals. Melting eliminates all free radicals making it more effective at oxidative sabilisation but reduces the mechanical properties of PE due to lowering the crystallinity. This is associated with reduced yield strength, fracture stress and fatigue resistance.

Second-generation HXLPE have been introduced to achieve HXLPE with good wear resistance whilst maintaining its mechanical properties. Techniques introduced include the use of Vitamin E and sequential annealing.

EXAMINER: What are the advantages of using HXLPE?

CANDIDATE: Compared to standard PE HXLPE has

- Better wear resistance
- The PE particles tend to be smaller in size and produce less osteolytic reaction
- There is generally a decreased number of particles generated Disadvantages include
- Decreases tensile strength (pulling force to break)
- Decreased fatigue strength (maximum cyclic stress the material can withstand)
- Decreased fracture toughness (force to propagate a crack)
- Decreased ductility (elongation without fracture)

EXAMINER: What do we mean by PE shelf life?

CANDIDATE: Irradiated PE packed in an oxygen-free environment minimizes oxidation. However, any remaining free radicals stay in the PE indefinitely and are an oxidation risk. When PE is oxidized, chain scission occurs which results in increased PE particle debris. Depending on packaging technique, oxygen can diffuse back into the PE product, which results in on the shelf oxidation.

PE packaging: The amount of oxygen that diffuses back into the package depends on two main variables:

1. Packaging material (most important). Defined as diffusion capability
2. Time on shelf

Figure 31.33 Clinical photograph of bearing surfaces in THA (a) CoC; (b) MoM; (c) MoP

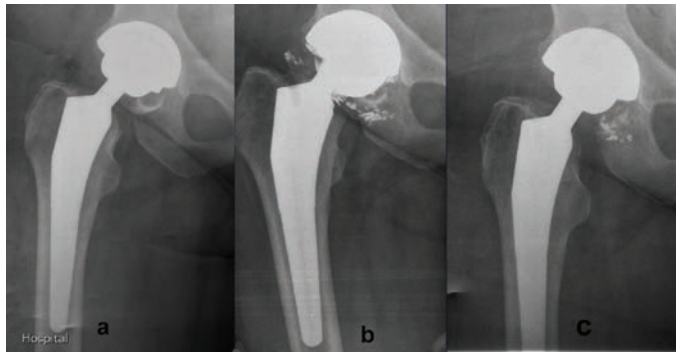


Figure 31.34 AP radiograph pelvis demonstrating: (a) gross acetabular mal-seating of ceramic liner immediately post op; (b) ceramic liner fracture at 6 weeks post op; (c) revision surgery included retention of the original acetabular component, removal of debris and careful seating of a new delta ceramic liner and head

Bearing surfaces in THA

Any or all of the three major bearing surfaces in THA (Figure 31.33) may be asked about in the basic science viva. Candidates may even be given a choice as to which bearing surface they want to discuss. Work through advantages and disadvantages of each bearing surface.

MoP: Advantages – Cheap, easy to manufacture, tried and tested with widely published evidence. Disadvantages – Be prepared to discuss aseptic loosening, PE wear and osteolysis.

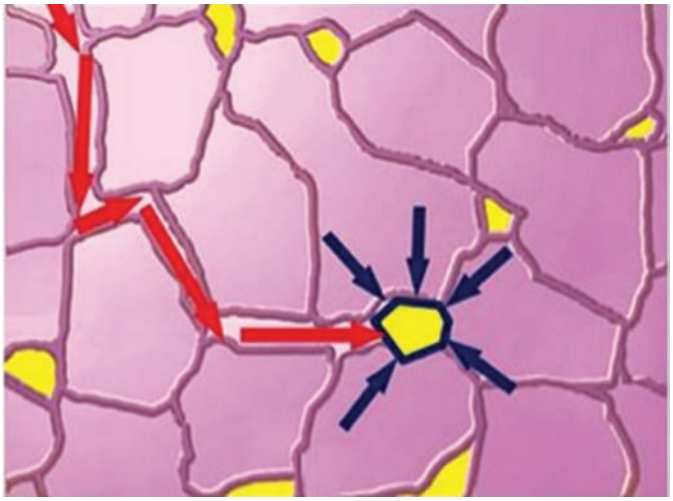


Figure 31.35 Toughening mechanisms for fourth-generation ceramic the introduction of Y-TZP in a stable alumina matrix

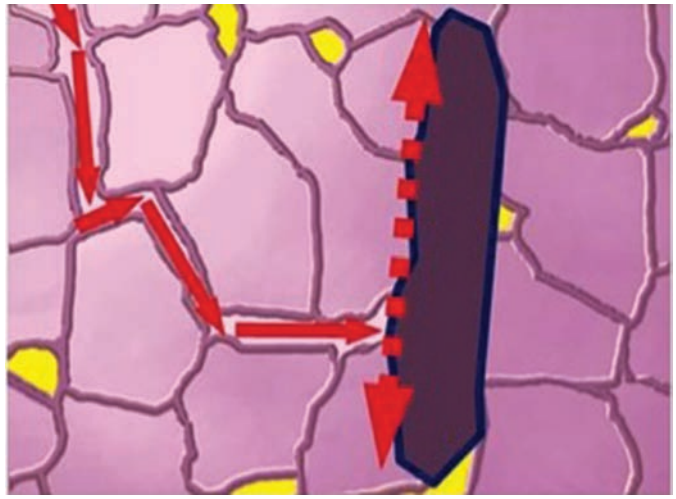


Figure 31.36 Platelit like crystals dissipate energy by deflecting and neutralizing crack energy

MoM: Advantages – Smaller particles do not generate a vigorous macrophage response. Disadvantages –ALVAL, pseudotumours, possible cancer risk, etc.

Ceramic: Advantages – High-wear resistance, biologically inert, high-compressive strength, hardness, wettability, chemically resistant and excellent scratch profiles. Disadvantages – Brittle, prone to catastrophic failure, squeaking, cost

The vagaries of the basic science viva mean that you may end up focusing on a very narrow area of the topic. A small branch of a large tree. Candidates may be shown an AP radiograph demonstrating gross mal-seating of an acetabular ceramic liner (Figure 31.34 a) or be asked about fourth-generation BIOLOX® delta ceramics and toughening mechanisms. A review of the postoperative images (Figure 31.34a) demonstrates gross mal-seating of the ceramic liner. The patient experienced some early subjective clunking of the hip and then presented suddenly with catastrophic ceramic liner fracture requiring revision (Figure 31.34 b–c).

The first toughening mechanism for fourth-generation ceramics was the introduction of nano-sized yttria-stabilised tetragonal zirconia particles (Y-TZP) in a stable alumina matrix (Figure 31.35). The spatial separation of these zirconia particles reduces the likelihood of structural transformation and prevents the initiation or propagation of cracks. The dispersion of fine particles in the matrix also improves fracture strength and toughness by producing local pressure peaks in the area of cracks and thereby counteracting their propagation. In simply terms the zirconia particles act like air bags by absorbing impacting forces.

The second reinforcement mechanism is achieved by adding a strontium oxide which forms platelet-like crystals (Figure 31.36). These platelets dissipate energy by deflecting and neutralizing cracks, thereby increasing the strength and toughness of BIOLOX®*delta.*

Examination corner

Basic science oral 1

Candidates are asked to draw the stress/strain curve for cobalt chromium and explain the diagram as they draw.

You will be asked:

- To identify features on the curve such as yield point, ultimate tensile strength and breaking strength
- The differences in the shape of the stress/strain curve between a ductile and brittle material
- What the area under the stress/strain curve represents

If the examiners want to make life difficult for you they will ask you to draw out the stress–strain curve of either a ligament or tendon. The examiners can also be very specific about definitions such as hardness and toughness. Hardness describes a materials resistance to localized plastic deformation. Toughness is the materials ability to absorb energy up to the fracture. Toughness is derived from both strength and ductility of a material.

Basic science oral 2: Stress–strain curve

Several questions were asked about various parts of the curve – Breaking point, yield point, etc.

Basic science oral 3

Draw the stress–strain curve for ceramic and silicone and explain as you go along

Basic science oral 4

Stress–strain curve – Discuss each part of the curve

Examination corner

Basic science oral 1

Exhibit – A metal hammer was shown which had broken off at the junction between the head and shaft

EXAMINER: This hammer broke while I was using it in theatre a couple of days ago and I brought it along so that we can discuss why it broke where it has. If you look carefully it is broken at the junction between its head and shaft. Can you explain why this is so?

CANDIDATE: A difficult question, which was also found tricky by most of the other candidates I discussed it with. I managed to scrape some sort of answer together in a roundabout way, albeit with much prompting by the examiner.

The answer appears reasonably straightforward in a non-exam situation but trying to work it out from first principles in the stress of the examination is an entirely different matter.

Basic science oral 2: Structural properties of titanium

EXAMINER: What is the head made of in a titanium on plastic THA implant?

COMMENT: This is a an unusual question and we think refers to the fact that titanium is no longer used as a material for femoral heads due to its poor wear characteristics. Extensive burnishing and scratching of titanium alloy femoral heads have been reported along with a 10-fold increase in wear rate.

Early failures of titanium alloys saw it fall out of favour as a material for cemented femoral stems. The adverse publicity about the controversial 3M Capital hip in 1998 reinforced a general reluctance to use cemented titanium stems. The problems associated with the Capital hip were attributed to overload of a narrow, proximomedial cement mantle (1 mm) leading to fracture of the cement and loosening, preceding the production of wear debris.

Recent data from the Norwegian Arthroplasty register suggest despite some positive reports the use of cemented titanium implants should be avoided[13].

Classic reference

Agins HJ, Alcock NW, Bansal M, et al. Metallic wear in failed titanium-alloy total hip replacements: A histological and quantitative analysis. *J Bone Joint Surg Am.* 1988;70:347–56.

Agins et al. reported a marked histiocytic, lymphoplasmacytic and foreign-body giant-cell reaction to abundant metal, polyethylene, and bone cement debris in the tissues around nine titanium-alloy femoral stems that had been revised because of aseptic loosening or infection. Focal necrosis was seen in all of the hips.

This paper raised the problem of the clinical significance of black metallic wear debris deposited in the periarticular tissues surrounding titanium alloy prostheses.

The authors undertook a quantitative and histological analysis of the tissues around failed Ti-Al-V total hip arthroplasties (THA). They demonstrated that when a titanium THA failed for whatever reason, copious metallic debris can be generated that can be locally irritating and possibly toxic to the surrounding tissues. The authors postulated that this may contribute to implant loosening.

The poor wear characteristic of titanium as an articulating surface on cemented THA resulted in extensive production of titanium debris with associated osteolysis and premature loosening of the implant.

Examination corner

Basic science oral 3

Fatigue fractures and endurance limit

Be prepared to draw out and/or explain S–N curve

The presenting history was of a elderly male who had sustained a mal-united distal radius fracture. This had been treated with an osteotomy and bone grafting but the osteotomy had gone onto a non-union. The volar locking plate had deformed and was bent. An AP and lateral radiograph of a distal radius was shown with the volar radial locking plate bent (Figure 31.37 a and b).

I was asked what I thought of the radiograph. I commented that there was little sign of bone healing and this may possibly be a non-union. I mentioned that as an implant had been used I must first exclude any obvious infection. This led onto a discussion of the factors which inluence fracture healing: (1) local biology; (2) systemic biology; (3) fracture mechanical enviroment.

Later on a laminate of the bent plate was shown (Figure 31.38). The plate would have eventually failed due to fatigue failure. This would be a mechanical failure due to repeated stresses on the plate secondary to the non-union. Fatigue cracks would occur at the locking holes. Factors that may affect the fatigue life of the plate include location, type and complexity of fracture, load acting on the plate, and types and sizes of plate used. Constructs with a short working length are less resistant to cyclic fatigue failure.

Genetics and cell biology

Application/relevance of modern genetics to orthopaedic disease and treatment

EXAMINER: A pregnant woman comes into your clinic. The sex of the unborn child is unknown. She has a son who was diagnosed with Duchenne muscular dystrophy at the age of 3 years and her younger brother died of the same condition. What is the chance of her unborn child also having the condition?

CANDIDATE: In this condition, which is a lethal X-linked recessive disorder in which the affected males generally do not reproduce, if the woman (individual II: 2 in Figure 31.39) had a brother (II: 3) and has a son (III: 1) who have both been affected by the condition, it is very likely that the woman herself is a carrier. It is not known whether the pregnant woman's unborn child (III: 2) is male or female. It is also unknown, whether or not she will have passed on her X chromosome that contains the gene mutation (rather than the normal X chromosome). The chance of the unborn child having the condition can, therefore, be calculated as the woman's chance of being a carrier (i.e. 1) multiplied by the chance of having a male child (i.e. ½) multiplied by the chance of her passing on the mutant (rather than the normal) X chromosome (which is ½). In summary, the chance is $1 \times ½ \times ½ = ¼$.

EXAMINER: If her brother died of the condition (as in the first scenario), but it was her sister who has the son with the condition, what would be the chance of this lady's child being a carrier?

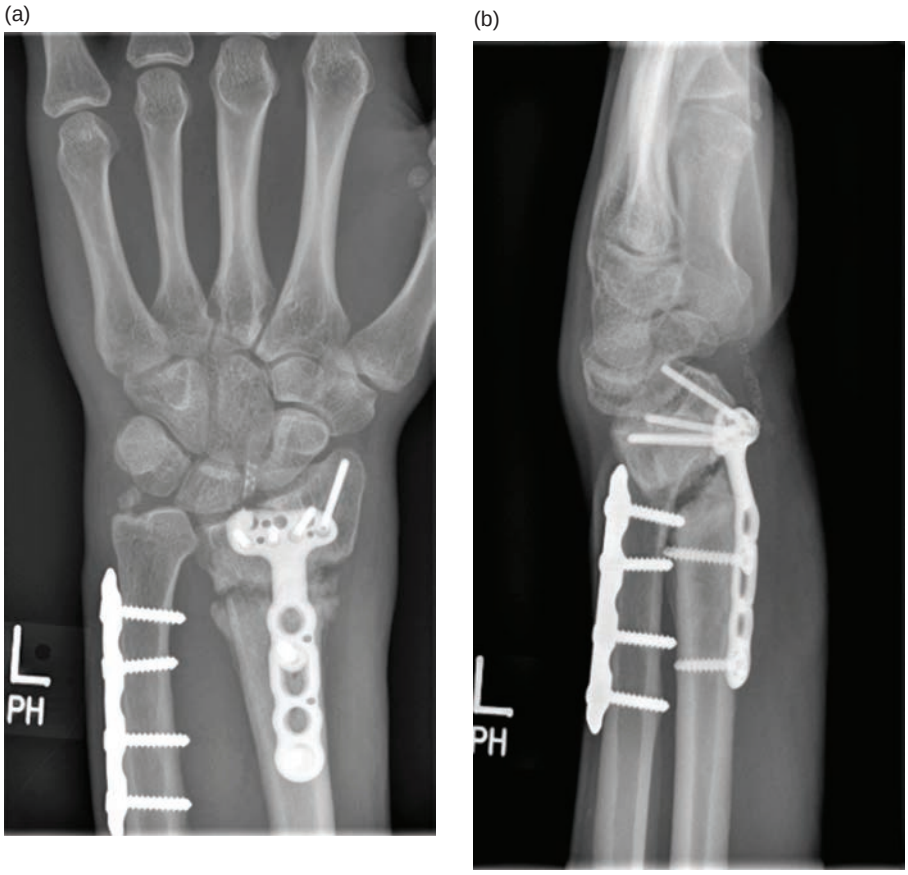


Figure 31.37 (a) AP and (b) lateral radiograph bent volar radial locking plate with non-union

Figure 31.38 Deformed Volar locking plate

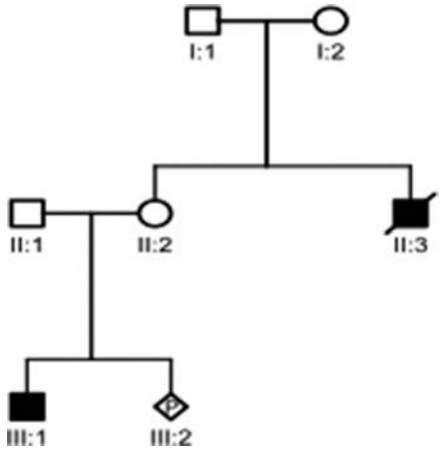


Figure 31.39 Lethal X-linked recessive disorder

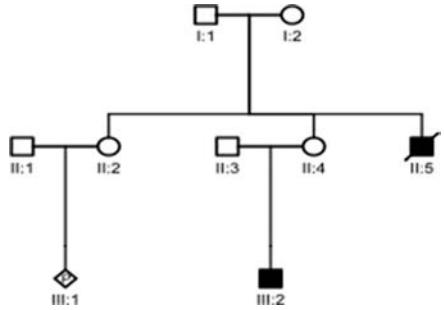


Figure 31.40 Lethal X-linked recessive disorder

CANDIDATE: It is useful to realize that the pregnant woman's mother (individual I: 2 in Figure 31.40), who has a carrier daughter (II: 4) and had an affected son (II: 5), would, almost certainly, also be a carrier herself. Thus, the pregnant woman (II: 2) would have a ½ chance of having inherited the mutant X chromosome from her mother. In other words, the pregnant woman will have a ½ chance of being a carrier. This means, in turn that the pregnant woman's chance of having a daughter who is a carrier will be ½ (i.e. the woman's chance of being a carrier) × ½ (i.e. the chance of having a daughter rather than a son) × ½ (i.e. the chance of passing on the mutant chromosome, if the woman is a carrier) = 1/8.

NB. There are multiple variations on this question. It may be useful to draw Punnett squares to work out the answer. In

practice, it would be important to involve the local Clinical Genetics department in providing advice in this situation[ee].

Examination corner

Basic science oral 2

EXAMINER: What is the mode of inheritance of achondroplasia? Why do most achondroplastic patients not have parents with the same condition?

CANDIDATE: This question can lead to a discussion of new mutations and the subjects of variable expressivity/expression and complete/incomplete penetrance. The mode of inheritance of achondroplasia is autosomal dominant with generally complete penetrance (i.e. everyone who inherits a mutation will develop the clinical signs or phenotype). The new mutation rate is 80% (i.e. 80% of cases arise as a result of a new mutation occurring in just the egg or sperm cell of the parent). This is why most patients with the condition will not have a similarly affected parent.

Examination corner

Basic science oral 3

- What is the mechanism of inheritance of Duchenne muscular dystrophy
- Describe the gene that encodes the dystrophin protein
- What do you know about dystrophin
- Do you know of other types of muscular dystrophy

Answer: The mechanism (pattern) of inheritance is X-linked recessive.

It is an unusual gene in that it is very large (in fact the largest in humans), with 2.2–2.4 million nucleotides. Only about 11 000 of these code for amino acids, however. This is because the RNA undergoes changes (processing) after it has been transcribed. The gene contains a large number of exons (79). Like many RNAs, dystrophin RNA undergoes extensive processing after transcription, involving the removal (by splicing) of large intervening sections (introns) that (unlike exons) are not required for translation into protein. Unusually, in Duchenne muscular dystrophy, the most common type of mutation is an intragenic deletion that removes several exons and severely disrupts the resulting protein. This accounts for 60–65% of cases.

The protein encoded by this gene, dystrophin, is large, shaped like a rod and plays a role in the muscle fibre membrane (the sarcolemma) where it links the F-actin of the inner structural cytoskeleton to extracellular matrix proteins. This protein is, therefore, an integral part of the muscle cells and its absence in Duchenne-affected individuals is the underlying cause of the condition.

A similar but milder condition, Becker muscular dystrophy, is caused by mutations in the same gene and is, therefore, also inherited in an X-linked recessive fashion. Its phenotype is milder as a result of less pathogenic mutations that generally do not result in the absence of dystrophin, although the protein produced is often shorter and less functional than the normal form of the protein.

There are also several other forms of muscular dystrophy that are associated with other genes, such as Emery–Dreifuss (most commonly X-linked and less commonly autosomal dominant), facioscapulohumeral (autosomal dominant) and oculopharyngeal (autosomal dominant) muscular dystrophies.

Diagnostics

Musculoskeletal imaging: X-ray, contrast studies, CT, MR, ultrasound, radioisotope studies

Examination corner

Basic science oral 1

- What is a bone scan?
- How does it work?
- What are the indications for its use?
- What are the various phases of a bone scan?

Basic science oral 2

EXAMINER: How do bone scans work?

CANDIDATE: A bone scan involves the intravenous injection of a bone-seeking radionuclide, which localizes at sites of increased bone blood flow with increasing osteoblastic activity. The tracer uptake has three phases: Vascular phase, static blood pool and bone phase

EXAMINER: How long after the injection are the bone images available?

CANDIDATE: Four hours.

EXAMINER: What is the half-life of technetium-99 phosphate?

CANDIDATE: Six hours

EXAMINER: Why use an isotope with a half-life of 6 hours?

CANDIDATE: You want to get rid of the radioactive material from the body as soon as possible.

EXAMINER: Yes you don't want to glow in the dark any longer than possible.

Basic science oral 3

Bone scan

- Principles of technetium and indium scanning

Basic science oral 4

Bone scan

[ee] For more explanations and information regarding relatively common medical genetics problems, the reader may wish to consult the latest edition of *Essential Medical Genetics* by Tobias, Connor and Ferguson-Smith, and the self-assessment questions provided at www.essentialmedgen.com.

- Half-life, advice re avoiding pregnant women and urinating!

Basic science oral 5
- Principles of the isotope bone scan
- Value of the triple phase component
- Other types of isotope scans
- Indium-labelled white cells
- SPECT scanning

Kinematics and gait analysis

Examination corner

Basic science oral
- What are Gage's five prerequisites for gait?
- What do you understand by gait analysis – How does it work (infra-red cameras, reflective markers)?
- What are the components of the gait cycle?

Clinical environment

Equipment design and use

Tourniquets

Examination corner

Basic science oral 1

General discussion about tourniquets
- Responsibility for maintenance of tourniquets
- Tourniquet pressures used in the upper and lower limb
- Complications of tourniquet application
- Safe tourniquet time

CANDIDATE: A bit of a vague, awkward and waffly start to the basic science oral. The examiner seemed to go on for what seemed like forever about who was responsible for tourniquet maintenance without asking me any specific question. The examiner asked me if I knew any papers published on the subject. I remembered a paper published from Liverpool a few years previously that had shown they were poorly maintained and looked after[14]. The examiner agreed and continued to waffle on for another minute or so before asking me about tourniquet pressures.

EXAMINER: It was just a general discussion about tourniquets. I wasn't after anything too complicated.

The Rhys–Davies exsanguinators in our local hospitals were poorly maintained (inflation pressure, storage conditions, replacement age) when compared to the manufacturers' recommendations.

Basic science oral 2

Use of tourniquet in orthopaedic surgery
- Complications

Evidence management

Data analysis

Data analysis and statistics – Principles and applications

Examination corner

Basic science oral 1

Scatter plot: Regression analysis, linear vs non-linear relationship, skewing of the curve

Basic science oral 2
- Survivorship analysis in joint replacement
- Kaplan–Meier curve: Draw one and explain the *x* and *y* axes
- Confidence intervals and their importance

EXAMINER: You can see each death as a downward step in the curve. These steps get bigger as you go along the graph. Does that mean the data are more accurate?

CANDIDATE: No. There are fewer patients in the study and, therefore, when one dies the step is bigger.

EXAMINER: You mean that the data are less accurate?

CANDIDATE: Yes.

EXAMINER: What happens to patients who are followed up for say five years but then move to a different part of the country and are not followed up? Should we include these data?

CANDIDATE: You are talking about censorship. Before the censored time, you know they were alive and following the experimental protocol, so these subjects contribute useful information. After they are censored, you can't use any information on the subjects.

Basic science oral 3
- Normal distribution curve, standard deviation
- Null hypothesis, *P*-values, type I and II errors, power of a study

Basic science oral 4
- Sensitivity
- Specificity
- Drawing of the squared box with an example to simplify the answer (practise this beforehand)
- Positive predictive value and negative predictive value

Basic science oral 5

EXAMINER: Your consultant asks you to review the results of their first 1000 THR. How would you go about this?

CANDIDATE: I would perform a survivorship analysis curve.

EXAMINER: What are the types of survival analysis curves that can be performed?

EXAMINER: What are the advantages of Kaplan–Meier over the actuarial method?

EXAMINER: How do you account for loss to follow up and then how do you calculate it within your survivorship curve?

EXAMINER: What about mortality?

EXAMINER: What is a confidence interval?

Basic science oral 6

EXAMINER: What will you do if you get five infections in close succession in your joint replacements?

CANDIDATE: I burbled and blabbered on about audit, etc.

EXAMINER: How do you know that this is not a random occurrence and is probably due to system failure?

CANDIDATE: I wasn't sure. The examiner prompted me – What about the organisms. I didn't get it. He answered for me. What if you get all five hips infected by the same organism. Only then did I see his reasoning.

Basic science oral 7

- How would you plan to start using a different type of knee replacement?
- Survival analysis – Details, methods, Kaplan–Meier curve
- Draw a survival analysis curve and describe it
- Confidence intervals

Basic science oral 8

Ten minutes of the basic science oral was spent on statistics. Topics covered included:

- Mean
- Median
- Mode
- Theory of central tendency
- Variance
- Standard deviation
- Correlation coefficient
- Regression analysis
- ANOVA

Basic science oral 9

- I was asked to draw the normal distribution curve and comment on it
- What is standard deviation?
- Setting up a study: Discussion on null hypothesis, *P*-values, type I and II errors, power, etc

Basic science oral 10

- What information do you need to know in order to perform a statistical power calculation?

Basic science oral 11

- Define what a *P*-value of 0.005 means

Basic science oral 12

EXAMINER: What types of studies exist?

CANDIDATE: I mentioned several but the examiner didn't seem impressed.

EXAMINER: Your boss wants you to review 10 years worth of results of the THA that they use.

How do you go about it?

What type of study would it be?

CANDIDATE: I hesitated with a fairly long pause but recovered. However, I barely got started before the examiner started to lose interest in my answer and switched to another topic.

EXAMINER: You don't seem to know a great deal on this topic lets move on to another one.

CANDIDATE: I hadn't anticipated this type of question and just was not sharp enough with the answer. You can't predict every question you will get asked.

(Fail)

CANDIDATE: I spent a lot of time studying statistics and I was not asked particularly much about it in the examination. I wish I had spent less time on it because it was wasted effort.

Much as we have been trying to steer you into topics that tend to be asked in the FRCS Orth exam, one cannot entirely predict what the examiners will ask any particular candidate at any particular time.

Resign yourself to the fact that some 'dead certain' topics learnt will not be asked. Accept it, don't dwell on it and move on to the next topic.

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Chapter 32

Surgical exposures oral core topics

Anish Kadakia and Jonathan Loughead

Introduction

This can be a tedious and dry area of orthopaedics to learn but, for various reasons, examiners are encouraged to ask candidates more questions on surgical approaches than ever before.

In real life it is a very important practical part of a practising orthopaedic surgeon's workload. The irony is that one would usually read up an unfamiliar or forgotten approach before undertaking the required surgery. It has been suggested that at least 10 minutes of the basic surgical oral should be spent discussing surgical approaches and anatomy with candidates.

Most candidates tend to forget this topic very quickly and may spend a fair amount of time re-learning it in the week or so before the FRCS (Tr & Orth) examination. We have attempted to summarize the important surgical approaches in a structured and concise but also comprehensive manner. This allows a candidate to quickly brush up his/her knowledge in the busy week before the exam without having to go back and spend a lot of time re-reading the bigger books.

The vast majority of surgical approach questions are asked as part of another question. Usually a radiograph will be shown, which leads on to a management question and then the preferred surgical approach.

'In my trauma oral I was shown a talar neck fracture. I described the radiograph, classified the injury as a Hawkins[a] *type 3 and was then asked what surgical approach I would use to fix this fracture.'*

'I described a medial malleolar osteotomy approach, which I had read about in a textbook but had never seen as a trainee. My answer wasn't very convincing and I am sure the examiners wanted me to be a bit more definite about the approach[b]*.'*

Sometimes candidates are shown a clinical picture/diagram, which then leads on to the approach, e.g. a cross-sectional diagram of the calf leading on to discussion of compartment syndrome and how you decompress a leg.

With any surgical approach it is vital to stick to a format to ensure that no important steps are missed during your answer. It is important to deal with general surgical principles and below is a suggested plan of action for any surgical approach, which you may be asked about:

- Preoperative issues
- Indications
- Anaesthetic
- Positioning
- Anatomical landmarks
- Incision
- Superficial surgical dissection
- Deep surgical dissection
- Internervous plane
- Structures at risk
- Extensile exposure (how to enlarge the approach)
- Wound closure

Delivering your answer confidently is important. Try not to bore the examiners with a pre-learned robotic answer.

Looking through various DVDs that outline surgical approaches hoping to learn some sort of model answer for the FRCS (Tr & Orth) exam may not be the most productive use of your time[c]. These DVDs are probably more useful as a junior consultant when learning an unfamiliar surgical approach with a particular operation in mind. U tube also has a number of surgical approaches uploaded on video format to view.

There are a number of surgical approaches courses that candidates can attend. These courses usually involve cadaveric prosection, lectures and small group viva sessions. Our experience is that most candidates sign up for these courses at the beginning of their registrar training to learn and become more

[a] I felt I was blurting out the Hawkins' classification system a bit clumsily at the time, but in hindsight it was being proactive. It possibly saved my neck and the day because my description of a medial malleolar osteotomy was ropy. There is a danger of getting too worked up with exam protocols: what to say, what not to say, how indirectly to let the examiners know that you know a classification system, etc. In any oral, I would always attempt to mention a classification system if I knew it well and it was appropriate.

[b] Most candidates never sound very convincing describing a surgical approach if they haven't seen it performed. Very occasionally we come across candidates who can describe an operative procedure so well and with so much confidence you wonder how many numbers as lead surgeon are in their e-logbook. Probably 10, perhaps 20, but incredibly they haven't even assisted for the procedure and have only read it from a book!

[c] Not to discount anything as everyone has different learning methods so this technique may just work for you.

familiar with the anatomy of the common surgical approaches for on the job knowledge rather than exam revision purposes.

That said, a one-day anatomy and surgical approaches course in the three months before the part 2 exam, although an expensive method of revision, may be a reasonable option if one can avoid a long distance to travel and any overnight hotel stay.

Time is of essence in the exam and although the surgical approaches are described in detail here, during the viva one has to be very concise and succinct in the description so as to allow time for discussion.

For example in this description of the deltopectoral approach: The patient is fully prepared, marked and consented for surgery. Case notes and relevant imaging are made available in theatre. The WHO checklist done.

The deltopectoral approach utilizes the internervous plane between the axillary nerve (supplying the deltoid) and the pectoral nerves (supplying the pectoralis major).

The structures at risk in this approach are the axillary nerve, cephalic vein and the musculocutaneous nerve.

I put the patient in beach chair position with the armpit of the operated side in line with the table edge and a sandbag under the medial border of the scapula to protract the shoulder forward and give better exposure of the glenoid. The forearm is supported on an armrest. Padded headrest with the head elevated 30–45°.
I screen the shoulder first before prepping and draping so that adjustments in position can be made if the image is poor.
I mark out the landmarks namely the coracoid process and the delto-pectoral groove.

I make a straight skin incision approximately 10–15 cm long starting from just above the tip of the coracoid process along the line of the deltopectoral groove, passing just lateral to the apex of the axilla. I deepen the incision through subcutaneous fat to the fascia overlying the deltoid and pectoralis major muscles. The fat is swept back with a swab to help identify the deltopectoral groove, marked by the large cephalic vein. The deltoid branches of the thoracoacromial artery, which lie in the deltopectoral groove may need cauterising. I retract the cephalic vein laterally and open the deltopectoral interval by blunt dissection and insert a self-retaining retractor. If the cephalic vein is injured during dissection, I ligate it proximally and distally. The conjoined tendon is identified and its lateral edge is freed by incising the clavipectoral fascia and the tendon is retracted medially being aware that the musculocutaneous nerve enters its medial edge approximately 4–6 cm distal from the coracoid process and mindful of the fact that retractors placed under the conjoint tendon can cause neuropraxia of the musculocutaneous nerve and, hence, vigorous retraction must be avoided. Palpating for the long head of biceps tendon will lead proximally to the rotator interval between subscapularis and supraspinatus. The lower border of subscapularis is identified by a series of small veins running along this border (anterior humeral circumflex vessels).

I cauterize the vessels as haemostasis of the medially retracted transected vessels can be difficult. The axillary nerve is usually palpated as it courses laterally over the belly of the subscapularis muscle before passing in close proximity to the inferior capsule. Two stay sutures are placed in the tendon of subscapularis. With the arm held in external rotation, as this will draw the lesser tuberosity away from the axillary nerve, the tendon is divided 2 cm from its lateral attachment onto the lesser tuberosity of the humerus. The capsule of the joint is now exposed and a plane is developed between the capsule and subscapularis, which is best developed inferiorly. Stay sutures are placed in the capsule and a vertical tenotomy is made in the capsule 0.5 cm medial to the sectioned stump of subscapularis (longitudinal capsule incision). Sometimes, a horizontal subscapularis muscle-splitting approach is used. Often, the subscapularis and the capsule are incised together vertically to enter the shoulder joint.

Go through the description of all the approaches and prepare a similar précis for each one of them.

Shoulder

- Anterior approach (deltopectoral)
- Lateral approach (deltoid-split)
- Posterior approach

Anterior approach to the shoulder

Indications

- Shoulder arthroplasty
- Open shoulder stabilisation
- Fractures of the proximal humerus
- Drainage of joint infections

Position

Supine position. The patient is pulled towards the edge of the table so that the armpit of the operated side is in line with the table edge. A sandbag is placed under the medial border of the scapula to protract the shoulder forward and give better exposure of the glenoid. The forearm is supported on an armrest. Padded headrest with the head elevated 30–45° or dental chair position. A neurosurgical type headrest is preferred as this allows access to the shoulder both anteriorly and posteriorly and allows better positioning if image intensifier is used. If the image intensifier is to be used, screen the shoulder first before prepping and draping so that adjustments in position can be made if the image is poor. The line of the skin incision may be infiltrated with local anaesthetic and adrenaline to reduce bleeding. Mark out the local anatomy of the shoulder with a marker pen. Draping is done so that the arm is freely moveable during the operation

Landmarks

- Coracoid process
- Deltopectoral groove

Incision

A straight incision approximately 10–15 cm long is made through the skin just above the tip of the coracoid process following the line of the deltopectoral groove, passing just lateral to the apex of the axilla. Do not go beyond the lateral edge of biceps

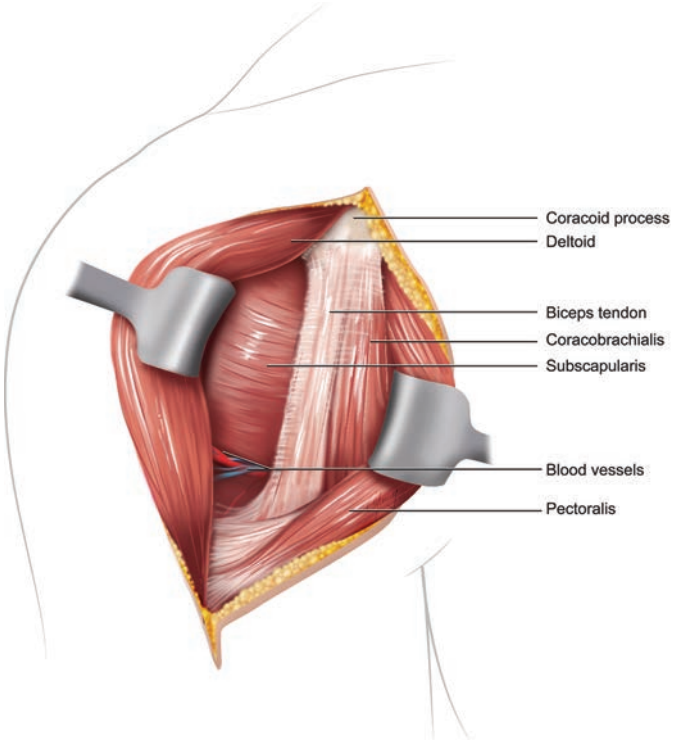

Figure 32.1 Deltopectoral approach. Deltoid is retracted laterally and the pectoralis major medially to expose the conjoint tendon of the short head of biceps and coracobrachialis muscle. Observe the series of small vessels at the inferior end of the subscapularis muscle

Internervous plane

The internervous plane lies between the deltoid muscle (axillary nerve) and the pectoralis major muscle (medial and lateral pectoral nerves).

Superficial dissection (Figure 32.1)

The incision is deepened through subcutaneous fat to the fascia overlying the deltoid and pectoralis major muscles. The fat is swept back with a swab to help identify the deltopectoral groove, marked by the large cephalic vein. Failure to find this internervous plane can lead to a difficult dissection through the deltoid and can lead to denervation of the anterior portion of the deltoid.

Cauterize the deltoid branches of the thoracoacromial artery, which lie in the deltopectoral groove. Retract the cephalic vein laterally and open the deltopectoral interval by blunt dissection and insert a self-retaining retractor. Alternatively, sometimes taking the cephalic vein medially allows an easier dissection (sometimes the vein just wants to go medially)[d]. Try to preserve the vein if possible as this may reduce postoperative swelling. If the cephalic vein is injured during dissection, ligate it proximally and distally.

[d] If you can throw in this type of practical detail it demonstrates a more thorough working knowledge of the surgical approach. Candidates will be scoring 7s instead of 6s.

Laterally reflect the anterior part of the deltoid muscle to expose structures about the coracoid process and the anterior part of the joint capsule.

The coracoid process with attached muscles (coracobrachialis, pectoralis minor and short head of biceps) is now exposed. Perform either an osteotomy of the coracoid process or, more simply, leave the coracoid process intact and retract the attached muscles medially. Retractors placed under the conjoint tendon can cause neuropraxia of the musculocutaneous nerve; vigorous retraction must be avoided.

The clavipectoral fascia is incised along the lateral border of the conjoined tendon (lateral side – Safe, medial side – Suicide!). The musculocutaneous nerve enters its medial edge approximately 4–6 cm distal from the coracoid process.

The subscapularis tendon is identified; it overlies the front of the humeral head. The lower border of subscapularis is identified by a series of small veins running along this border (anterior humeral circumflex vessels). The vessels must be carefully avoided or cauterized; if transected vessels are allowed to retract medially, haemostasis can be difficult.

Palpate for the long head of biceps tendon which will lead proximally to the rotator interval between subscapularis and supraspinatus. Identify the axillary nerve, which courses laterally over the belly of the subscapularis muscle, before it dives into the quadrilateral space around the inferior margin of the subscapularis at the muscle-tendon junction. Exposure of the inferior capsule places this nerve at risk, especially if there is scar tissue in this region. It is usually not necessary to expose the nerve. Simple palpation is usually enough; however, if dissection is difficult and the nerve cannot be palpated, exposure of the nerve may be indicated.

Deep dissection (Figure 32.2)

Two stay sutures are placed in the tendon of subscapularis. The arm is then held in external rotation as this will draw the lesser tuberosity away from the axillary nerve, and the tendon is divided 2 cm from its lateral attachment onto the lesser tuberosity of the humerus. The capsule is now exposed and a plane developed between the capsule and subscapularis, which is best developed inferiorly. Stay sutures are placed in the capsule and a vertical tenotomy is made in the capsule 0.5 cm medial to the sectioned stump of subscapularis (longitudinal capsule incision). One can incise the subscapularis tendon vertically or perform a horizontal subscapularis muscle-splitting approach (Figure 32.2). For maximum exposure, a vertical tenotomy is usually preferred. A subscapularis muscle-splitting approach is sometimes performed to decrease the risk of muscle shortening and failure of healing of the tendon repair. To gain better exposure, a pectoralis major tenotomy can be performed at the superior third of its insertion and the deltoid can be partially released from the clavicle. Specialised shoulder retractors greatly improve surgical exposure (spiked retractors beneath subscapularis, glenoid retractors etc).

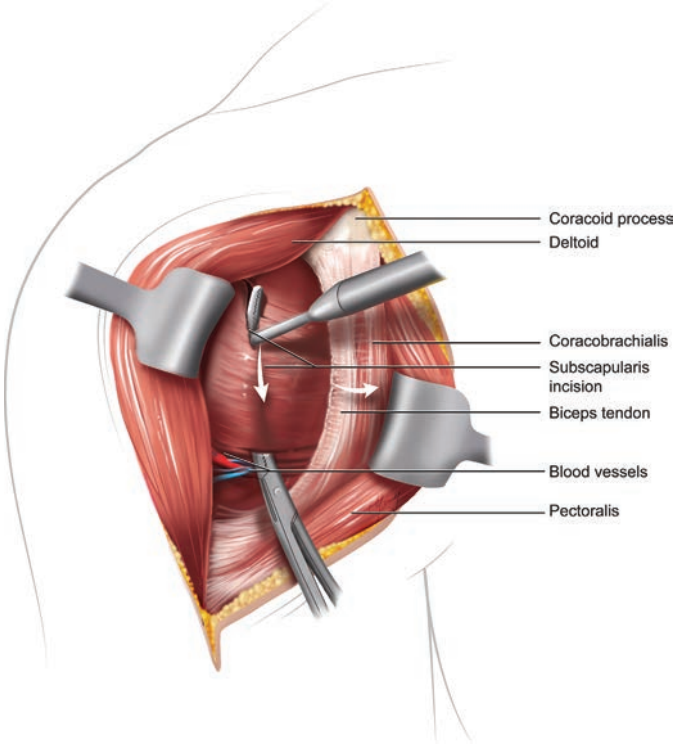

Figure 32.2 Deep dissection (deltopectoral approach). A blunt instrument is passed between capsule and subscapularis muscle. The small vessels on the inferior border of subscapularis often require cautery. Coracoid process osteotomy is often omitted by shoulder surgeons unless additional shoulder exposure is required

Structures at risk

- Musculocutaneous nerve
- Cephalic vein should be preserved if possible

Examination corner

Trauma oral 1: Radiograph of displaced 4-part proximal humeral fracture in healthy 53-year-old woman

- Discuss management options
- Preferred surgical approach for fixation and describe the approach (deltopectoral)

Trauma oral 2: Posterior dislocation of shoulder in 19-year-old man post trauma

- Identify the abnormality (lightbulb sign)
- Options for management
- Candidates preferred approach if closed reduction fails under GA late in the middle of the night

You need to be careful with the answer here. If a candidate answers that he would perform a posterior approach, the next obvious question would be how many he/she has done this before. This candidate said that he would do an anterior deltopectoral approach to reduce the shoulder and went on to describe the approach. He passed the exam!

Lateral approach to the shoulder (deltoid-split)

The lateral (deltoid-splitting) approach provides excellent access to the tuberosities, for example, for rotator cuff repairs.

Indications

- Repair rotator cuff
- Open reduction with internal fixation (ORIF) proximal humerus fractures
- Shoulder replacement

Position

Supine, sandbag under patient's shoulder, affected arm at the edge of the table. Alternatively, beach chair position, sandbag under medial border scapula, head and hand rest.

Landmarks

- Acromion

Incision

Mark out the outline of the acromion, clavicle and coracoid process beforehand with marker pen. Infiltrate adrenaline 1 : 200 000 along the incision site. A 5-cm longitudinal incision is made from the anterolateral tip of the acromion and is carried down the lateral aspect of the arm.

Internervous plane

There is no true internervous plane; the lateral approach involves splitting the deltoid muscle.

Superficial surgical dissection

Split the deltoid muscle in line with its fibres downwards for 5 cm from the acromion. Define the raphe between the anterior and middle thirds of the deltoid. Insert a stay suture at the inferior apex of the split. Expose the subdeltoid portion of the subacromial bursa by retracting the deltoid muscle anteriorly and posteriorly.

Deep surgical dissection

The lateral aspect of the upper humerus and its attached rotator cuff lie directly under the deltoid muscle and the subacromial bursa. Split the subacromial bursa and incise longitudinally to provide access to the upper lateral portion of head of humerus. A portion of the anterior deltoid can be reflected off the anterior edge of the acromion but must be carefully repaired at the end of the procedure. Rotating and abducting the arm brings different parts of the rotator cuff into view in the floor of the wound.

The incision can be extended superiorly and medially across the acromion and parallel to the upper margin of the spine of the scapula. The trapezius muscle is incised parallel to the spine of the scapula. Distally the axillary nerve has split at this level and can be identified on the undersurface of the deltoid and protected. Having done this, a second window onto the more distal humeral shaft can be created and is enhanced by partial release of the deltoid from its insertion.

Structures at risk

The **axillary nerve** enters the deltoid muscle posterior from its deep surface 5–7 cm below the tip of the acromion and then spreads anteriorly. Damaging the nerve will paralyse the anterior part of the deltoid. The deltoid should not be split in abduction as the nerve is much closer to the acromion and at increased risk of damage. Distal extension of the approach is only possible by performing a second, separate deltoid split distal to the axillary nerve.

Posterior approach to the shoulder

Indications

- Routine approach to recurrent posterior instability of the shoulder
- Fixation of scapula and glenoid fractures
- Tumour surgery

Position

Lateral decubitus position with the operated shoulder uppermost, or prone.

Landmarks

- Acromion
- Spine scapula

Incision

A linear incision is made over the entire length of the scapula spine, extending to the posterior corner of the acromion.

Alternatively, a vertical incision may be used, which is more cosmetic but provides poorer exposure of the joint. The incision is centred 2 cm inferomedially to the posterior corner of the acromion.

Internervous plane

This approach uses the internervous plane between infraspinatus (suprascapular nerve) and teres minor (axillary).

Superficial dissection

Skin and subcutaneous flaps are raised widely. Detach the origin of deltoid off the scapular spine. The plane between the deltoid and infraspinatus muscles may be difficult to define. The plane is easier at the lateral end of the incision (Figure 32.3).

Deep dissection

Deep dissection involves identifying the interval between infraspinatus and teres minor. This is an important plane that is difficult to define; it is best developed by blunt finger dissection. The fibres of infraspinatus muscle are multi-ennate, whereas the fibres of teres minor are unipennate. Retract the infraspinatus superiorly and the teres minor inferiorly.

The posterior aspect of the shoulder joint capsule is now exposed and the joint entered by incising the joint capsule close to the glenoid. If a vertical incision is used, the tendon of infraspinatus needs to be divided 1 cm medial to its insertion in to the middle area on the greater tuberosity. During closure, the posterior third of deltoid is reattached to the spine of the scapula with absorbable sutures passed through drill holes in the scapular spine.

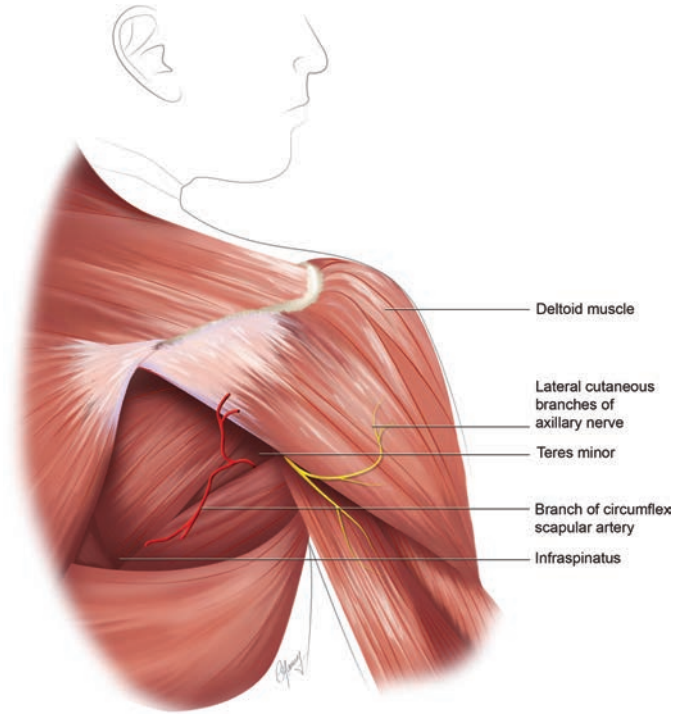

Figure 32.3 Superficial dissection of the posterior aspect of the shoulder. The plane between deltoid and infraspinatous is easiest to identify at the lateral end of the incision

To enhance access, the infraspinatus muscle can be detached 1 cm from its insertion into the greater tuberosity. Take care not to injure the suprascapular nerve, which lies on the undersurface of the muscle below the spine of the scapula. The incision cannot be extended usefully.

Structures at risk

The **axillary nerve** runs though the quadrangular space beneath the teres minor. Dissection carried out inferior to teres minor can damage the axillary nerve, so it is critical to identify the muscular interval between infraspinatus and teres minor muscles correctly and stay within that plane.

The **suprascapular nerve** passes around the base of the spine of the scapula as it runs from the supraspinous fossa to the infraspinous fossa. The infraspinatus muscle must not be retracted too far medially or neuropraxia may result from stretching the nerve.

The **posterior circumflex humeral artery** runs with the axillary nerve in the quadrangular space and can be damaged, leading to troublesome haemorrhage.

The **radial nerve** leaves the axilla by passing through the triangular interval bounded above by the teres major muscle. Triangular interval is formed by inferior border of the teres major, the long of the triceps medially and the humerus laterally.

The **circumflex scapular artery** runs in the triangular space and forms part of the extremely rich blood supply to the scapula. Dissection between teres major and minor may damage this vessel, causing haemorrhage that is difficult to control. Triangular space is formed by the inferior border of teres minor/subscapularis, superior border of the teres major and laterally the long head of triceps.

Humerus

- Anterior
- Posterior approach
- Anterolateral approach to distal humerus

Anterior approach to the humerus

This approach gives access to the proximal and middle thirds of the humeral shaft.

Indications

- ORIF fracture of humerus
- Biopsy and resection of tumours
- Management of osteomyelitis

Position

The patient is positioned supine on the operating table, with the arm on an arm board, abducted 60°.

Landmarks

- Coracoid process
- Lateral border of biceps muscle
- Deltoid tuberosity

Skin incision

A longitudinal incision from the tip of the coracoid process extending laterally and distally along the deltopectoral groove to the deltoid tuberosity on the lateral aspect of the humerus, halfway down its shaft (Figure 32.4). From there the incision continues along the lateral border of the biceps muscle and stops 5 cm above the flexion crease of the elbow.

Internervous plane

The anterior approach makes use of two different internervous planes.

Proximally the plane lies between the deltoid muscle (axillary nerve) and the pectoralis major muscle (medial and lateral pectoral nerves). Distally the plane lies between the medial fibres of the brachialis muscle (musculocutaneous nerve) and the lateral fibres of the brachialis muscle (radial nerve).

Surgical dissection

Proximal humeral shaft

The superficial and deep fasciae are divided in line with the skin incision. Identify the deltopectoral groove and separate the deltoid and pectoralis major muscles, and develop the muscular interval distally down to the insertion of the deltoid into the deltoid tuberosity and the insertion of pectoralis major into the lateral lip of the bicipital groove.

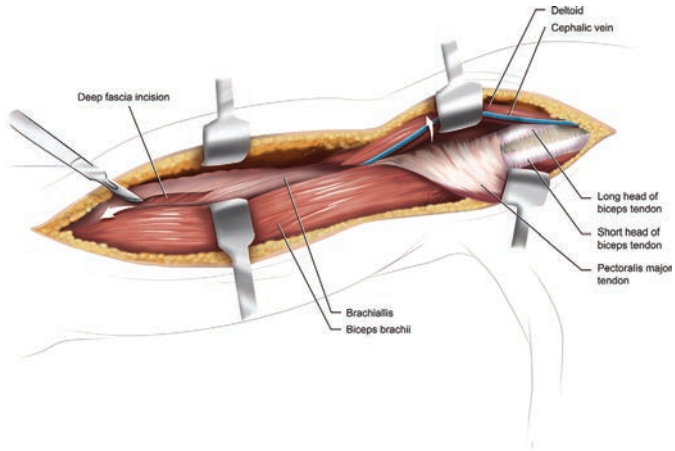

Figure 32.4 Anterior approach to the humeral shaft. Proximally the internervous plane lies between deltoid and pectoralis muscle. Distally the internervous plane lies between brachialis and biceps brachii. The cephalic vein can be retracted laterally in the deltopectoral groove and the muscular interval developed down to the deltoid insertion. Incise the deep fascia distally to identify the distal interval between the brachialis and biceps brachii

Proximally detach the insertion of pectoralis major from the lateral bicipital groove and continue the dissection subperiosteally to expose the upper humerus. The anterior humeral artery crosses the field of dissection in a lateral direction and must be ligated. With proximal shaft fractures partial deltoid release to accommodate plate placement is often required.Keep any stripping to only the minimum necessary and definitely avoid complete detachment as this may interfere with deltoid function.

Proximally the incision can be extended and modified into an anterior approach to the shoulder.

Distal humeral shaft

Distally incise the deep fascia of the arm in line with the skin incision. Identify the musculocutaneous nerve distally (Figure 32.5). Identify the muscular interval between the biceps and the brachialis, and retract the biceps medially (beneath it is the anterior aspect of brachialis, which cloaks the humeral shaft). Flex the elbow to take the tension off the brachialis.

Split the fibres of brachialis longitudinally along its midline to expose the periosteum of the anterior surface of the humeral shaft.

Distally the incision cannot be usefully extended.

Structures at risk

- The **radial nerve** is vulnerable at two points as it courses along the humerus
  1. In the spiral groove at the back of the middle third of the humerus, dissect muscle from bone, starting in a subperiosteal plane without straying onto the posterior surface of the bone

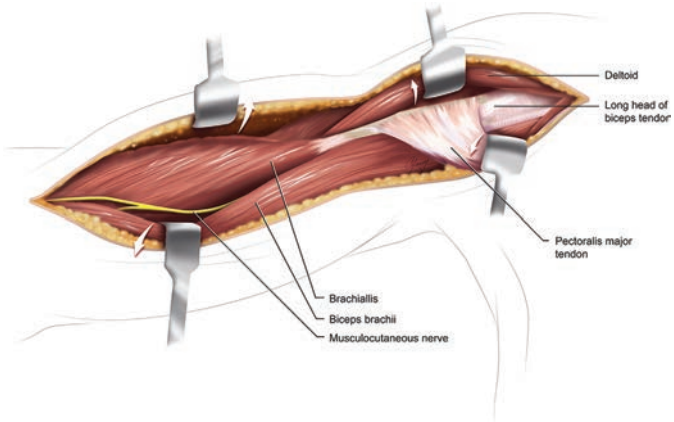

Figure 32.5 Anterior approach to the humeral shaft distal dissection. Identify the musculocutaneous nerve distally. Retract the biceps medially. Beneath it lies the anterior aspect of brachialis that covers the humeral shaft

2. In the anterior compartment of the distal third of the arm as it pierces the lateral intermuscular septum and lies between brachioradialis and brachialis muscles. Split brachialis along its midline; the lateral portion of the muscle then serves as a cushion

- **Axillary nerve** with over-retraction of the deltoid
- **Anterior circumflex humeral vessels** cross the operative field and have to be sacrificed
- **Musculo-cutaneous nerve** runs between the biceps and the brachialis muscles and emerges from the undersurface of the biceps a little above the elbow as the lateral cutaenous nerve of the forearm

Posterior approach to the humerus

Indications

- ORIF of humeral fractures (distal two-thirds)
- Management of humeral non-unions
- Exploration of radial nerve in the spiral groove

Position

Two positions are possible:

- Lateral position on operating table, affected side uppermost
- Prone, arm abducted 90°, elbow flexed 90° over a support and forearm dependent. A sandbag is placed under the affected shoulder

Landmarks

- Acromion
- Olecranon fossa

Incision

A longitudinal midline incision is made on the posterior aspect of the arm from 8 cm below the acromion to the olecranon fossa.

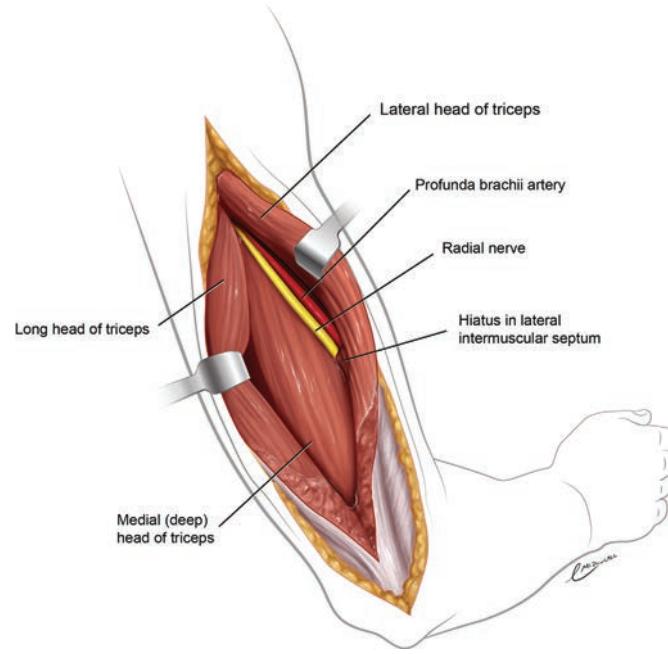

Figure 32.6 Posterior approach to humerus. The medial head lies below the other two heads. Develop the interval between the two heads by blunt dissection retracting long head medially and lateral head laterally. Identify and protect the radial nerve

Internervous plane

There is no true internervous plane. Dissection involves separating the long and lateral heads of the triceps.

Superficial dissection

Incise the deep fascia of the arm in line with the skin incision. Identify the gap between the lateral and long heads of the triceps muscle (this should be done high up proximally, which is easier) and develop the interval by blunt dissection. Distally split their common tendon along the line of the skin incision by sharp dissection. One must preserve the large motor branches of the lateral and long heads of triceps.

Deep dissection

The medial head of the triceps muscle lies beneath the long and lateral heads (V-shaped interval) (Figure 32.6).

The radial nerve and profunda brachii artery lie in the spiral groove and course obliquely laterally and distally along the superior border of the deep medial head of triceps.

Incise the medial head of the triceps in the midline down to the periosteum of the humerus and then strip muscle off bone by subperiosteal dissection.

Proximal extension is not possible since the deltoid muscle and the axillary nerve cross the operative field. Distal extension is possible over the olecranon onto the subcutaneous surface of the ulna. Avoid being too far proximally or distally for the pathology as you will struggle and need to extend your incision.

Structures at risk

The **radial nerve** is vulnerable in the spiral groove. At its most distant extent, the ulnar nerve is constrained at the point at which pierces the medial intermuscular septum as it enters the posterior compartment in the distal arm. It is usually necessary to identify and pass a vessel loop around the ulnar nerve with complex fractures of the distal humerus.

The **profunda brachii artery**, which travels with the radial nerve, may also be at risk.

Anterolateral approach to the distal humerus

Exposes the distal quarter of the humerus. The major advantage over the brachialis-splitting anterior approach is that it can be extended both proximally and distally, whereas the brachialis-splitting approach cannot be extended distally. Although can be used for ORIF distal humeral fractures a posterior approach is generally preferred.

Indications

- Exploration of the radial nerve

Position

The patient is positioned supine on the operating table. Tourniquet is applied. The arm is on an arm board, abducted 60°.

Landmarks

- Biceps muscle
- Flexion crease of the elbow

Incision

A curved longitudinal incision is made over the lateral border of biceps starting 10 cm proximal to the flexor crease of the elbow and ending just above the flexor crease of the elbow.

Internervous plane

There is no true internervous plane because both the brachioradialis muscle and the lateral half of the brachialis muscle are supplied well proximal to the incision by the radial nerve.

Superficial surgical dissection

Incise the deep fascia of the arm in line with the skin incision. The lateral cutaneous nerve of the forearm emerges between biceps and brachialis muscles and should be retracted clear of this incision. Identify and retract the biceps medially. Identify the interval between brachialis and brachioradialis and develop the intermuscular plane. Find the radial nerve at the elbow by exploring this oblique intermuscular plane gently with blunt dissection. Retract brachioradialis laterally and brachialis and biceps medially. Trace the radial nerve proximally until it pierces the lateral intermuscular system.

Deep surgical dissection

Staying on the medial side of the radial nerve, incise the lateral border of the brachialis muscle longitudinally, cutting down to bone, lifting it off the anterior aspect of the bone by subperiosteal dissection to expose the anterior aspect of the distal humerus.

The incision can be extended proximally by developing the plane between brachialis medially and the lateral head of triceps posterolaterally. Distally, the incision can be extended into the anterior approach of the elbow, developing the plane between the brachioradialis muscle and pronator teres. Care must be taken to avoid injury to the lateral cutaneous nerve of the forearm which emerges along the lateral side of the biceps tendon.

Structures at risk

- The **radial nerve** must be identified before any incision is made into the brachialis muscle or before periosteal elevation of the brachialis off the humerus occurs
- The **lateral cutaneous nerve of the forearm** can be injured at the distal end of the incision as it exits the biceps laterally

Examination corner

Trauma oral

Approaches and structures at risk for:

- Humeral shaft fracture with radial nerve palsy
- Extra-articular distal humeral fracture
- Intra-articular distal humeral fracture

Elbow

- Anterior approach
- Anterolateral approach
- Posterolateral approach
- Medial approach

Anterior approach to the elbow

The main use of this approach is to provide access to the neurovascular structures that are found in the cubital fossa.

Indications

- Repair lacerations to the median nerve, brachial artery and radial nerve
- Repair injuries to the biceps tendon
- Decompression of the median nerve

Position

The patient is positioned supine on the operating table, with a tourniquet. The arm is in the anatomical position, with the shoulder abducted and externally rotated, supported on a side table and the surgeon is facing the axilla.

Landmarks

- Mobile wad of three (brachioradialis, extensor carpi radialis longus (ECRL) and extensor carpi radialis brevis (ECRB))
- Medial border of the biceps tendon

Incision

A lazy-S incision is made over the anterior aspect of the elbow. Begin 5 cm above the flexor crease on the medial side of biceps. Curve the incision across the front of the elbow, then complete it by incising the skin along the medial border of the brachioradialis muscle.

Internervous plane

Proximally between brachioradialis (radial nerve) and brachialis (musculocutaneous nerve). Distally between pronator teres (median nerve) and brachioradialis (radial nerve).

Superficial surgical dissection

Mobilize the skin flaps widely. Incise the deep fascia in line with the skin incision and ligate the numerous veins that cross the elbow in this region. However, try to preserve the cephalic and basilic veins if possible.

The lateral cutaneous nerve of the forearm is identified and preserved; it is located in the interval between the biceps tendon and the brachialis muscle.

Identify the bicipital aponeurosis and cut it close to its origin at the biceps tendon. Be careful, as the brachial artery lies underneath it. By retracting the biceps laterally, the brachial artery, vein and median nerve will be found lying on the brachialis. The median nerve lies medial to the artery.

To identify the radial nerve, look between the brachialis and brachioradialis muscles – The nerve crosses in front of the elbow joint.

Deep surgical dissection

The approach is only really useful for exploring the neurovascular structures. It can be extended as far as the axilla and distally along the radial border of the forearm. Deep dissection is seldom required.

Structures at risk

Neurovascular structures in the cubital fossa may easily be damaged

- **Lateral cutaneous nerve of forearm** (vulnerable to injury in the distal quarter of the arm during incision of deep fascia)
- **Radial artery** which lies immediately deep to the bicepital aponeurosis.

Anatomy of the cubital fossa

Lying superficial to the bicipital aponeurosis are the median cubital vein, median cephalic vein and medial cutaneous nerve of the forearm.

The contents of the fossa from medial to lateral side are: The median nerve, brachial artery, brachial vein, tendon of biceps and, farther laterally, the radial nerve.

The relationship of the median nerve, brachial artery and brachial vein can be remembered by the mnemonic 'VAN' (vein, artery, nerve), which labels the structures from lateral to medial. They all pass medial to the biceps tendon under the bicipital aponeurosis.

Anterolateral approach elbow

Indication

- ORIF capitulum fractures
- Biceps avulsion injuries
- Neural compressions

Position

Supine on radiolucent arm board.Tourniquet

Landmarks

- Brachioradialis
- Biceps

Incision

Curved incision starting 5cm proximal to flexor crease along the lateral border of biceps. Continue distally by following the medial border of brachioradialis

Superficial dissection

Incise the deep fascia along the medial border of the brachioradialis. Identify radial nerve proximally at level of the elbow joint (between brachialis and brachioradialis). Develop the interval between brachiaradialis and the pronator teres interval

Deep dissection

To expose the capitulum and lateral compartment of the elbow a longitudinal incision is made in the anterior capsule of the elbow. To expose the proximal radius supinate the forearm to bring the supinator muscle anteriorly. Incise the muscle origin down to bone, lateral to the insertion of the biceps tendon

Dangers

- Lateral antebrachial cutaneous nerve of the forearm
- Radial nerve
- PIN
- Recurrent branch of the radial artery

Posterolateral approach elbow (Kocher's approach)

This approach stays behind the lateral epicondyle

Indications

- ORIF of radial head/neck fractures
- Radial head excision/replacement
- Terrible triad injuries

Position

The patient is positioned supine with a well-padded arm board. The arm is abducted, the elbow flexed and the forearm pronated. A high arm tourniquet may be used

Landmarks

- Lateral epicondyle of the humerus
- Radial head

Incision

A curved incision is made beginning posterior to the lateral epicondyle and passing distally and medially to the subcutaneous border of the ulna around 6 cm from the tip of the olecranon.

Internervous plane

This lies between the anconeus (radial nerve) and ECU (posterior interosseous nerve – PIN).

Superficial surgical dissection

The deep fascia is incised in line with the skin incision. The interval between the triceps muscle posteriorly and brachioradialis and ECRL anteriorly is developed to expose the lateral condyle, lateral joint capsule and the radial collateral ligament. Distally develop the plane between the extensor carpi ulnaris (ECU) and the anconeus is easier to develop.

The supinator lies deep to and between the anconeus and ECU.

Deep surgical dissection

Reflect the periosteum from the anterior and posterior surfaces of the distal humerus. Fully pronate the forearm to move the posterior interosseous nerve (PIN) away from the operative field. Incise the capsule of the elbow joint longitudinally to reveal the capitellum, radial head and annular ligament. Do not incise the capsule too far anteriorly as the radial nerve runs over the front of the anterolateral portion of the elbow capsule.

Do not continue dissection below the annular ligament because the PIN is vulnerable to injury.

Structures at risk

- Posterior interosseous nerve in the supinator muscle is at risk if the dissection is carried distal to the annular ligament. Pronation of the forearm moves the nerve away from the approach
- Radial nerve is at risk anteriorly

Posterior approach to the elbow

Indications

- ORIF of distal humeral fractures
- Treatment of non-unions

Position

The patient is either prone or in the lateral decubitus position. The elbow is flexed 90° over a support and the forearm is dependent. A tourniquet is applied.

Landmarks

- Olecranon process

Incision

The incision is a longitudinal midline incision on the posterior aspect of the elbow beginning 5 cm proximal to the olecranon. The incision curves round lateral to the olecranon (avoiding the tip of the olecranon).

Internervous plane

There is no internervous plane in this approach as the approach involves detaching the extensor mechanism of the elbow.

Superficial surgical dissection

Incise the deep fascia of the forearm and develop a medial and lateral skin flap. The medial skin flap should be large enough to expose the medial epicondyle. Identify the ulnar nerve as it lies on the bony groove at the back of the medial epicondyle. Fully dissect out the ulnar nerve and pass tapes around it.

Deep surgical dissection

Either a triceps turndown or a chevron osteotomy of the olecranon. The chevron points distally. Dissect around the medial and lateral borders of the humerus to expose the entire posterior surface of the distal fourth of the humerus. Distally, exposure can be extended along the subcutaneous border of the ulna.

Structures at risk

- The **ulnar nerve** must be kept clear of the operative field during all stages of dissection
- The **median nerve** lies anterior to the distal humerus and may be endangered if anterior structures are not stripped off the distal humerus in a strict subperiosteal plane
- The **radial nerve** as it pierces the lateral intermuscular septum 7–10 cm proximal to the lateral epicondyle
- The **brachial artery** lies with the median nerve in front of the elbow

Medial approach to the elbow

Indications

- MCL repair
- ORIF of coronoid process/medial humeral condyle

Position

The patient is positioned supine on the operating table, and rolled slightly towards the involved upper extremity by padding under the contralateral shoulder and hip. The involved extremity is placed on a side table, the shoulder is abducted and externally rotated, and the elbow joint is flexed.

Landmarks

- Medial epicondyle of the humerus

Incision

A curved incision 10 cm long is made, centred on the medial epicondyle.

Superficial surgical dissection

Proximally the dissection is between the brachialis (musculocutaneous nerve) anteriorly and the triceps (radial nerve) posteriorly. Incise the subcutaneous tissue and develop anterior and posterior skin flaps. The ulnar nerve is found proximal to the cubital tunnel. The superficial flexor muscles of the forearm are visible as they pass directly from their common origin on the medial epicondyle of the humerus.

Distally, the ulnar nerve becomes tethered and splits as it enters the flexor muscle mass; therefore, the plane of dissection is between pronator teres (median nerve) and brachialis (musculocutaneous nerve), taking care not to injure the median nerve, which enters the pronator teres anteriorly. Exposure of the elbow joint can be enhanced with a medial epicondyle osteotomy.

Structures at risk

- **Ulnar nerve**
- **Median nerve**
- Branches of the **medial cutaneous nerve** of the forearm

Forearm

- Anterior approach to the radius
- Exposure of the shaft of the ulna
- Dorsal approach to the radius

This offers excellent safe exposure of the distal two-thirds of the radius. Exposure of the proximal one-third endangers the PIN.

Anterior approach to the radius (Figure 32.7)

Indications

- ORIF of radius fracture
- Exploration of the radial artery
- Exploration, biopsy or excision of tumours

Position

The patient is positioned supine. The arm is on an arm board, with the shoulder abducted and externally rotated. The elbow is extended and the forearm supinated. Tourniquet with exsanguination.

Landmarks

- Biceps tendon
- Brachioradialis
- Styloid process of radius

Incision

A straight incision is made from the anterior flexor crease of the elbow, just lateral to the biceps tendon down to the styloid process of the radius.

Internervous plane

Proximally, it lies between the brachioradialis (radial nerve) and pronator teres muscle (median nerve). Distally, the internervous plane lies between the brachioradialis (radial nerve) and the flexor carpi radialis (FCR) (median nerve).

Superficial dissection

Reflect the skin edges. Incise the deep fascia of the forearm in line with the skin incision. The medial border of brachioradialis is identified and a plane is developed between it and FCR distally. More proximally the plane is between pronator teres and brachioradialis.

Begin distally and work proximally. Identify and protect the superficial radial nerve, running on the undersurface of brachioradialis.

May need to ligate the recurrent radial leash of vessels from the radial artery to allow mobilization of the brachioradialis laterally as they act as tethers. The radial artery is beneath brachioradialis in the middle part of the forearm; therefore, it is quite close to the medial edge of the wound.

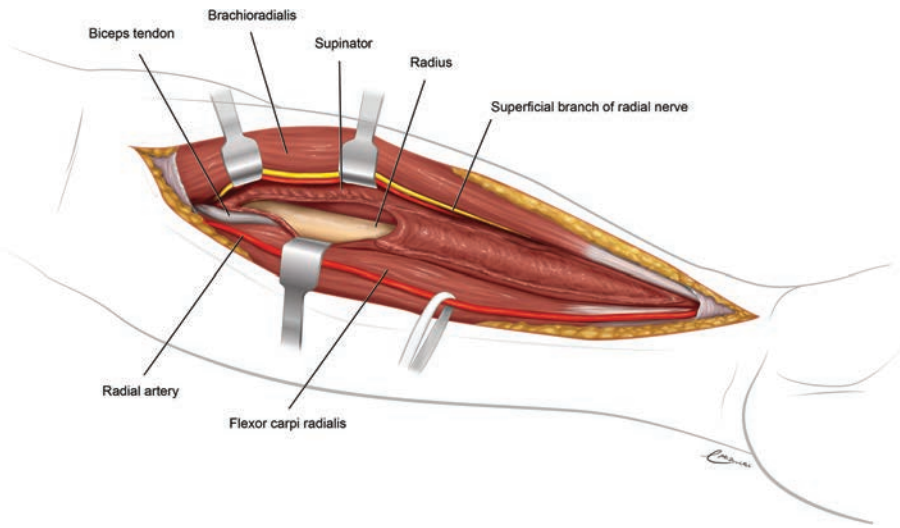

Figure 32.7 Anterior approach to the radius (Henry)

Deep dissection

Deep dissection of the proximal third

Follow the biceps tendon to its insertion into the bicipital tuberosity of the radius. Just lateral to the tendon is a small bursa. Incise the bursa to gain access to the proximal part of the shaft of the radius.

Deepen the wound on the lateral side of the biceps tendon, as the radial artery lies superficial and just medial to the tendon. Fully supinate the forearm and expose the insertion of the supinator muscle into the anterior aspect of the radius. Next, incise the supinator muscle along the line of its broad insertion. Continue subperiosteal dissection laterally, stripping the muscle off bone; it carries with it and protects the PIN. Keep the periosteal elevator hard against the bone to protect the PIN.

Note that the line of the muscle fibres of supinator and the adjacent pronator teres lie approximately at right angles to one another.

Deep dissection of the middle third

Deep to the brachioradialis and FCR are the supinator, pronator teres, flexor digitorum superficialis (FDS) and, most distally, pronator quadratus. To reach the anterior surface of the radius, pronate the arm so that the insertion of pronator teres on to the lateral aspect of the radius is exposed.

Detach this insertion, stripping the muscle off medially in a subperiosteal plane. This manoeuvre also detaches FDS from the anterior aspect of the radius.

Deep dissection of the distal third

- Two muscles, flexor pollicis longus (FPL) and pronator quadratus, arise from the anterior aspect of the distal third of the radius
- To reach the bone, partially supinate the forearm
- Incise the periosteum of the lateral aspect of the radius and continue a subperiosteal dissection, stripping the two muscles medially towards the ulna
- Can be extended distally to expose the wrist joint and also proximally into an anterolateral approach to the elbow and humerus

Structures at risk

- Posterior interosseous nerve
- Superficial branch of the radial nerve
- Radial artery

Exposure of the shaft of the ulna

This approach is along the subcutaneous border of the ulna exploiting the internervous plane between the extensor carpi ulnaris (ECU) (supplied by the PIN) and flexor carpi ulnaris (FCU) (supplied by the ulnar nerve). In the region of the olecranon it is between anconeus (supplied by the radial nerve) and FCU (supplied by the ulnar nerve)

Posterior approach to the radius (Thompson)

Indications

- ORIF of radius fractures
- Exploration of the PIN

Position

The patient is positioned supine, with an arm board. The shoulder is abducted, the elbow is flexed and the forearm is pronated. Tourniquet with exsanguination.

Landmarks

- Lateral epicondyle of humerus
- Lister's tubercle – One-third of the way across the dorsum of the wrist from the styloid process of the radius

Incision

A straight skin incision is made from just anterior (1.5 cm) to the lateral epicondyle of the humerus along the dorsal aspect of the forearm to just distal to the ulnar side of Lister's tubercle at the wrist.

Internervous plane

- Proximally between ECRB (radial nerve) and EDC (PIN)
- Distally between ECRB (radial nerve) and EPL (PIN)

Superficial dissection

The incision is carried through subcutaneous tissue and deep fascia. The space between the extensor carpi radialis brevis (ECRB) and the extensor digitorum communis (EDC) is developed to reveal the upper third of the shaft of the radius covered by supinator muscle (Figure 32.8).

Deep dissection

Proximal

The supinator cloaks the upper third of the radius. The PIN emerges 1 cm proximal to the distal edge of the supinator (Figure 32.9).

Two techniques exist for identifying and preserving the PIN as it travels through supinator:

- Proximal to distal: Detach the origin of ECRB and ECRL from the lateral epicondyle. Identify the PIN and carefully dissect out the nerve through the substance of supinator in a proximal to distal direction, taking care to preserve multiple motor branches to the muscle itself
- Distal to proximal: Identify the PIN as it emerges from the supinator. Follow the nerve proximally through the substance muscle

After the nerve has been identified fully supinate the arm to bring the anterior surface of the radius into view. Detact the supinator insertion off the anterior aspect of the radius subperiosteally to expose the proximal third of the radial shaft.

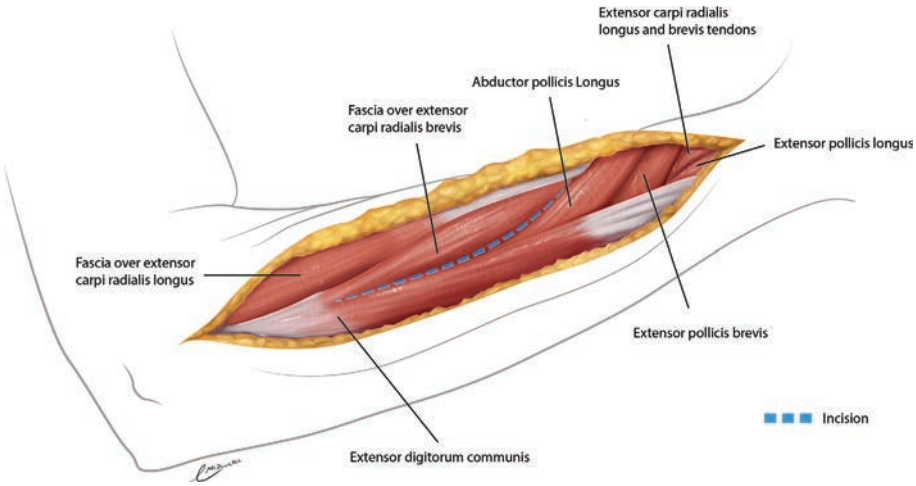

Figure 32.8 Dorsal approach to the radius (superficial dissection). The deep fascia is divided in line with the skin incision. The internervous plane lies between the ECRB and the EDC.

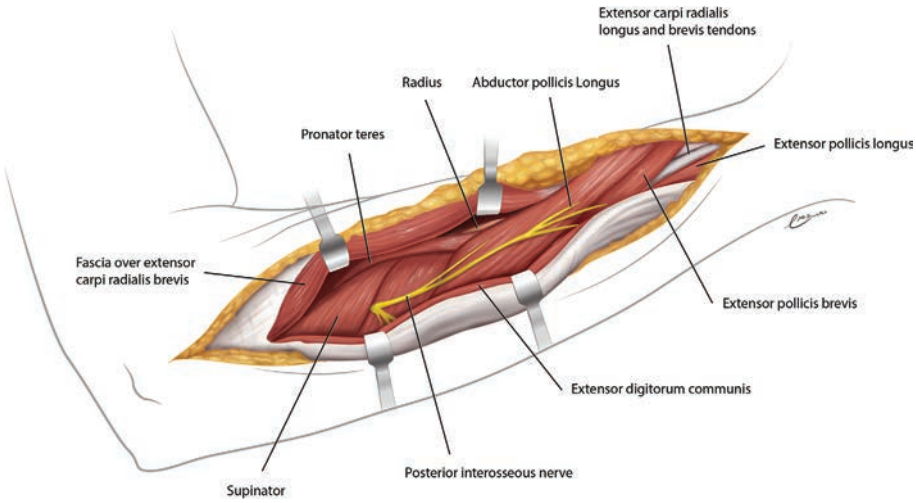

Figure 32.9 Deep dissection proximally. The posterior interosseous nerve emerges between the superficial and deep head of the supinator muscle about 1 cm proximal to the distal edge of the muscle.

Distal

The abductor pollicis longus and extensor pollicis brevis cover the dorsal aspect of the radius. Retract them off bone, making an incision along the lateral border (to expose the middle third of the radius), or the medial border (to expose the distal third of the radius).

This can be extended distally on to the dorsal side of the wrist or proximally to the lateral epicondyle of the humerus.

Structures at risk

- PIN (Figure 32.10)

Wrist and hand

Dorsal approach to the wrist (Figure 32.11)

Indications

- Synovectomy and repair of extensor tendons in rheumatoid arthritis
- Wrist fusion
- ORIF of wrist fractures

Position

The patient is placed supine on the operating table. The arm is on an arm board, forearm pronated. Tourniquet with exsanguination.

Landmarks

- Radial and ulnar styloid

Incision

An 8-cm longitudinal incision is made on the dorsal aspect of the wrist, crossing the wrist joint midway between the radial and ulnar styloid processes.

The incision begins 3 cm proximal to the wrist joint and ends 5 cm distal to it.

Internervous plane

There is no true internervous plane. As the muscles involved are innervated proximally, the intermuscular plane can be used safely.

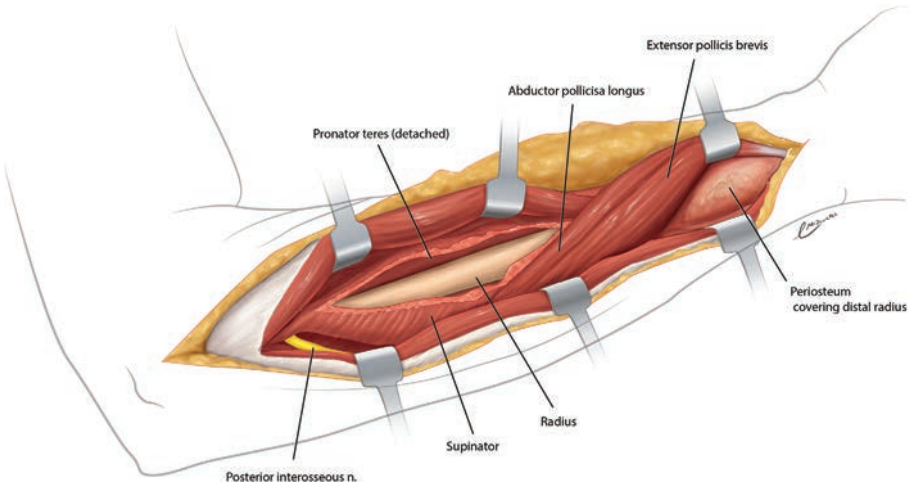

Figure 32.10 The PIN is kept away from the incision by fully supinating the forarm and detaching the insertion of the supinator from the anterior aspect of the radius

Superficial surgical dissection

Incise the subcutaneous fat in line with the skin incision to expose the extensor retinaculum.

Deep surgical dissection

This is most commonly through the third extensor compartment.

Incise the extensor retinaculum over the third extensor compartment and take the extensor pollicis longus tendon out of the compartment. Subperiosteally lift the tendons of the fourth compartment from the dorsal aspect of the distal radius. Mobilize the extensor tendons in an ulnar and radial direction to expose the wrist joint capsule. The extensor retinaculum is preserved; if the EPL tendon is tight during closure, consider leaving it outside the extensor retinaculum.

Structures at risk

- **Superficial radial nerve** emerging from the dorsal margin of brachioradialis
- **Radial artery**

Extensile measures

Can extend exposure proximally over the distal third of the radius, reflecting APL and EPB.

Hip

- Anterior approach (Smith–Petersen)
- Anterolateral approach (Watson–Jones)
- Direct lateral approach (Hardinge)
- Trochanteric approach
- Posterior approach
- Medial approach

Anterior approach to the hip (Smith–Petersen)

This gives safe access to the hip joint and ilium. It uses the internervous plane between sartorius (femoral nerve) and tensor fascia lata (superior gluteal nerve).

Indications

- Open reduction for developmental dysplasia of the hip (DDH)
- Synovial and joint biopsies
- Total hip arthroplasty (THA)
- ORIF of anterior column fractures
- Exploration of femoral nerve
- Pelvic osteotomy

Position

The patient is positioned supine, close to the edge of the table. A sandbag may be used to elevate the affected hip.

Landmarks

- Anterior superior iliac spine (ASIS)
- Iliac crest

Incision

The traditional incision has both vertical and horizontal components. Make a longitudinal incision along the anterior half of the iliac crest to the ASIS and then curve the incision down vertically 10 cm heading towards the lateral side of the patella.

An alternative is the bikini type incision that continues the incision medially from the ASIS and gives better cosmesis but is less extensile.

Internervous plane

The approach is a true internervous one between the femoral nerve and the superior gluteal nerve. In the superficial dissection this is between sartorius (femoral nerve) and tensor fascia lata (superior gluteal nerve), and in the deep dissection the plane lies between the rectus femoris (femoral nerve) and the gluteus medius (superior gluteal nerve).

Superficial dissection

Deepen the incision through fat and then divide superficial and deep fascia. Externally rotate the leg to stretch the

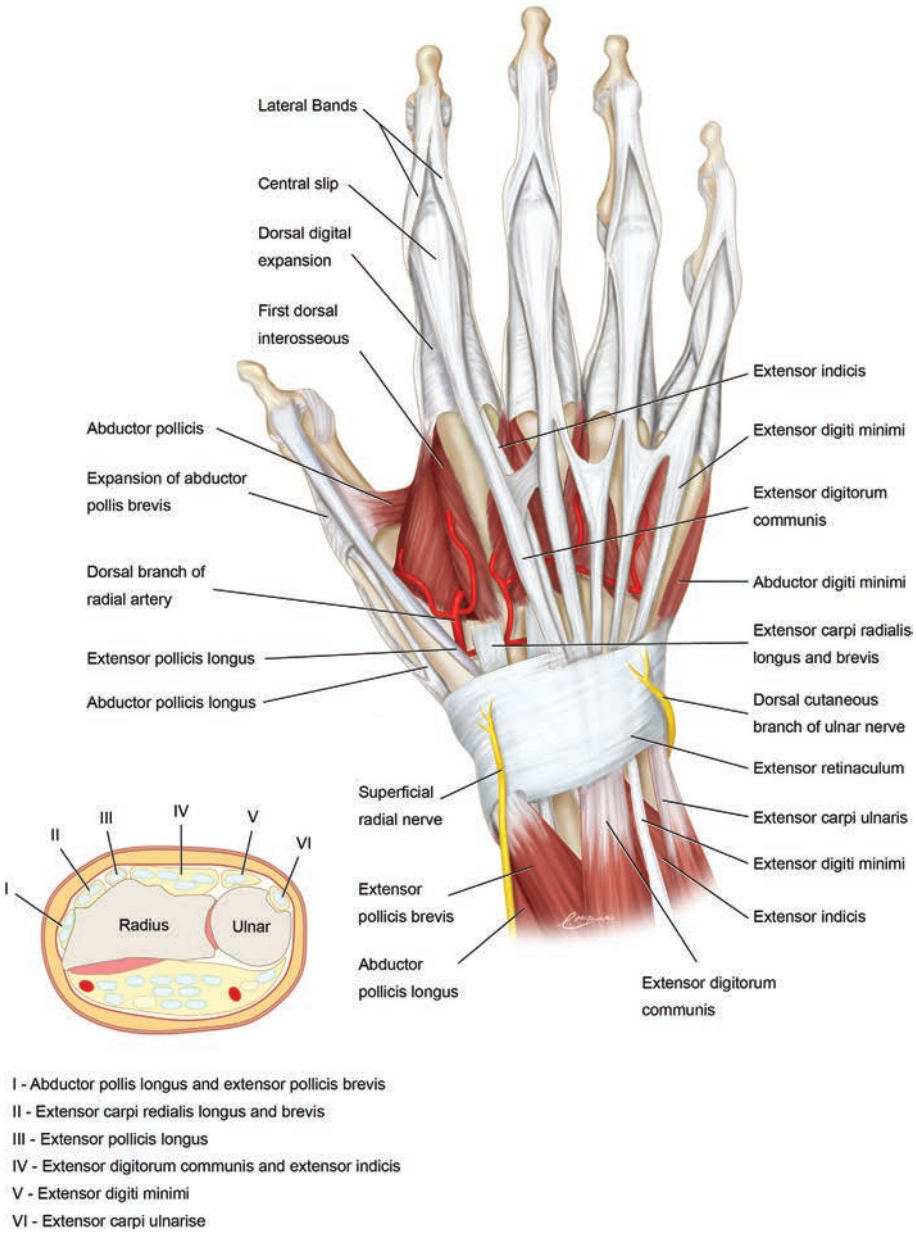

Figure 32.11 Dorsal approach to the wrist joint. The six extensor tendon compartments of the wrist. I – Extensor pollicis brevis (EPB), abductor pollicis longus (APL); II – Extensor carpi radialis longus (ECRL), extensor carpi radilalis brevis (ECRB) these two compartments are divided by Lister's tubercle of the radius; III – Extensor pollicis longus (EPL), IV – Extensor digitorum communis (EDC); V – Extensor digiti minimi (EDM); and VI – Extensor carpi ulnaris (ECU)

sartorius muscle and make it more prominent. Open up the interval between the tensor fascia lata laterally and sartorius medially. The best place to find the gap is 2–3 cm below the ASIS.

Avoid cutting the lateral femoral cutaneous nerve, which pierces the deep fascia of the thigh close to the intermuscular interval.

Retract sartorius upwards and medially and the tensor fascia lata downwards and laterally. Detach the iliac origin of the tensor fascia lata to develop the internervous plane. Ligate the ascending branch of the lateral femoral circumflex artery as it crosses the gap between the two muscles below the ASIS.

Deep dissection

Open up the plane between rectus femoris and gluteus medius. Detach the straight head of rectus femoris from its origin on the AIIS and separate the reflected head from the hip joint capsule. Retract the gluteus medius laterally.

The capsule of the hip joint is now exposed. Inferomedially, iliopsoas attaches to the lesser trochanter. Adduct and fully externally rotate the leg to put the capsule on stretch. The joint capsule over the anterior aspect of the femoral neck is brought into view and a capsulectomy performed. The hip is then dislocated.

Dissection can be extended along the iliac crest and down the femur, keeping in the internervous plane to expose the

entire length of the anterior femur. The interval between vastus lateralis and rectus femoris is split, which gives excellent exposure of the shaft of femur.

Structures at risk

Injury to the lateral cutaneous nerve of the thigh with disturbing dysaesthesia of the thigh is a common problem. It reaches the thigh by passing over, behind or through the sartorius muscle about 2.5 cm below the ASIS.

In the distal part of the wound the ascending branch of the lateral circumflex artery should be identified prior to ligation and division.

The femoral nerve is at risk if one strays from the internervous plane.

Anterolateral approach to the hip (Watson-Jones)

Indications

- Open reduction of femoral neck fractures
- Hemiarthroplasty hip
- THA. This approach avoids cutting the gluteus medius muscle but involves considerable pulling (traction) on the gluteus medius and TFL muscles and potentially on the superior gluteal nerve. If used for THA it often requires additional division of gluteus medius and minimus which lie over the anterior capsule for adequate exposure which may lead to a Trendelenburg gait. Therefore rarely used for THA. More recently a mini invasive technique has been introduced.

Position

- Traction table (for open reduction of femoral neck fractures), or
- Supine with a large sandbag under the buttock

Incision

The incision is begun 2–3 cm distal and lateral to the ASIS and continues to the posterior border of the greater trochanter. It then curves anteriorly along the line of the femoral shaft.

Internervous plane

There is no true internervous plane as gluteus medius and tensor fascia lata share the same nerve supply (superior gluteal nerve).

Surgical dissection

The interval between gluteus medius and tensor fascia lata is often difficult to delineate but can be identified more easily by beginning the separation midway beween the ASIS and the greater trochanter. Separate these muscles up to the iliac crest. Small branches of the superior gluteal nerve run in this interval and should be preserved if possible. Beneath this is a layer of fat which must be mobilized and the reflected head of rectus femoris should be detached to expose the hip capsule. The leg is externally rotated and adducted to deliver the femur into the wound.

Stuctures at risk

- Femoral neurovascular structures
- Superior gluteal nerve
- Potential damage to gluteus medius during femoral canal preparation (abductor limp)

Advantages

- Reduced risk of dislocation with THA
- Muscle sparing if minimally invasive technique is used
- Good acetabular exposure

Disadvantages

- Difficult visualization of femoral canal
- Risk of femoral shaft fracture, especially when dislocating the hip

Extensile approach

- Detach anterior one-third gluteus medius from greater trochanter
- Osteotomy greater trochanter

Direct lateral approach to the hip (Hardinge approach)

Indications

- THA
- Hemiarthroplasty hip

Position

- Lateral position with kidney supports and hip trough, or
- Supine with greater trochanter at the edge of the table to allow the buttock muscles and gluteal fat to fall away posteriorly from the operative field

Landmarks

- ASIS
- Greater trochanter
- Line of the femur

Incision

A longitudinal midlateral incision centred over the tip of the greater trochanter is made, running 10 cm proximal and distal to this point in line with the femoral shaft.

Internervous plane

There is no true internervous plane. The fibres of gluteus medius and vastus lateralis are split in their own line.

Superficial surgical dissection

Incise the fat in line with the skin incision; a swab may be used to sweep away fat for 1 cm or so from the fascia lata to facilitate closure at the end of the operation.

The fascia lata is incised in line with the skin incision. A Charnley bow retractor is inserted into the anterior and

posterior borders of the fascia lata. The trochanteric bursa should be excised if thickened.

Deep surgical dissection

The gluteus medius and vastus lateralis should come into view. Fibres of gluteus medius are split 3 cm above the tip of the greater trochanter in the direction of the fibres at the junction of its anterior and middle thirds. Do not go more than 3 cm above the upper border of the greater trochanter as a more proximal dissection may damage branches of the superior gluteal nerve.

The incision is carried down to bone over the greater trochanter and then distally into the vastus lateralis muscle along the anterior surface of the femur. Elevate an anterior flap of gluteus medius and vastus lateralis from the greater trochanter using cutting diathermy whilst externally rotating the leg. The tendon of gluteus minimus is divided at its insertion on the anterior part of the greater trochanter.

Enter the capsule joint using a longitudinal T incision and dislocate the femoral head. Define the level of femoral neck cut by feeling for the lesser trochanter.

Template the femoral neck cut if necessary. The femoral neck is then divided using an oscillating saw.

With an intracapsular fractured neck of femur the remnant of the femoral neck is cut and the femoral head extracted from the acetabulum using a corkscrew, sharply dividing capsular remnants and ligamentum teres with scissors if these impede head extraction.

The approach can be extended distally, splitting the vastus lateralis muscle in the line of its fibres to expose the shaft of the femur; however, this requires more muscle dissection than with a posterior approach. The incision cannot be extended proximally.

Structures at risk

- **Superior gluteal nerve** (see above)
- **Femoral nerve**: The most lateral structure in the anterior neurovascular bundle of the thigh is vulnerable to inappropriately placed retractors
- The **femoral artery and vein** are also vulnerable to inappropriately placed retractors
- The transverse branch of the **lateral circumflex femoral artery** is cut as the vastus lateralis is mobilized and can cause persistent bleeding, especially if the end retracts into muscle

Trochanteric approach to the hip

Advantages

- Excellent exposure of acetabulum and proximal femur

Disadvantages

- Increased bleeding
- Trochanteric bursitis
- Technical difficulty wiring back the trochanter
- Wire breakage and non-union of the trochanter

Indications

- Complex primary THA
- Revision hip surgery

Position

The patient is positioned supine, close to edge of the operating table so that the buttock hangs over the sandbag under the buttock. The buttock skin and fat should fall away posteriorly.

Landmarks

- ASIS
- Greater trochanter
- Shaft of femur

Incision

Flex the leg 30° and adduct it across the opposite knee. This makes the greater trochanter more prominent and moves the tensor fascia lata anteriorly.

A straight 15-cm longitudinal incision is made, centred on the tip of the greater trochanter. The incision crosses the posterior third of the trochanter before running down the shaft of the femur.

Internervous plane

There is no true internervous plane as gluteus medius and tensor fascia lata share the same nerve supply (superior gluteal nerve).

Superficial surgical dissection

Incise fat in the same line as the skin incision. Incise fascia lata at the posterior margin of the greater trochanter. Charnley retractors are used to retract the tensor fascia lata. Place a retractor deep to gluteus medius and minimus and bluntly dissect the fat pad off the anterior portion of the joint capsule.

Identify the origin of vastus lateralis at the vastus lateralis ridge.

Deep surgical dissection

A gallbladder clamp is inserted in the joint and pushed through the capsule posterior to the insertion of the gluteus medius on the greater trochanter.

Be sure that the instrument is inserted in the interval between the superior aspect of the neck and the capsule rather than outside the capsule.

Furthermore, make sure that the instrument pierces the capsule posteriorly rather than posterolaterally because otherwise the full thickness of the trochanter with the insertions of the gluteus medius and minimus will not be removed.

Osteotomize the trochanter with a Gigli saw and reflect it upwards with the attached gluteus medius and minimus muscles. Make sure that the sciatic nerve is not trapped

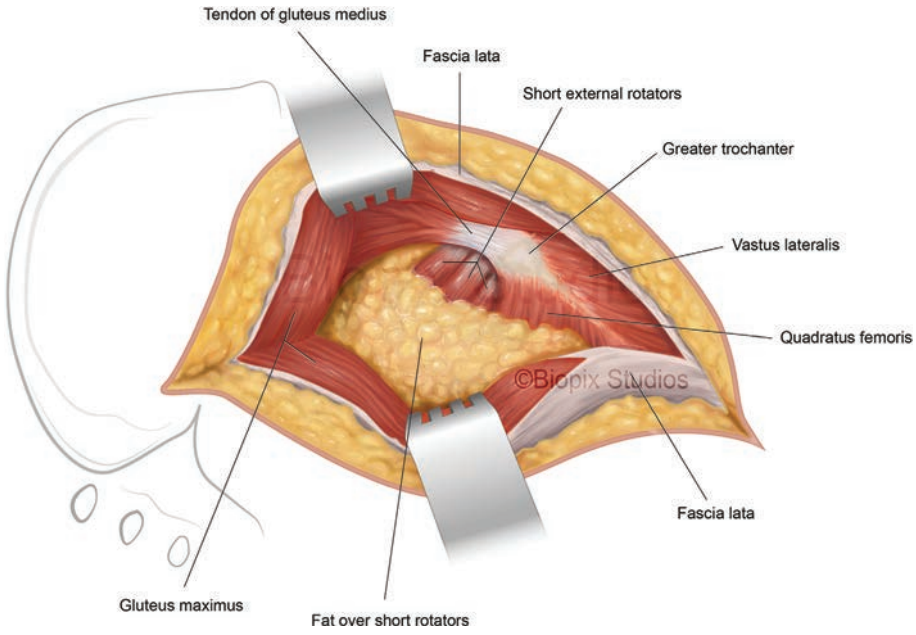

Figure 32.12 Posterior approach to the hip. The short external rotators are exposed. Check the position of the sciatic nerve within the substance of the fatty tissue. Carefully position Charnley bow retractors around the gluteus maximus muscle and avoid catching the nerve. Place stay sutures into the piriformis and gluteus maximus insertion onto the femur

between Gigli saw and bone[e]. Release the soft tissues. The short rotators are detached from the trochanter.

Retract the trochanter with attached gluteus medius and minimus and the underlying capsule posteriorly.

Longitudinal incision into the capsule of the hip joint

Dislocate the hip by adducting the thigh and, to a lesser extent, by external rotation. After the hip has been dislocated use a Gigli saw to perform an osteotomy of the femoral neck.

Structures at risk

- Femoral nerve
- Femoral artery and vein
- Sciatic nerve

Extensile approach

The skin incision can be extended down the lateral aspect of the thigh, splitting vastus lateralis to gain access to the lateral aspect of the femur.

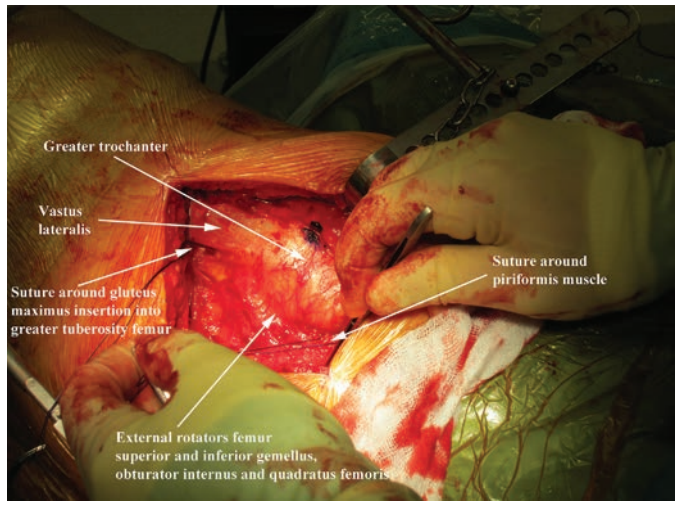

Figure 32.13 Intraoperative picture posterior approach to hip wuth sutures around piriformis tendon and gluteus maximus inserion into greater tuberosity femur

Posterior approach to the hip (Figure 32.12 and Figure 31.13)

'My preference in total hip replacement would be . . . because . . .'

Indications

- THA
- Hemiarthroplasty
- Open reduction of posterior hip dislocations
- ORIF of posterior acetabular wall fractures
- Resurfacing hip arthroplasty
- Revision hip surgery

Advantages

- Easier
- No damage to the hip abductors
- Good access to the femur and particularly the acetabulum

Disadvantages

- Higher dislocation rate (controversial)

Position

- Lateral position, affected limb uppermost

[e] Safety check: Touch the Gigli saw with diathermy.

Landmarks

- Greater trochanter

Incision

- A longitudinal straight incision (with the hip flexed 60°) beginning a hand's breadth below the iliac crest, moving forwards over the greater trochanter and down the shaft of the femur. When the leg is straightened, the incision is curved
- Alternatively, a 15–20-cm curved incision centred over the posterior aspect of the greater trochanter

Internervous plane

There is no true internervous plane as the gluteus maximus is split in the line of its fibres, but is not significantly denervated because its nerve supply is well medial to the split.

Superficial surgical dissection

Incise fascia lata on the lateral aspect of the femur to uncover the vastus lateralis. Split the fibres of gluteus maximus proximally by blunt dissection. Retract the split edges of fascia lata and gluteus maximus with a Charnley bow retractor. The short external rotators of the hip lie under a layer of fat; a useful landmark to locate them is the posterior border of the gluteus medius.

Deep surgical dissection

The short external rotators are seen on the posterolateral aspect of the femur (piriformis, superior gemellus, obturator internus, inferior gemellus, quadratus femoris).

Internally rotate the leg to put the short external rotators on stretch and to pull the operative field away from the sciatic nerve. Identify and protect the sciatic nerve lying on the short external rotators encased in fatty tissue. Insert stay sutures into the short external rotator muscles. Identify piriformis and detach it sharply with a scalpel fairly close to the femur origin or you may have difficulty reattaching it at the end of the procedure. Detach the muscles close to their femoral insertions and reflect them backwards, laying them over the sciatic nerve. Try not to detach all of quadratus femoris if possible as the muscle contains vessels from the lateral circumflex femoral artery, which can cause persistent bleeding. The capsule of the hip joint is now exposed and can be excised with a T-shaped incision. The hip is dislocated by internally rotating and adducting the leg.

The exposure can be extended distally by detaching all of quadratus femoris and the insertion of gluteus maximus tendon on to the proximal femur. Beware of a large perforating vessel beneath the tendon of gluteus maximus. This is needed for a resurfacing procedure. It can be further extended by dissecting behind the vastus lateralis to expose a fracture or to perform an extended trochanteric osteotomy (osteotomize one-third of the lateral femur to a length of 12–14 cm from the tip of the greater trochanter). The posterior column can be reached by dissecting behind the gluteus medius at the proximal extent of the exposure.

Structures at risk

- Sciatic nerve is very near the operative field and must be appreciated at all times during surgery
- Branches of the inferior gluteal artery are invariably injured when the gluteus maximus is separated

We have described some variations in technique to be comprehensive. Mention your own preferred incision if asked by the examiners to describe the posterior approach to the hip joint. Mentioning the different variations described may unnecessarily complicate your answer and confuse the examiners.

Examination corner

Basic science oral 1: Picture of posterior aspect of the hip with short external rotators
Candidate was asked to name them and describe the posterior approach for a THA.

Medial approach to the hip

Indications

- Open reduction of DDH
- Psoas release
- Obturator neurectomy

Position

The patient is positioned supine with the affected hip flexed, abducted and externally rotated. The sole of the foot on the affected side should lie along the medial side of the contralateral knee.

Landmarks

- Adductor longus
- Pubic tubercle

Incision

A longitudinal incision is made on the medial side of the thigh starting at a point 3 cm below the pubic tubercle. The incision runs down over the adductor longus as long as is required for exposure of the femur.

Internervous plane

The superficial dissection does not use a true internervous plane since adductor longus and gracilis are both supplied by the anterior division of the obturator nerve. More deeply, the plane lies between the adductor brevis (anterior division of obturator nerve) and adductor magnus (two nerve supply: Adductor portion by the posterior division of the obturator nerve, and ischial portion by the tibial part of the sciatic nerve).

Superficial surgical dissection

Develop a plane between gracilis and adductor longus. This is best done using blunt dissection.

Deep surgical dissection

Continue the dissection between adductor brevis and adductor magnus to uncover the posterior division of the obturator nerve. Feel the lesser trochanter in the floor of the wound. Iliopsoas can be divided, which exposes the anterior and inferior aspect of the hip capsule.

Structures at risk

The anterior and posterior branches of the **obturator nerve** lie on either side of adductor brevis and are at risk during the dissection. The **medial femoral circumflex artery** passes around the medial side of the distal part of the psoas tendon; it is in danger, especially in children, if the psoas tendon is not isolated and cut under direct vision.

Trochanteric slide osteotomy (Ganz)

Indications

- Surgical dislocation
- THA or revision

Position

- Lateral position, affected limb uppermost

Landmarks

- Greater trochanter

Incision

A longitudinal incision is made which is 15–20 cm in length and centred over the greater trochanter.

Internervous plane

Deep dissection is in the plane between the gluteus medius (superior gluteal nerve) and piriformis (n. to piriformis S2).

Superficial surgical dissection

The fascia lata is incised in line with the skin incision. A Charnley bow retractor is inserted into the anterior and posterior borders of the fascia lata. The trochanteric bursa may obscure the posterior border of gluteus medius and vastus lateralis.

Deep surgical dissection

A soft-tissue plane is created 5 mm anterior to the posterior border of the gluteus medius and dissection continued to the posterior border of the vastus lateralis at the vastus ridge. An osteotomy is performed using an oscillating saw to create a sleeve of the greater trochanter which is 10–15 mm thick. The piriformis should remain attached posteriorly. The osteotomy fragment is then retracted anteriorly and the gluteus minimus is reflected off the capsule to expose the anterior surface of the capsule.

Z- or reverse Z-capsulotomy is made which preserves the branches of the medial circumflex femoral artery. The medial capsular split is continued to separate it from the femoral neck and calcar. The proximal limb follows the margin of the acetabulum posteriorly, taking care not to damage the acetabular labrum. The hip can be now be dislocated.

Structures at risk

- Deep branch of the medial circumflex femoral artery

Pelvis and acetabulum

Ilioinguinal approach

It may be worthwhile mentioning that you would seek help from a general surgeon as dissection involves isolating and mobilizing femoral vessels/nerves and spermatic cord until you have become familiar with the approach. The approach is used to expose the inner aspect of the innominate bone from the sacro-iliac joint to the pubic symphysis allowing access to the anterior wall, anterior column and quadrilateral plate.

Indications

- Anterior wall and column fractures
- Transverse and T-type fractures with mainly anterior displacement
- Selected both column fractures
- Anterior column with posterior hemi-transverse fractures

Position

Supine with arms abducted. Radiolucent fracture table

Landmarks

- ASIS, pubic tubercle

Incision

- Line starting over the anterior iliac crest, 5 cm above the ASIS to around 1 cm above pubic tubercle

Internervous plane

There is no true internervous plane.

Superficial surgical dissection

The lateral cutaneous nerve of the thigh will lie in the lateral aspect of the incision and often will need to be sacrificed. Dissection is carried out through the aponeurosis of external oblique and rectus sheath medially. The spermatic cord in men and the round ligament in women are present in the superficial inguinal ring, and need to be isolated in a sling. Iliacus is elevated off the inner wing of the ilium.

Deep surgical dissection

The rectus abdominis is divided medially just above the pubic symphysis to develop space between it and the bladder. This part of the approach can be used to fix pubic symphysis diastasis.

Divide the internal oblique and transversus abdominis that form the posterior wall of the inguinal canal. Be careful of the inferior epigastric vessels which lie just medial to the deep inguinal ring at this stage. This should expose the peritoneum. Push it up with a swab to expose the femoral vessels.

One sling is passed around the femoral vessels and one around the iliopsoas tendon with femoral nerve. The key is

creating three windows through which bony surgery can be performed:

- Medial window – Lies medial to the femoral vessels, gives access to symphysis pubis
- Middle window – Lateral to the femoral vessels and medial to iliopsoas, gives access to the pelvic brim quadrilateral plate
- Lateral window – Lies lateral to iliopsoas, gives access to the inner surface of the ilium

Can be extended proximally to expose the sacroiliac joint. By extending the incision posteriorly and stripping the iliacus off the ilium, the sacroiliac joint can be exposed. The approach cannot be extended distally.

Structures at risk

- **Femoral nerves and vessels**
- **Lateral cutaneous nerve of thigh** – As it passes close to ASIS
- **Inferior epigastric vessels**
- **Spermatic cord** – Can cause ischaemic damage to the cord. At risk with the medial window
- **Obturator artery and nerve** – At risk with the medial window
- **Urinary bladder**
- **Sacral nerve roots** – When exposing the sacroiliac joint

Stoppa approach

Stoppa initially described a midline approach for the treatment of complicated groin and incisional hernias. This approach has been modified to provide direct exposure to the low anterior column and quadrilateral plate fractures. It is not useful for fracture patterns with significant anterior displacement. Compared to the ilioinguinal approach, it is less invasive and does not require dissection of the inguinal canal, femoral vessels or femoral nerve.

Indications

Acetabular fractures especially medial wall
Pelvic ring fractures

Position

Supine. Injured lower limb draped free with hip and knee slightly flexed to relax the iliopsoas muscle and external iliac vascular bundle. Foley urinary catheter inserted to protect bladder, improve visualisation and monitor urine output. Radiolucent operating table.

Surgeon opposite affected side.

Landmarks

Symphysis pubis
ASIS

Incision

Pfannenstiel incision used. Transverse skin incision 1–2 cm above the pubic symphysis.

Internervous plane

No true internervous plane

Superficial surgical dissection

Dissection through skin and subcutaneous tissue to the underlying anterior rectus fascia

The rectus abdominis fascia is split vertically in line with its fibers and the transversalis fascia incised just superior to the pubic symphysis. Blunt dissection of the Retzius space is performed

Deep surgical dissection

A subperiosteal dissection is performed along the posterior surface of the pelvic brim into the quadrilateral surface and posterior column, allowing exposure of the quadrilateral surface and the medial aspect of the posterior column. The external iliac vessels and femoral nerve are protected placing a retractor under the iliopsoas, and the rectus muscle is retracted to the inner side.

The corona mortis a connecting vessel between the obturator and femoral artery, is identified if present and ligated to allow continued dissection further along the pelvic brim and quadrilateral surface.

Structures at risk

- **Obturator nerve and vessels**-direct contact with quadrilateral surface
- **External iliac vessels**
- **Corona mortis(10–30% cases)**
- **Bladder**

Posterior approach to the acetabulum (Kocher–Langenbeck)

Does not allow access to the anterior column.

Indications

- Fracture of posterior column and posterior lip of acetabulum
- Transverse fractures of acetabulum

Position

- Lateral – For posterior column/lip fractures
- Prone – If transverse fractures, to keep femoral head from migrating medially (which can occur if patient is in lateral position) during the operation and aid in reduction of the fracture. Can use distal femoral skeletal traction if needed

Landmarks

- Greater trochanter
- Posterior superior iliac spine
- Shaft of femur

Incision

Starts just below the iliac crest and lateral to the PSIS and goes anteriorly to the greater trochanter and then around it and distally around 10 cm below the greater trochanter along the femoral shaft.

Internervous plane

There is no true internervous plane.

Superficial surgical dissection

The fascia lata is in line with the wound. Split the gluteus maximus proximally along its anterior margin. This should expose the piriformis and short external rotators.

Deep surgical dissection

Detach the short external rotators. Piriformis leads to the greater sciatic notch and obturator internus leads to the lesser sciatic notch. Retractors are placed carefully in the greater and lesser sciatic notch. Capsulotomy of the hip joint may be required if still intact as this is usually torn in cases of trauma. This should expose the posterior lip of the acetabulum which can be fixed.

Posterior column exposure can be increased by an osteotomy of the greater trochanter. Visualisation of the acetabulum can be improved by skeletal traction on the femur. The anterior aspect of the acetabulum/hip joint capsule can be visualized by flexion and external rotation of the hip and mobilization of the insertion of gluteus minimus and the proximal portion of vastus intermedius from the femur.

Structures at risk

- **Sciatic nerve**
- **Inferior gluteal artery** – This leaves the pelvis just below piriformis. To control bleeding, apply pressure and will need to put patient in supine position. If transected, may retract into pelvis and vascular control is best achieved by interventional radiology
- **Superior gluteal artery and nerve**

Extended Iliofemoral approach to the acetabulum

This allows simultaneous access to both columns by exposing the external aspect of the innominate bone. It gives access to the entire lateral aspect of the iliac wing, entire retroacetabular surface and interior aspect of the hip joint. Routine use is rare as most complex fractures can be managed through combined Kocher–Lanenbeck and ilioinguinal approaches.

It is technically challenging and carries a high risk of complications. There is a large amount of soft-tissue stripping and a risk of devitalizing the gluteal muscles.

Indications

- Both column acetabular fractures with posterior comminution
- Certain T-shaped or transtectal transverse fractures with acetabular dome impaction or associated posterior wall fractures
- Several weeks old acetabular fractures – transverse, transverse plus posterior wall, T-shaped, associated anterior plus posterior hemitransverse, and both column fractures

Position

Lateral decubitus position with entire leg draped free

Landmarks

Iliac crest
ASIS

Incision

Inverted J-shaped incision

Begins PSIS and extends around the iliac crest to the ASIS. From here extend the distal end of the incision inferiorly by curving it down so that it runs vertically for 5 cm, heading toward the lateral side of the patella.

Internervous plane

The superficial plane lies between the sartorius (femoral nerve) and the tensor fasciae latae (superior gluteal nerve).

The deep plane lies between the rectus femoris (femoral nerve) and the gluteus medius (superior gluteal nerve

Superficial surgical dissection

Identify the fascia of the anterior thigh and incise it longitudinally on the lateral border of Sartorius. The gap between tensor fascia lata and sartorius is identified and developed. Retract the sartorius upward and medially and the tensor fascia lata downward and laterally. Dissection is continued between rectus femoris medially and gluteus medius laterally. The reflected head of rectus femoris tendon can be released from its origin on the ilium

Deep surgical dissection

The origins of the gluteal muscles are elevated in continuity from the external aspect of the iliac wing subperiosteally The elevation of the gluteal muscles from the iliac wing is continued in a posterior and distal direction until the greater sciatic notch is reached. The greater sciatic notch must be approached with care to avoid injury to the superior gluteal nerve or vessels.

Structures at risk

- Lateral femoral cutaneous nerve(lateral cutaneous nerve of the thigh)
- Femoral nerve
- Ascending branch of the lateral femoral circumflex artery
- Superior gluteal vessels

Knee

- Anteromedial approach (medial parapatellar)
- Medial approach
- Lateral approach
- Posterior approach

Anteromedial approach to the knee

- Workhorse approach of the knee

Indications

- Total knee replacement
- ORIF tibial plateau fractures
- Patellectomy
- Synovectomy
- Removal of loose bodies

Position

- Supine position
- Thigh tourniquet
- Skin prepared and draped
- Sandbag to allow unsupported flexion of the leg
- Lateral support

Landmarks

- Patella
- Tibial tubercle

Incision

A longitudinal straight midline skin incision is made, extending 5 cm above the superior pole of the patella to below the level of the tibial tubercle.

Internervous plane

There is no internervous plane in this approach as all the parts of the quadriceps muscle are supplied by the femoral nerve.

Superficial dissection

Develop a medial skin flap reflecting both subcutaneous tissue and superficial fascia. Medial parapatellar capsular incision cutting through the joint capsule and along the patellar ligament and quadriceps tendon to gain access to the joint. Leave a cuff of tissue medial to the patella and lateral to the quadriceps muscle to facilitate closure. Incise down the medial side of the patellar ligament.

Deep dissection

Displace the patella laterally and rotate it 180° and then flex the knee. If the patella does not dislocate, perform either a quadriceps turndown/snip or remove the patellar tendon attachment with a block of bone (tibial tubercle osteotomy).

Structures at risk

Division of the infrapatellar branch of the saphenous nerve may result in a painful neuroma. Avulsion of the patellar tendon from its insertion into the tibial tubercle is a very serious complication as it is difficult to reattach and ultimately compromises the outcome of surgery.

Extensile measures

Can be extended proximally between rectus femoris and vastus medialis, then splitting the fibres of intermedius to expose the anterior part of the femur, but only in the distal third to avoid damage to their nerve supply (femoral nerve).

In the tight knee during knee replacement or, more commonly, revision surgery it may be necessary to enhance the exposure and lessen the risk of avulsing the patellar tendon. The most common techniques described are:

- Quadriceps snip
- Tibial tubercle osteotomy
- Quadriceps turndown

Medial approach to the knee for medial meniscectomy

Although arthroscopic surgery has drastically reduced the need for this approach it still remains useful.

Indications

- Partial medial meniscectomy
- Removal of loose bodies
- Treatment of osteochondritis of the medial femoral condyle

Position

- Supine, sandbag under affected buttock, table removed, knee flexed 90° (the knee is at the end of the operation table, flexed 90°) with a thigh tourniquet, or
- Supine, thigh tourniquet, sandbag under affected thigh

Landmarks

- Medial joint line
- Inferomedial corner of the patella

Incision

Begin the incision from the inferomedial corner of the patella. Angle it inferiorly and posteriorly, ending 1 cm below the joint line. Incisions farther inferiorly may cut the infrapatellar branch of the saphenous nerve.

Internervous plane

There is no internervous plane in this approach because the deep incision is made through the medial patellar retinaculum and joint capsule.

Superficial surgical dissection

Incise down to the anteromedial aspect of the joint capsule. Incise the joint capsule in line with the incision, which is reinforced by the medial retinaculum of the patella.

Deep surgical dissection

Open the synovium well above the joint line to gain access to the anteromedial portion of the joint. Opening the joint above the joint line avoids damage to the intrasynovial fat pad and medial meniscus.

Structures at risk

- **Coronary ligament** (meniscotibial element of the deep medial ligament) with an incision made at the joint line
- **Superficial medial ligament** (tibial collateral ligament) if the incision is made too far medially
- **Fat pad**
- **Medial meniscus**

Medial approach to the knee

Indications

- Exploration and repair of medial collateral ligament (MCL) of the knee

Position

The patient is positioned supine with a tourniquet. The knee is flexed 60°, the hip is abducted and externally rotated, with the foot on the opposite shin.

Landmarks

- Adductor tubercle on the medial surface of the medial femoral condyle. It lies on the posterior part of the condyle in the distal end of the natural depression between vastus medialis and hamstring muscles

Incision

A long curved incision is made, beginning 2 cm proximal to the adductor tubercle of the femur. Curve the incision anteroinferiorly to a point 6 cm below the joint line on the anteromedial aspect of the tibia. The middle of this incision runs parallel to the medial border of the patella and 3 cm medial to it.

Internervous plane

There is no true internervous plane.

Superficial dissection

Raise skin flaps to expose the fascia of the knee. The infrapatellar branch of the saphenous nerve crosses the operative field transversely and is cut.

The saphenous nerve itself, which emerges between gracilis and sartorius, must be preserved.

Deep dissection

Exposing the deep structures within the knee involves incising the layers that cover them, either anterior to the superficial MCL or posterior to it. These separate incisions provide access to the anterior and posterior parts of the medial side of the joint.

Anterior approach

Expose the superficial medial ligament, the anterior part of the medial meniscus and the anterior cruciate ligament. Incise fascia along the anterior border of sartorius. The anterior border of sartorius can be hard to define at the level of the knee joint, so look for it either at its tibial insertion or at the proximal end of the wound.

Flex the knee further to allow the sartorius muscle to retract posteriorly, uncovering the two other components of the pes anserinus (semitendinosus, gracilis), which lie beneath and behind sartorius.

Retract all three muscles posteriorly to expose the tibial insertion of the superficial medial ligament, which lies deep and distal to the anterior edge of sartorius. The ligament inserts 6–7 cm below the joint line. Make a longitudinal medial parapatellar incision to gain access to the inside of the front of the knee joint, starting well above the joint line.

Posterior approach

Expose the posteromedial corner of the joint by separating the medial head of gastrocnemius muscle from the semimembranous muscle. Next, separate the medial head of gastrocnemius muscle from the posterior capsule of the knee joint almost to the midline by blunt dissection. Arthrotomy allows entry into the joint.

Structures at risk

- The **infrapatellar branch of the saphenous nerve** is sacrificed in this approach and should be buried in fat to prevent the formation of a postoperative neuroma
- **Saphenous nerve**
- **Medial inferior genicular artery**
- **Popliteal artery**

Extensile measures

The incision cannot be extended usefully in either direction.

Lateral approach to the knee

Provides access to all the supporting structures on the lateral side of the knee.

Indications

- Fracture of the lateral tibial plateau

Position

The patient is positioned supine on the operating table, with a tourniquet and the knee flexed at 90°. A sandbag is placed under the buttock on the affected side.

Landmarks

- Lateral border of the patella
- Gerdy's tubercle (the lateral tubercle of the tibia)
- Lateral joint line

Incision

A long curved incision is made, starting at the level of the middle of the patella, 3 cm lateral to it, extending downwards over Gerdy's tubercle to 5 cm distal to the joint line. Complete the incision by curving its upper end to follow the line of the femur.

Internervous plane

The internervous plane lies between the iliotibial band (superior gluteal nerve) and the biceps femoris muscle (sciatic nerve).

Superficial dissection

Mobilize skin flaps widely. Underneath are two main structures: The iliotibial band and the biceps femoris muscle. Incise the fascia in the interval between the iliotibial band and biceps femoris muscle and dissect between them. Retract the iliotibial band anteriorly and biceps femoris muscle posteriorly and uncover the superficial lateral ligament (fibular collateral ligament) as it runs from the lateral epicondyle of the femur to the head of the fibula.

Deep dissection

The knee joint is entered either in front of or behind the superficial lateral ligament.

Anterolateral arthrotomy

Incise the capsule in front of the ligament. Watch the lateral meniscus; begin the arthrotomy 2 cm above the knee joint.

Posterolateral arthrotomy

Dissect between the lateral head of gastrocnemius and the posterolateral corner of the knee. Branches of the lateral superior genicular artery must be coagulated in this area.

Structures at risk

- **Common peroneal nerve**
- **Lateral superior genicular artery**
- **Popliteus tendon**

Extensile approach

The exposure cannot be extended usefully.

Posterior approach to the knee

The posterior approach to the knee is a primarily neurovascular approach; orthopaedically it is used only very rarely (Figure 32.14).

Indications

- Repair neurovascular structures that run behind the knee
- Repair avulsion fractures of the PCL
- Soft-tissue release for knee contracture
- Excision of Baker's cyst
- ORIF significant posterior fragment tibial plateau fracture (combined with anterior approach if necessary)

Position

- Prone. Tourniquet with exsanguination

Landmarks

- The two heads of the gastrocnemius muscle originate from the posterior femoral surface just above the medial and lateral condyles
- Semimembranous and semitendinosus on the medial border of the popliteal fossa and biceps femoris along the lateral border

Incision

- Curvilinear incision centred over the popliteal fossa

A gentle curved incision is made, starting laterally over the biceps femoris bringing the incision obliquely across the popliteal fossa and then down over the medial head of gastrocnemius and into the calf.

Internervous plane

There is no true internervous plane as the boundaries of the popliteal fossa are separated to expose its contents.

Superficial surgical dissection

Reflect skin flaps with the underlying subcutaneous fat.

Identify the small saphenous vein as it passes upwards in the midline of the calf. On the lateral side identify the medial sural cutaneous nerve. Incise the fascia of the popliteal fossa just medial to the small saphenous vein. Trace the medial sural cutaneous nerve back up to the tibial nerve at the apex of the fossa. The apex of the popliteal fossa is formed by the semimembranosus muscle on the medial side and the biceps femoris muscle on the lateral side.

Roughly at the apex of the fossa the common peroneal nerve separates from the tibial nerve. The popliteal artery and vein lie deep and medial to the tibial nerve. The vein is medial to the artery as it enters the fossa from below. It then curves, lying directly posterior to the artery in the fossa whilst above the knee it moves to the posterolateral side of the artery.

Deep surgical dissection

Deep surgical dissection involves retracting the muscles that form the boundaries of the popliteal fossa to expose the posterior joint capsule of the knee.

The medial and lateral heads of the gastrocnemius can be detached close to its origin and turned inferiorly to expose the respective corner.

Structures at risk

- **Medial sural cutaneous nerve**, which lies lateral to the small saphenous vein
- **Tibial nerve**

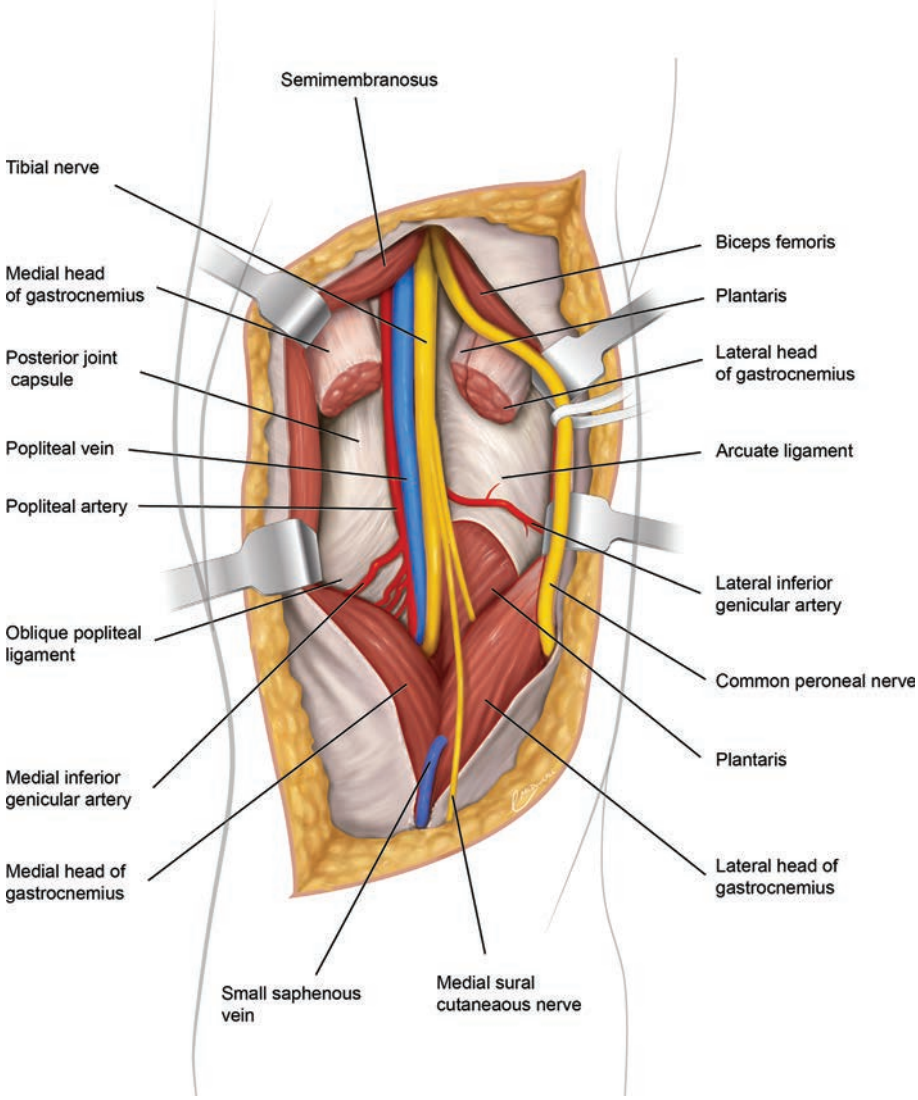

Figure 32.14 Deep dissection of popliteal fossa. Both the medial and the lateral gastrocnemius heads are detached, but one can detach the origin of the lateral head to expose the posterolateral corner of the joint capsule or the medial head to expose the posteromedial side

- **Common peroneal nerve**
- **Popliteal vessels**

Extensile measures

The exposure cannot be extended beyond the bounds of the popliteal fossa but may enable exposure of the trifurcation of the popliteal artery distally.

Hamstring tendon harvest (Figure 32.15)

Semitendinosus, gracilis and sartorius, have a common insertion into the anterior-medial aspect of the tibia, the pes anserinus. These muscles act as flexors of the knee but also provide tibial rotation and act as rotatory and valgus constraints to the knee. Although gracilis and semitendinosus are separate structures proximally, they converge prior to their insertion onto the tibia. The insertion of gracilis is superior to that of semitendinosus. The pes anserinus insertion is about 2 cm distal and 2.5 cm medial to the apex of the tibial tuberosity.

Hamstring tendon graft is harvested through a 3–5 cm paramedian incision centered at the level of the tibial tuberosity.

The sartorius fascia is exposed, and the tendons of gracilis and semitendinosus are palpated underneath it before they converge into the pes anserinus. The sartorial fascia is incised and reflected so as to expose the underlying tendons. The tendons are more horizontal than vertical as they insert in an oblique fashion. The gracilis tendon insertion is more superior of the two. Both the tendons are mobilized by blunt and sharp dissection keeping its insertion intact till the end. All tendinous slips (vinculae) are freed. It is important to release these attachments before using a tendon stripper or else the tendon gets inadvertently cut as this level. This is more consistent especially in the semitendinosus where one or more large vinculae attach it to the medial head of the gastrocnemius. Once harvested, the tendons are detached from their insertion and stripped off their muscle fibres before placing a whipstitch as necessary.

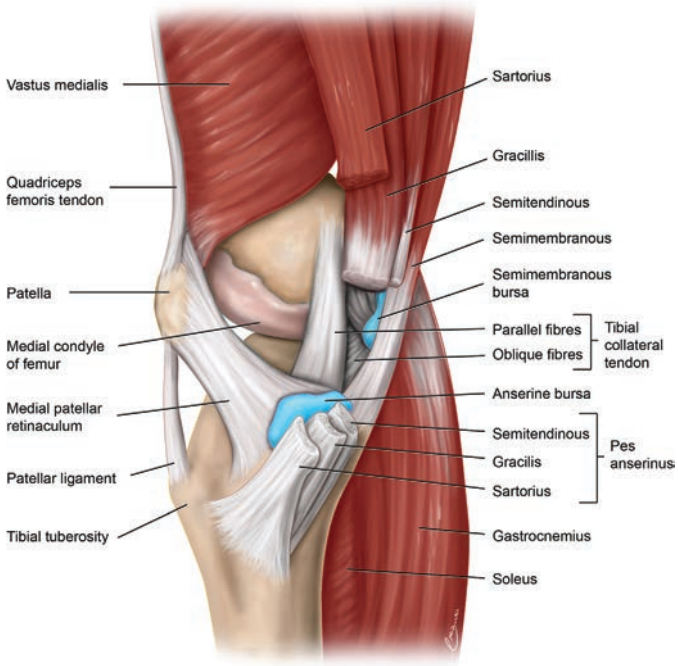

Figure 32.15 Hamstring tendon harvest. Semitendinosus, gracilis and sartorius, have a common insertion into the anterior-medial aspect of the tibia, the pes anserinus

Tibia and fibula

- Anterior approach to the tibia
- Anterolateral approach to the tibia
- Posterolateral approach to the tibia

Anterior approach to the tibia

The anterior approach offers safe and easy access to the medial (subcutaneous) and lateral (extensor) surfaces of the tibia.

Indications

- ORIF of tibial fractures
- Plating and bone grafting non-union
- Osteotomy of the tibia

Position

- Supine, tourniquet, leg marked

Landmarks

- Shaft of tibia

Incision

A longitudinal skin incision is made 1 cm lateral and parallel to the anterior border of the tibia.

Internervous plane

There is no internervous plane in this approach.

Superficial surgical dissection

Elevate skin flaps to expose the subcutaneous surface of the tibia.

Deep surgical dissection

Through this incision one can expose either the subcutaneous (medial) surface or lateral (extensor surface) of the tibia. Exposing the lateral surface of the tibia involves reflecting the tibialis anterior muscle subperiosteally off the tibia in a lateral direction. Strip the periosteum as little as possible because its circulation is a source of nutrition for the bone.

Structures at risk

- **Long saphenous vein**

Anterolateral approach to the tibia

The anterolaeral approach is used to expose the middle two thirds of the tibia when the skin over the subcutaneous surface of the tibia is of poor quality. Although the exposure is technically simple it provides only limited exposure of the tibia and is generally unsuitable for ORIF.

Indication

- Delayed and non-union of the tibia

Position

- Lateral position with affected side uppermost
- Protect bony prominences of the lower legs

Landmarks

- Subcutaneous surface of the fibula
- Head of fibula

Incision

A longitudinal incision is made centred over the shaft of the fibula at the level of the tibial pathology.

Internervous plane

Superficially, the internervous plane lies between peroneus brevis muscle (superficial peroneal nerve) and extensor digitorum longus (EDL) muscle (deep peroneal nerve). Deeply, the plane lies between the tibialis posterior muscle (tibial nerve) and the extensor muscles of the ankle and foot (deep peroneal nerve). These muscles are separated by the interosseous membrane.

Superficial surgical dissection

Deepen the incision taking care not to damage the short saphenous vein that may appear in the posterior aspect of the wound. Develop a plane between the anterior aspect of the peroneus brevis muscle and the EDL muscle to come down onto the anterolateral aspect of the fibula. Protect the superficial peroneal nerve which can be seen lying on the peroneal brevis muscle.

Deep surgical dissection

Gently detach the extensor muscles from the anterior aspect of the interosseous membrane. Follow the anterior aspect of this membrane onto the lateral border of the tibia. Make sure to stay firmly on the interosseous membrane, straying anteriorly may cause damage to the anterior neurovascular bundleExpose the posterolateral corner of the tibia.

Structures at risk

- **Small saphenous vein** – May be damaged in the posterior skin flap
- **Superficial peroneal nerve** – Runs down the leg in the lateral compartment
- **Anterior tibial artery and deep peroneal nerve** – Runs down the leg in the anterior compartment which is anterior to the interosseous membrane

Posterolateral approach to the tibia

This approach is used to expose the posterior middle two-thirds of the tibia when the skin over the subcutaneous surface is scarred or infected.

Indications

- ORIF of tibial fractures
- Treatment tibia non-union

Position

The patient is positioned in the lateral decubitus. Protect the bony prominences on the bottom leg. The affected leg is prepared and draped free. Tourniquet. Alternatively, the patient can be prone.

Landmarks

- Lateral border gastrocnemius.

Incision

A longitudinal straight line incision is made over the lateral border of gastrocnemius. The incision lies behind the shaft of the fibula in the interval between the peroneal tendons anteriorly and the gastrocnemius-soleus posteriorly. The length of incision depends on the length of bone that must be exposed.

Internervous plane

The internervous plane lies between the gastrocnemius, soleus and flexor hallucis longus (FHL) (tibial nerve) and the peroneal muscles (superficial peroneal nerve) between the posterior and lateral muscular compartments.

Superficial surgical dissection

Reflect the skin flaps. Incise the deep fascia in line with the skin incision. Find the plane between the lateral head of gastrocnemius and soleus muscle posteriorly and the peroneus brevis and longus muscles anteriorly. This interval is opened down to the fibula.

Deep surgical dissection

The lower part of the origin of soleus from the fibula is detached and retracted posteriorly. Detach the FHL from its origin on the fibula and retract it posteriorly and medially. The posterior surface of the shaft of the fibula is now exposed. By following the interosseous membrane to the lateral border of the tibia, the origin of tibialis posterior from the interosseous membrane can be detached and retracted posteriorly to expose the posterior surface of the tibia.

Structures at risk

The **short saphenous vein** may be damaged when mobilizing the skin flaps. The posterior tibial artery and nerve are safe as long as you stay on the interosseous membrane and do not wander into a plane posterior to FHL and tibialis posterior.

Foot and ankle

- Anterior approach to the ankle
- Approach to the medial malleolus
- Approach to the lateral malleolus
- Posteromedial approach to the ankle
- Lateral approach to the foot
- Lateral approach to the tarsus

Anteriolateral approach to the ankle

This approach has been popularized for management of distal tibial pilon fractures. It offers the benefit of improved soft-tissue coverage and a lower ate of wound healing complications by avoiding incision placement over the subcutaneous border of the tibia.

Indications

- ORIF pilon fractures of the tibia

Position

- Supine on the operating table
- Tourniquet with exsanguinations

Landmarks

- Level of the ankle joint
- Medial malleolus
- Lateral malleolus

Incision

A skin incision centered at the ankle joint parallels the fourth metatarsal distally and runs between the tibia and fibula proximally. The incision is usually not extended above >7 cm above the ankle joint as the origin of the anterior compartment muscle bellies are encountered.

Internervous plane

No true internervous plane is present, but a very clear intermuscular plane exists between extensor hallucis longus (EHL) and EDL.

Superficial dissection

Full thickness skin flaps should be maintained. The superficial nerve should be mobilized and retracted. The fascia over the anterior compartment of the distal tibia and extensor retinaculum are sharply incised.

Deep surgical dissection

The anterior compartment tendons are retracted medially. The extensor digitorum brevis fascia can be incised and the muscle retracted medially.

Structures at risk

The **superficial peroneal nerve** is at risk of injury because it lies directly beneath the skin and crosses the surgical approach at the ankle. It must be identified, protected and retracted during the operation.

Anterior approach to the ankle

Indications

- Remove loose bodies
- ORIF pilon fractures of the tibia
- Ankle fusion

Position

- Supine on the operating table
- Tourniquet with exsanguinations

Landmarks

- Level of the ankle joint
- Medial malleolus
- Lateral malleolus

Incision

A 15-cm longitudinal incision is made over the anterior aspect of the ankle, beginning 10 cm above the joint, crossing the joint midway between the malleoli, ending on the dorsum of the foot. Identify and protect the superficial peroneal nerve.

Internervous plane

No true internervous plane is present, but a very clear intermuscular plane exists between EHL and EDL. The plane must be used with great care because it contains the neurovascular bundle distal to the ankle.

Superficial dissection

Reflect the skin edges. Identify and split the extensor retinaculum in line with the skin incision. Identify the plane between EHL and EDL a few centimetres above the ankle joint. Medial to the EHL is the neurovascular bundle (anterior tibial artery and deep peroneal nerve). Isolate, ligate and divide the anterolateral, malleolar and lateral tarsal arteries to allow mobilization of the neurovascular bundle. The tendon of EHL and the neurovascular bundle are retracted medially and the tendon of EDL is retracted laterally.

Deep surgical dissection

Incise the remaining soft tissues longitudinally to expose the anterior surface of the distal tibia and anterior capsule of the ankle joint. Incise the joint capsule longitudinally in line with the skin incision as required.

This approach can if necessary be extended proximally to expose structures in the anterior compartment. Distal extension to the dorsum of the foot is possible but is very rarely required.

Alternatively, the incision may be curved medially at its distal extent and the tibialis anterior retracted laterally to expose the distal tibia.

Structures at risk

Cutaneous branches of the **superficial peroneal nerve** run close to the line of the skin incision. **Anterior tibial artery** and **deep peroneal nerve** are at risk with superficial surgical dissection.

Above the ankle, the neurovascular bundle lies between tibialis anterior and EHL whilst below the ankle joint the neurovascular bundle comes to lie between EHL and EDL. The EHL tendon crosses over the neurovascular bundle in a lateral to medial direction at the level of the ankle joint.

Structures anterior to the ankle

Tom **H**as **A** **N**asty **D**irty **P**...
Tibialis anterior
Extensor **H**allucis
Artery
Nerve
Extensor **d**igitorum
Peroneus tertius

The evertor tendons – Peroneus longus and peroneus brevis (superficial peroneal nerve) pass behind the lateral malleolus.

There is a classic story a few years ago of a candidate who was shown various colour pictures of the anatomy of the front of the ankle and asked to name various structures. These colour pictures were being quickly flashed in the direction of the candidate from a laptop computer by an eminent professor of orthopaedic surgery. The EHL and EDL were labelled the wrong way round. There was much debate when the candidate pointed this fact out. The professor eventually conceded this point to the candidate. Point one is to know your anatomy well and point two is to stick to your guns if you definitely know you are correct.

Approach to the medial malleolus

Both anterior and posterior approaches to the medial malleolus can be used.

Indications

- ORIF fractures of the medial malleolus

Position

- Supine on the operating table

Incision

The anterior approach consists of a longitudinal curved incision on the medial aspect of the ankle with its midpoint just anterior to the tip of the medial malleolus. The incision begins 5 cm proximal to the medial malleolus and then curves forwards to end anteriorly and distal to the malleolus. The incision should not cross the most prominent portion of the malleolus.

The posterior approach involves a 10-cm incision on the medial aspect of the ankle, beginning 5 cm above the ankle on the posterior border of the tibia, curving the incision downwards following the posterior border of the medial malleolus. The incision is curved forwards below the medial malleolus to end 5 cm distal to it.

Internervous plane

No true internervous plane exists but the approach is safe because the incision cuts down onto subcutaneous bone.

Superficial surgical dissection

Skin flaps are mobilized. Identify and preserve the saphenous nerve and long saphenous vein, which lie anterior to the medial malleolus.

Deep surgical dissection

The periosteum of the medial malleolus is incised longitudinally. With an anterior approach a small incision is made in the anterior capsule of the ankle joint so that the joint surfaces can be visualized.

With a posterior approach the retinaculum behind the medial malleolus is incised. The tibialis posterior (TP) tendon is retracted anteriorly whilst the remaining structures are freed up and retracted posteriorly.

Structures at risk

Anteriorly the **saphenous nerve,** which, if cut, may form a painful neuroma and cause numbness over the medial side of the dorsum of the foot.

Posteriorly all structures that run behind the medial malleolus (**TP, FDL posterior tibial artery and vein, tibial nerve and FHL**) are at risk.

Anatomy of the medial side of the ankle

The posterior neurovascular bundle runs behind the medial malleolus between the tendons of FDL and FHL.

The posterior tibial artery passes behind FDL before entering the sole of the foot where it divides into medial and lateral plantar arteries.

Tall **D**octors **A**re **N**ever **H**appy
Tibialis Posterior
Flexor **D**igitorum Longus
Artery
Nerve
Flexor **H**allucis longus

Approach to the lateral malleolus

Indications

- ORIF of lateral malleoli fractures

Position

The patient is positioned supine on the operating table, with a sandbag under the buttock. Tourniquet with exsanguination.

Landmarks

The subcutaneous surface of the fibula and lateral malleolus are palpated. The short saphenous vein runs along the posterior border of the lateral malleolus.

Incision

A longitudinal incision is made along the posterior margin of the fibula all the way to its distal end.

Internervous plane

There is no internervous plane as the dissection is being performed down to a subcutaneous bone.

Superficial surgical dissection

Elevate the skin flaps. Take care not to damage the short saphenous vein, which lies posterior to the lateral malleolus. The sural nerve, which runs with the short saphenous vein, should also be preserved.

Deep surgical dissection

Dissection is performed down to the subcutaneous surface of the bone. The periosteum of the fibula is incised longitudinally.

Posteromedial approach to the ankle

Position

- Supine. Knee flexed, hip externally rotated and the ankle laid on the shin of the other leg (the 'figure-of-4' position). Tourniquet, or
- Lateral with the affected ankle closer to the table and the opposite knee flexed so as to get the contralateral ankle out of the way

Landmarks

- Medial malleolus

Incision

A 10-cm longitudinal incision is made midway between the medial malleolus and the Achilles tendon.

Superficial surgical dissection

Deepen the incision in line with the skin incision to enter the fat surrounding the Achilles tendon.

Deep surgical dissection

Retract the Achilles tendon and retrotendinous fat laterally, exposing the fascia of the deep flexor compartment. The

compartment is opened and the FHL identified. Next the posterior tibial artery and tibial nerve are mobilized. A plane is developed between the neurovascular bundle with the FHL laterally and the tendon of FDL medially. The joint capsule on the posterior aspect of the ankle joint is incised longitudinally.

Structures at risk

All the structures that run behind the medial malleolus (**TP, FDL, posterior tibial artery and vein, tibial nerve and FHL**) are at risk.

Posterolateral approach to the ankle

Indications

- ORIF of the posterior malleolus

Position

- Lateral or prone
- Ankle elevated off the table with a foam block (if lateral)
- Tourniquet

Landmarks

- Fibula and the lateral border of tendo-Achilles

Incision

A 10-cm longitudinal incision is made midway between the fibula and the Achilles tendon.

Internervous plane

The internervous plane lies between the peroneus brevis muscle (superficial peroneal nerve), and the flexor hallucis longus (tibial nerve).

Superficial surgical dissection

The short saphenous vein and sural nerve lie superficial to the deep fascia and should be identified and preserved if possible. The deep fascia is incised in line with the skin incision. Identify the peroneal tendons (brevis is muscular down to the ankle joint) and retract them anteriorly.

Deep surgical dissection

To expose the posterior border of the fibula, strip some of the peroneus brevis muscle off the fibula in an anterior direction and elevate the fibres of FHL in a posterior direction. To expose the tibia, continue the subperiosteal dissection from the posterior border of the fibula across the interosseous membrane and elevate the periosteum of the distal tibia. The dissection can be extended proximally (as for the posterolateral approach to the tibia). This approach provides excellent exposure to reduce and stabilise the posterior malleolus with a buttress plate; the posterior border of the fibula can also be plated.

Structures at risk

- Sural nerve
- Short saphenous vein
- **Posterior tibial neurovascular bundle** (if straying too far medial on the tibia)

Lateral approach to the foot

This approach gives good access to the os calcis, peroneii and the lateral ligaments of the ankle.

Indications

- ORIF of calcaneal fractures.

Position

The patient is positioned supine with a sandbag under the buttock of the affected side. A tourniquet is applied. Tilt the table 20–30° away from the surgeon to improve access.

Landmarks

- Lateral malleolus
- Peroneal tubercle

Incision

A curved incision ~12 cm long is made on the lateral aspect of the ankle. The incision begins 4 cm above the tip of the lateral malleolus on the posterior border of the fibula. The posterior border is followed down to the tip of the lateral malleolus and then the incision is curved in a hockey stick fashion forwards, passing over the peroneal tubercle parallel to the course of the peroneal tendons. The incision is carried straight down onto bone, especially at the angle as it overlies the calcaneus.

Internervous plane

No internervous approach exists in this approach.

Superficial surgical dissection

Mobilize full-thickness skin flaps minimally. Take care not to damage the sural nerve as it runs behind the lateral malleolus with the short saphenous vein. Incise and open the deep fascia in line with the skin incision to uncover the two peroneal tendons. Continue the fascial incision distally, following the course of the two tendons. The inferior peroneal retinaculum is incised and the peroneal tendons are exposed more fully and mobilized.

Deep surgical dissection

The peroneal tendons are mobilized and retracted anteriorly over the distal end of the fibula. The calcaneofibular ligament is identified and incised transversely to open the capsule of the posterior talocalcaneal joint.

To expose the bare lateral surface of the calcaneum, incise the periosteum over its lateral surface and strip it inferiorly by sharp dissection.

Structures at risk

- **Sural nerve**

Lateral approach to the hindpart of the foot (Ollier's approach)

This approach is excellent for a triple arthrodesis. The three joints are exposed through a small opening without much retraction, and the wound usually heals well because the proximal flap is dissected full thickness and the skin edges are protected during retraction.

Indications

- Triple arthrodesis

Position

The patient is supine on the operating table with a large sandbag beneath the affected buttock. Tilt the table 20–30° away from the surgeon to improve access further. Tourniquet after exsanguination.

Landmarks

- Lateral malleolus
- Lateral wall of calcaneus
- Sinus tarsi

Incision

An oblique incision ~10 cm long is made, starting below the lateral malleolus extending across to the talonavicular joint. The incision begins just distal to the distal end of the lateral malleolus and slightly posterior to it. Continue distally along the lateral side of the hindpart of the foot, over the sinus tarsi, then medially toward the talocalcaneonavicular joint.

Internervous plane

The internervous plane lies between the peroneus tertius tendon (deep peroneal nerve) and the peroneal tendons (superficial peroneal nerve).

Superficial surgical dissection

Do not mobilize skin flaps widely. Divide the inferior extensor retinaculum in the line of the skin incision. At the distal end of the incision, retract the tendons of peroneus tertius and EDL medially, preferably without opening their sheaths. At the proximal end of the wound, expose the peroneal tendons.

Deep surgical dissection

Partially detach the fat pad that lies in the sinus tarsi by sharp dissection.

Under it lies the origin of the extensor digitorum brevis muscle. Detach this origin by sharp dissection and reflect the muscle distally to expose the dorsal capsule of the talocalcaneonavicular joint and more laterally the dorsal capsule of the calcaneocuboid joint. Incise the respective capsules and open up the joints by inverting the foot.

The peroneal retinacula can be incised and the peroneal tendons reflected anteriorly to expose the posterior talocalcaneal joint.

Structures at risk

- **Skin flap**

Cervical spine

- Anterior approach to the cervical spine
- Posterior approach to the cervical spine

Anterior approach to the cervical spine (Figure 32.16)

Indications

- Excision of herniated nucleus pulposus (HNP)
- Interbody fusion
- Excision tumours
- Drainage abscesses
- Treatment of osteomyelitis
- Excision of osteophytes

Landmarks

- Anterior border of sternocleidomastoid
- Low border of mandible C2–C3
- Hyoid bone C3
- Thyroid cartilage C4–5
- Cricoid cartilage C6

Position

The patient is positioned supine on the operating table, with a sandbag between the shoulder blades, head turned away from the planned incision and the table is elevated 30° to reduce bleeding.

Skin incision

This is made at the level where the pathology is localized. A transverse collar incision along the skin crease is made at the appropriate level of vertebral pathology from the midline to the posterior border of sternocleidomastoid muscle. A longitudinal incision along the anterior border of sternocleidomastoid is used if more than three vertebrae have to be visualized.

Superficial surgical dissection

Incise the fascial sheath over platysma in line with the skin incision. The platysma is split longitudinally parallel to its long fibres. Identify and, if necessary, ligate the external jugular vein. The key to the approach is to identify the anterior border of sternocleidomastoid. Longitudinally incise the fascia immediately anterior to it. The sternocleidomastoid muscle is retracted laterally. The sternohyoid and sternothyroid strap muscles along with the trachea and oesophagus are retracted medially so as to expose the carotid sheath. Incise the pretracheal fascia immediately medial to the carotid sheath. Develop a plane by gentle blunt dissection between the medial edge of the carotid sheath and the midline structures. Retract the carotid sheath laterally. Two arteries connect the carotid sheath to the midline structures namely the superior and inferior thyroid arteries and they may ligated and cut for better exposure.

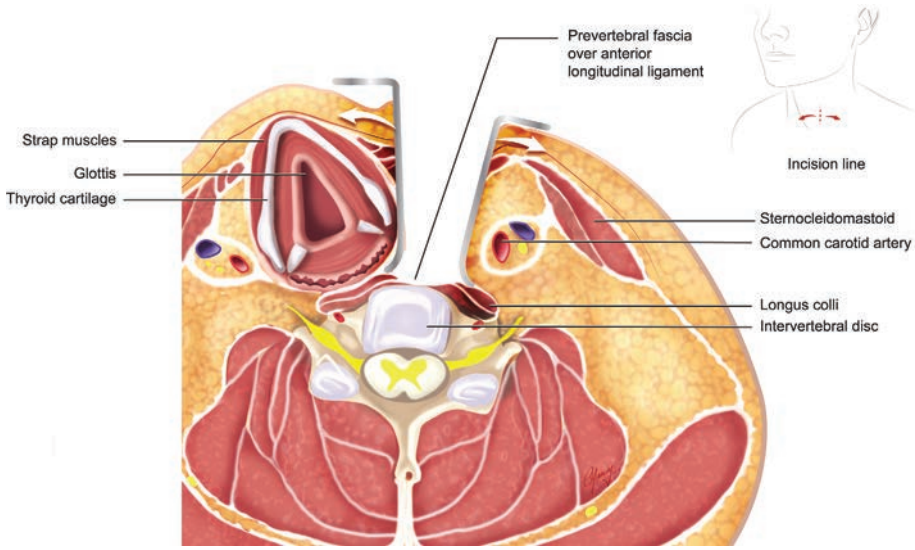

Figure 32.16 Anterior approach to cervical spine. The sternocleidomastoid and carotid sheath are retracted laterally and the strap muscles, trachea and oesophagus medially. The longus coli and pretrachial fascia are exposed

Deep surgical dissection

Develop a plane deep to the medial structures under the oesophagus, which is retracted from the midline. Split the prevertebral fascia along with the anterior longitudinal ligament and subperiosteally reflect this laterally along with the longus colli muscles, to expose the anterior surface of the vertebral bodies. Place the retractors underneath each of the longus colli muscles as the sympathetic chain lies on the longus colli muscle just lateral to the vertebral bodies.

Structures at risk

- Cervical sympathetic chain
- **Recurrent laryngeal nerve (0.2%)**, more common with right-sided approach
- Perforation of the **oesophagus**

Left- vs right-sided approach?

The right recurrent laryngeal nerve arises from the vagus as the latter crosses the first part of the subclavian artery. It hooks backwards and upwards behind the artery before ascending into the neck alongside the trachea and oesophagus behind the common carotid artery.

On the left side, the recurrent laryngeal nerve arises from the vagus as the latter crosses the arch of the aorta. It hooks around beneath the arch and then ascends back into the neck, running between the trachea and oesophagus to supply the larynx.

The thoracic duct lies anterior to the intercostal branches of the aorta. It arches across the dome of the left pleura to enter the left brachiocephalic vein.

There is much debate about whether this approach should be made from the right or left side.

A left-sided incision is generally preferred because of the more constant anatomy of the recurrent laryngeal nerve and less risk of inadvertent injury to the nerve.

On rare occasions the right recurrent laryngeal nerve can be aberrant, arising at a higher level than normal and crossing the operative field at the level of the thyroid gland. Others would advocate the right side to avoid the thoracic duct.

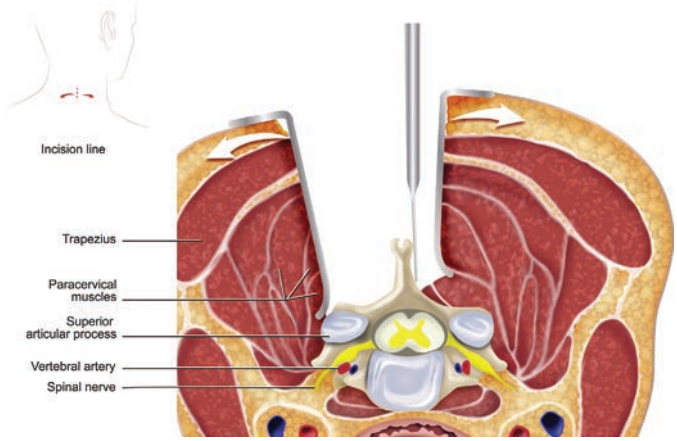

Figure 32.17 Posterior approach to cervical spine

Posterior approach to the cervical spine (Figure 32.17)

Indications

- Posterior spinal fusion
- Excision of HNP
- Treatment of tumours
- Treatment of facet joint dislocations
- Nerve root exploration

Position

The patient is placed in the prone position, with a padded ring and the head in a few degrees of flexion (to open the interspinous spaces). A neurosurgical head fixation device is used and skull traction may be necessary if the neck is unstable. The proposed surgical area is shaved to allow adequate skin preparation. Drapes are sewn into place. Eye padding is used and

head end away from anaesthetist with long endotracheal tubing.

Landmarks

- External occipital protruberance
- Spinous processes of the cervical spine

Incision

- Generous straight midline neck incision through skin and subcutaneous fascia

Internervous plane

The midline plane is between paracervical muscles supplied by branches of the left and right posterior primary rami of the proximal cervical nerve roots.

The plane is internervous and extensile.

Superficial surgical dissection

The wound is deepened in line with the skin incision down to the spinous processes by incising through fascia and the nuchal ligament. Cobb elevators are used to clean the tips of the spinous processes, and then the elevators are reversed and swept laterally to clear muscle attachments from the lamina (paraspinal muscles).

Using deep self-retaining retractors, carry dissection as far laterally as necessary to expose the lamina, facet joints and beginnings of the transverse processes.

Deep surgical dissection

Identify the ligamentum flavum that runs between the laminae.

Using a sharp blade or Cobb elevator remove the ligamentum flavum from the leading edge of the lamina of the inferior vertebrae. Place a flat-shaped spatula in the midline between the ligamentum flavum and underlying dura. Incise down onto the ligamentum flavum. Perform a laminectomy (partial or complete), removing as much of the lamina as necessary to see the blue-white dura that lies immediately below it covered by epidural fat.

Gently retract the spinal cord medially. Occasionally, thin epidural veins surrounding the cord may bleed significantly.

Structures at risk

- **Spinal cord** and **nerve roots**
- **Venous plexus in the cervical canal**
- **Vertebral artery**

Anatomy of the paracervical muscles

The paracervical muscles in the cervical spine run in three layers. The most superficial layer consists of the trapezius muscle. The intermediate layer consists of splenius capitis. The deep layer is subdivided into three portions: Superficial, middle and deep.

The superficial portion consists of semispinalis capitis. The middle portion consists of semispinalis cervicis and the deep layer consists of the multifidus muscles and the short and long rotator muscles.

Lumbar spine

- Anterior approach
- Posterior approach

Anterior approach to the lumbar spine

Position

- Supine on operating table
- Urinary catheter
- Nasogastric tube

Landmarks

- Umbilicus
- Pubic symphysis

Incision

A longitudinal midline incision is made from just below the umbilicus to just above the pubic symphysis.

The incision is extended superiorly, curving it just to the left of the umbilicus.

Internervous plane

The midline plane lies between the abdominal muscles on each side, segmentally supplied by branches from the 7th to the 12th intercostal nerves.

Superficial surgical dissection

The wound is deepened in line with the skin incision, cutting through fat to reach the fibrous rectus sheath. The sheath is incised longitudinally. The two rectus abdominis muscles are separated using finger dissection to expose the peritoneum. The peritoneum is picked up carefully between two pairs of forceps and, after making sure that no viscera have been trapped underneath it, it is incised open. The incision is extended proximally and distally, with one hand inside the abdominal cavity to protect the viscera.

Deep surgical dissection

Insert a self-retaining retractor to retract the rectus abdominis muscles away laterally. Carefully pack the bowel away, keeping it inside the abdominal cavity. Incise the posterior peritoneum in the midline over the sacral promontory. The middle sacral artery runs distally in the midline and should be tied off. Preserve any nerve fibres in this layer (presacral plexus) if possible. The L5–S1 disc space is accessible below the bifurcation of the aorta and the inferior vena cava (IVC). To access the L4/5 disc space requires mobilization of the great vessels. This is performed by incising the peritoneum on the left side of the aorta, and mobilizing the colon and left ureter. The left fourth and fifth lumbar vessels must be identified and ligated before mobilizing the aorta, inferior vena cava (IVC) and left common iliac vein to the right.

Structures at risk

- **Middle sacral artery**
- **Aorta**
- **Inferior vena cava**
- **Presacral plexus of parasympathetic nerves** (important for sexual function)
- **Ureter**
- **Left lumbar vessels**

Posterior approach to the lumbar spine

Indications

- Excision of HNP
- Exploration of nerve roots
- Spinal fusion
- Removal of tumours

Position

- Kneeling position with flexion hips and knees to flex spine and open up interspinous spaces (90/90 position), or
- Prone on cushions, or
- Lateral with affected side uppermost
- It is important to reduce venous plexus filling around the spinal cord by permitting the venous plexus to drain directly into the inferior vena cava

Landmarks

- Iliac crest (L4 spinous process)
- Radiographs with needle at the correct disc level
- Spinous processes

Incision

A midline longitudinal incision is made over the spinous processes. The length of the incision depends on the number of levels to be explored. A line drawn across the highest points of the iliac crests (crest of the ilium) intersects the L4 spinous process.

A line drawn between the two posterior superior iliac spines crosses the second part of the sacrum.

Superficial surgical dissection

Deepen the incision through fat and fascia using cutting diathermy in line with the skin incision until the spinous processes themselves are reached. Incise through the lumbodorsal fascia. The paraspinal muscles are detached subperiosteally as one unit from bone using Cobb elevators as far as the facet joints. Continue dissecting laterally. Branches of the lumbar vessels bleed during stripping of the muscles. If the transverse processes must be reached, continue dissecting down the lateral side of the ascending facet and onto the transverse process itself.

Deep surgical dissection

Use a deep self-retaining retractor used to improve exposure. Excise the ligamentum flavum on the leading edge of the inferior lamina using a pituitary rongeur.

The ligament flavum consists of yellow elastic tissue. The ligament takes origin from the leading edge of the lower lamina and inserts into the anterior surface of the lamina above. It is best removed from the leading edge of the lower lamina through sharp dissection or curettage. Once the ligamentum flavum is entered, a thin spatula should be placed beneath it to protect the underlying dura from being torn. Immediately beneath the epidural fat is the blue-white colour of the dura.

The approach can be extended proximally or distally, detaching the posterior spinal musculature from the posterior spinal elements as required. To gain better exposure locally to visualize the dura, nerve root or disc better, additional portions of the lamina can be removed. Part of the facet joint can be removed if required.

Structures at risk

Each **nerve root** must be identified and protected. The **venous plexus** surrounding the nerves and the floor of the vertebra may bleed during the blunt dissection needed to reach the disc. The **iliac vessels** lying on the anterior aspect of the vertebral bodies may be injured if instruments pass through the anterior portion of the annulus fibrosus.

Anatomy of the paraspinal muscles

The muscles of the lumbar spine are made up of superficial and deep layers. The superficial layer consists of latissimus dorsi. The deep layer consists of the paraspinal muscles, which are divided into two layers. The superficial portion consists of the sacrospinalis muscle (erector spinae) and the deep portion consists of multifidus and rotator muscles. The dorsal lumbar fascia is a broad, relatively thick, white sheet of tissue that forms a sheath for the sacrospinalis muscle.

Notes on scoliosis surgery

- Know the indications for surgery
- Approach could be anterior/posterior/combined
- Should be done in specialist centres

Perioperative care

The patient needs thorough evaluation by the multi-isciplinary team, including orthopaedics, paediatrics if child, anaesthetist, physician, etc.

Investigations

- Plain x-ray, MRI to look for associated cord abnormality, lung function tests
- May need intraoperative cord monitoring

Complications

- Infection 1–2%
- Mortality 0.03%
- Neurodeficits 0.03–1.50%
- Blindness 0.03–0.30%
- Non-union 5%.

Chapter 33

Anatomy for the FRCS (Tr & Orth)

Apurv Sinha and Fazal Ali

Introduction

Anatomy forms the basis of understanding pathology. Surgical approaches can only be appreciated if the candidate has the required knowledge of the anatomy. Also, understanding clinical examination techniques and special tests is easier when it is related to the underlying anatomy.

It is with this in mind that this chapter should be read. In the FRCS (Tr & Orth) examination, the examiners frequently discuss the relevance of anatomy to the topic.

Upper limb osteology and joints

Clavicle

The clavicle is a flat bone that serves as a strut between the scapula and the sternum. It also protects the neurovascular bundle that supplies the upper limb. The divisions of the brachial plexus are covered by the clavicle.

It is the only long bone in the body that lies horizontally and is doubly curved. Its rounded medial end articulates with the manubrium of the sternum forming the sternoclavicular joint. At its flattened lateral end it articulates with the acromion of the scapula to form the acromioclavicular joint.

The acromial end has a rough inferior surface that bears the trapezoid line and the conoid tubercle for attachment of the respective ligaments (Table 33.1).

It is the first bone to ossify in the body (5 weeks of gestation) and last to fuse (medial epiphysis fuses at 25 years).

Scapula

This is a wide, flat bone lying on the thoracic wall from the second to seventh rib and provides attachment for various groups of muscles (Table 33.2).

The scapular glenoid provides articulation for the shoulder joint and is retroverted by about 5–7°.

The processes of the scapula include:

a. Scapular spine
b. Coracoid
c. Acromion

Suprascapular notch (Figure 33.1) – The suprascapular nerve lies in the notch separated from the artery by superior transverse scapular ligament. (Water flows under the bridge.)

Spinoglenoid notch (Figure 33.1) – Both the artery and the nerve lie in the notch inferior to the inferior transverse scapular ligament. A ganglion arising from the labrum in the notch can cause compression resulting in atrophy and weakness of the infraspinatus muscle.

Coracoacromial arch – The coracoacromial ligament extends between the coracoid process and the acromion and with the coracoid process and the acromion, forms the coracoacromial arch. It is an important supero-anterior restraint for the humeral head in massive rotator cuff tears.

Shoulder joint

The shoulder is a ball and socket joint made by the humeral head and glenoid of scapula. It provides the greatest range of movement than any other joint in the body and, hence, is the least stable as well.

Stability of the shoulder joint

1. Static stabilisers
    a. Articular anatomy: retroversion of the humeral head and relative anteversion of the glenoid
    b. Glenoid labrum
    c. Articular cartilage: Thicker at periphery
    d. Negative intra-articular pressure
    e. Capsule
    f. Ligaments
2. Dynamic stabilisers
    a. Rotator cuff
    b. Biceps tendon and superior labrum
    c. Scapulothoracic motion

Important ligaments of the shoulder joint (Figure 33.2)

1. Glenoid labrum
    The laburm is a fibrocartilaginous rim attached around the margin of the glenoid cavity. It helps to deepen the glenoid cavity. At the superior margin it gives origin to the long

Table 33.1 Attachments to the clavicle

Superior surface	Deltoid muscle Trapezius muscle
Inferior surface	Subclavius muscle Conoid ligament Trapezoid ligament
Anterior border	Pectoralis major muscle
Posterior border	Sternocleidomastoid muscle Sternohyoid muscle Trapezius muscle
Lateral end	Acromioclavicular ligaments
Medial end	Sternoclavicular ligaments

Table 33.2 Attachments to the scapula

Region	Muscle
Coracoid process	Pectoralis minor Coracobrachialis Biceps brachii (short head) Coraco-acromial ligaments Coraco-clavicular ligaments
Medial border	Serratus anterior Rhomboid major Rhomboid minor Levator scapulae
Infraglenoid tubercle	Triceps brachii (long head)
Supraglenoid tubercle	Biceps brachii (long head)
Subscapular fossa	Subscapularis
Spine of scapula	Trapezius Deltoid
Supraspinous fossa	Supraspinatus
Infraspinous fossa	Infraspinatus
Lateral border	Teres minor Teres major
Inferior angle	Latissimus dorsi
Superior border	Omohyoid muscle

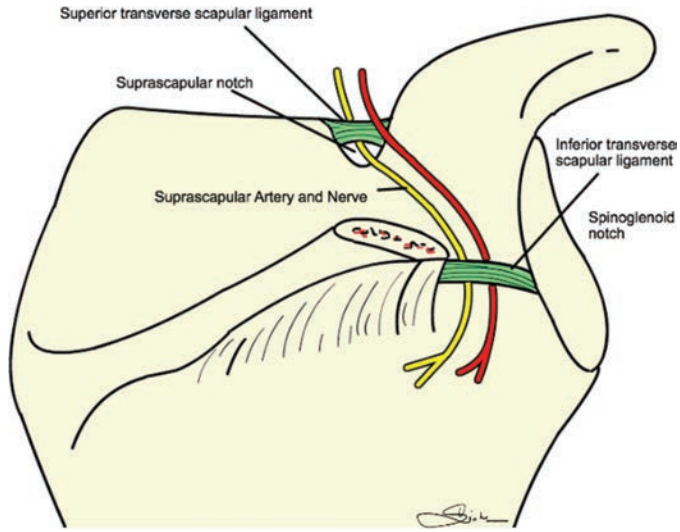

Figure 33.1 Scapula. The suprascapular nerve and vessel in the suprascapular and spinoglenoid notches

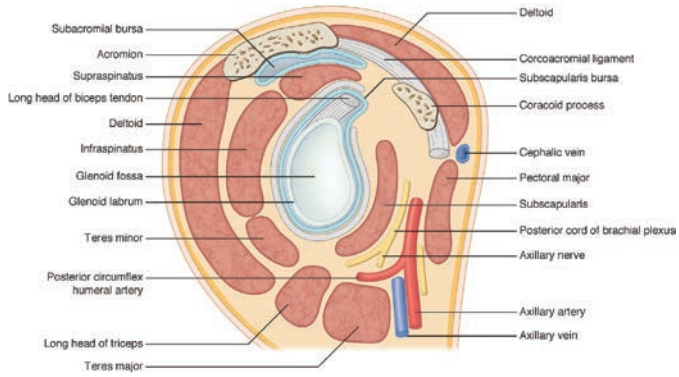

Figure 33.2 Shoulder ligaments: Cross-section of upper limb

head of biceps tendon. It is here that a superior labrum from anterior to posterior (SLAP) tear can occur

2. Glenohumeral ligaments (GHL)
   a. Superior GHL: is tightest in the adducted position of shoulder and prevents anterior and inferior glide of humeral head over the glenoid
   b. Middle GHL: is tightest in the externally rotated position and prevents anterior glide
   c. Inferior GHL: most important of three ligaments. It has anterior and posterior parts, which are taut in abducted, and externally and internally rotated positions respectively
3. Coracohumeral ligament: Arises from the lateral border of the coracoid process and passes obliquely downward and laterally to the front of the greater tuberosity of the humerus, blending with the tendon of the supraspinatus muscle
4. Transverse humeral ligament: runs from the lesser to the greater tuberosity of the humeral head and converts the intertubercular groove into a canal where long head of biceps tendon runs

Sternoclavicular (SC) joint (Figure 33.3)

The SC joint is a double-gliding joint with an articular disc that allows approximately 30° of movement with shoulder elevation.

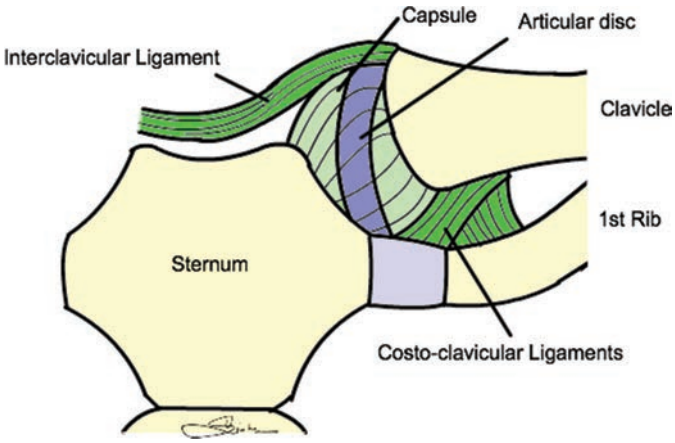

Figure 33.3 The SC joint and its ligaments

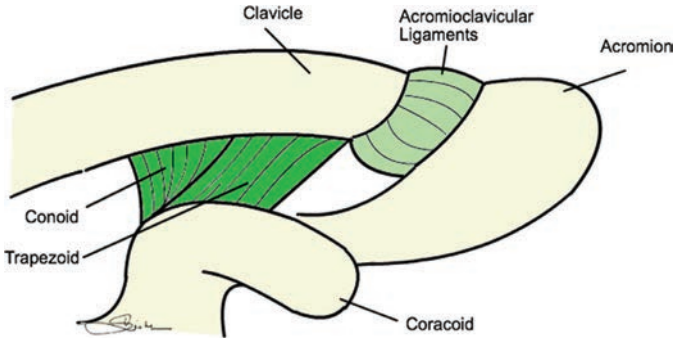

Figure 33.4 The AC joint and its ligaments

Ligaments stabilising SC joint:

a. Capsule
b. Anterior and posterior sternoclavicular ligaments
c. Interclavicular ligament
d. Costoclavicular ligament

Acromioclavicular (AC) joint (Figure 33.4)

The AC joint also consists of a fibrocartilaginous disc and allows gliding movement.

Ligaments stabilising AC joint:

a. Capsule
b. Acromioclavicular (superior and inferior) ligaments
c. Coracoclavicular ligaments
   - Trapezoid (anterolateral)
   - Conoid (posteromedial)

The acromioclavicular ligaments provide anteroposterior stability and the coracoclavicular ligaments provide superoinferior stability.

Scapulothoracic joint

The scapulothoracic joint is not a true joint but provides motion between the scapula and rib cage. For every 2° of glenohumeral joint movement there is 1° of scapulothoracic joint movement. The muscles, which mediate scapulothoracic joint rotation, are serratus anterior (anterolateral movement of inferior angle of scapula) and trapezius (superior and media pull on spine of scapula) (Figure 33.5).

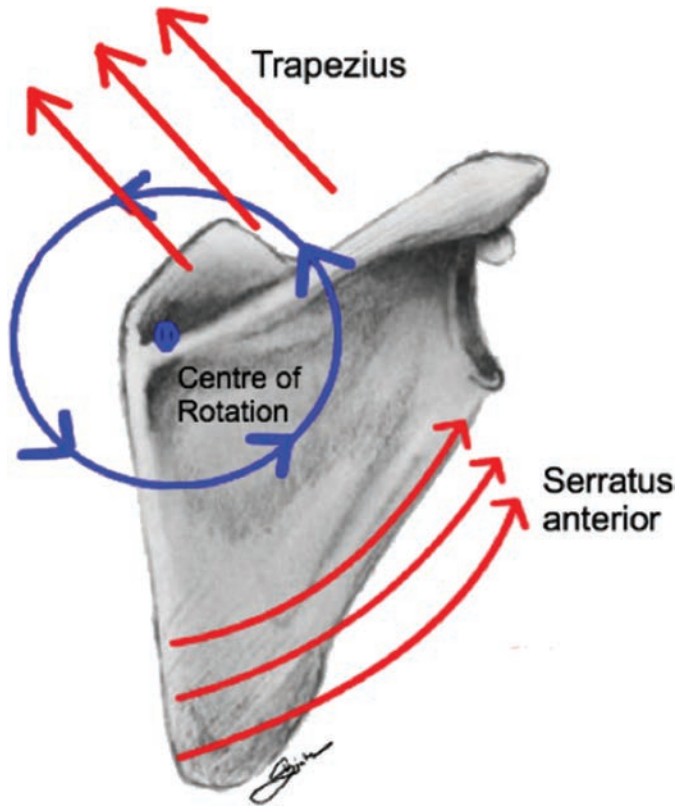

Figure 33.5 Scapula rotation – Scapular muscles involved in rotation

Scapulothoracic joint motion is important for:

- Enhancing glenoumeral joint stability
- Elevating the acromion and preventing impingement
- Maintaining an effective working length of the scapulohumeral muscles

Important spaces around shoulder (Figure 33.6)

There are three important spaces of the shoulder that allow passage of important neurovascular structures and have been tested regularly in FRCS EMQs and MCQs. The spaces are:

1. **Quadrangular space**
   The quadrangular space contains the axillary nerve and the posterior humeral circumflex artery and is bounded by
   - Medial: Long head of triceps
   - Lateral: Humeral shaft
   - Superior: Teres minor
   - Inferior: Teres major
2. **Triangular space**
   The triangular space contains the circumflex scapular vessels and is bounded by

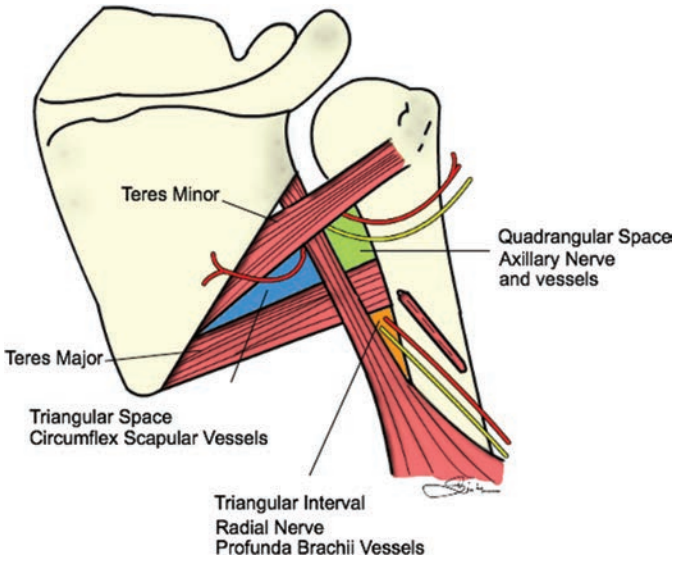

Figure 33.6 Spaces about the shoulder and their contents

- Inferior: Teres major
- Lateral: Long head of triceps
- Superior: Lower border of teres minor

3. **Triangular interval**
   The triangular interval contains the radial nerve and the profunda brachii vessels and is bounded by
   - Superior: Teres major
   - Lateral: Lateral head of the triceps or the humerus
   - Medial: Long head of the triceps

Cubital fossa (Figure 33.7 and Tables 33.3 and 33.4)

Also called the antecubital fossa, it is a triangular area located on the anterior aspect of elbow.

Elbow joint

Important ligaments of elbow joint

1. Radial collateral ligament complex (Figure 33.8)
   a. Radial collateral ligament proper
      Runs from the lateral epicondyle to the annular ligament deep to the common extensor tendon
   b. Lateral ulnar collateral ligament (LUCL)
      Runs from the lateral epicondyle to the supinator crest on the ulna. It is the chief stabiliser of elbow against varus stress
   c. Annular ligament
      Runs from the posterior to the anterior margins of radial notch on the ulna and encircles the head of radius
   d. Accessory lateral collateral/accessory annular ligament
      Runs from the inferior margin of the annular ligament to the supinator crest
2. Ulnar collateral ligament (Figure 33.9)

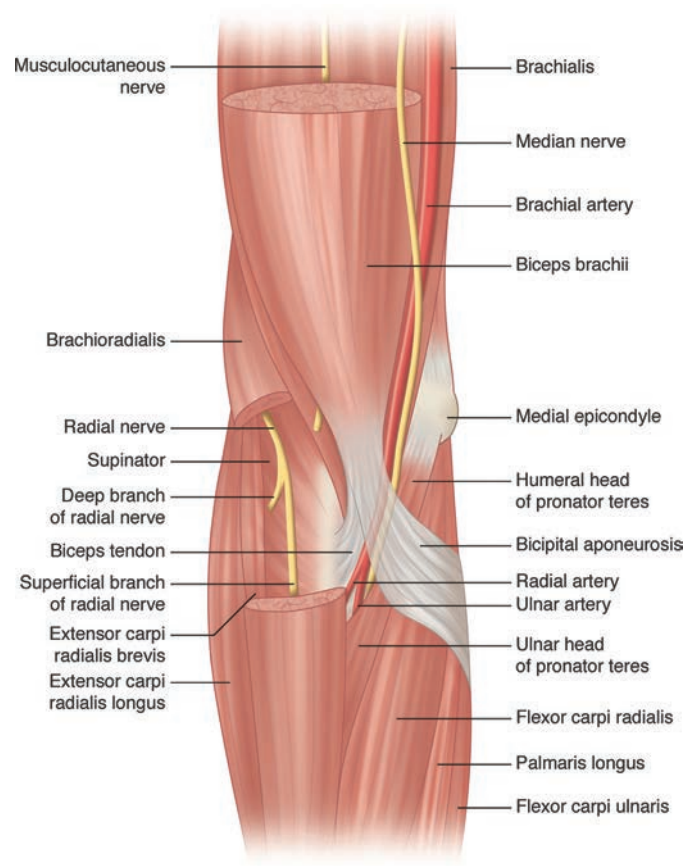

Figure 33.7 Cubital fossa (antecubital fossa)

Table 33.3 Boundaries of cubital fossa

Superior (proximal) boundary	Imaginary line joining the medial and lateral epicondyles of humerus
Medial (ulnar) boundary	Lateral border of pronator teres
Lateral (radial) boundary	Medial border of brachioradialis
Apex	Junction of the lateral and medial boundaries
Floor	Brachialis and supinator
Roof	Skin and soft tissues

Table 33.4 Contents: Lateral to medial

Radial nerve, between brachioradialis and brachialis muscles
Biceps brachii tendon
Brachial artery
Median nerve
Veins, median cubital vein, cephalic vein and basilic vein

Not to be confused with the LUCL. It is the chief stabiliser of elbow joint against valgus stress. The ulnar collateral ligament is triangular in shape. Its apex is

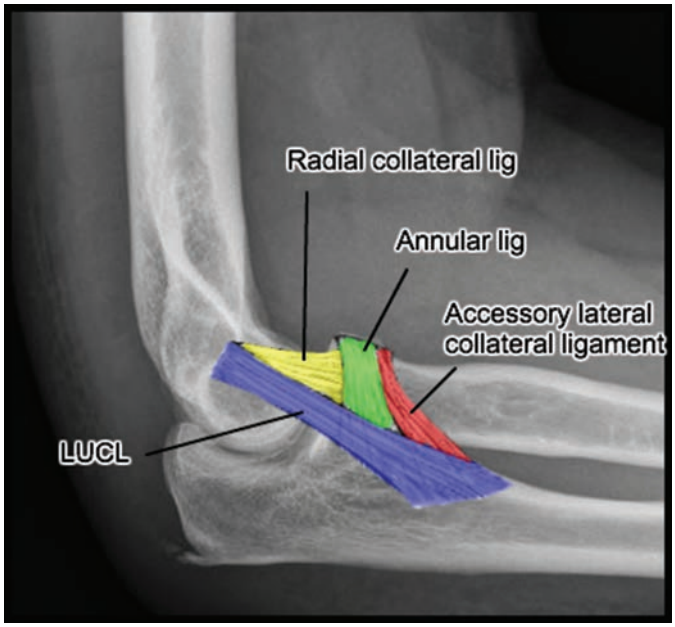

Figure 33.8 Lateral ligament complex of elbow

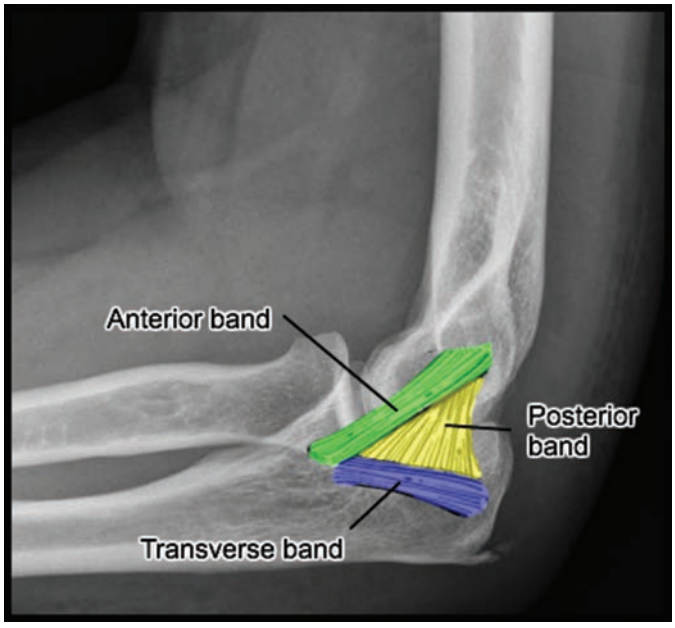

Figure 33.9 Medial ligament complex of elbow

attached to the medial epicondyle of humerus and its base to the ulna. The ligament has thick anterior and posterior bands

a. The anterior band, directed obliquely forward, is attached above by its apex to the front part of the medial epicondyle of the humerus and below, by its broad base, to the medial margin of the coronoid process of the ulna (sublime tubercle)

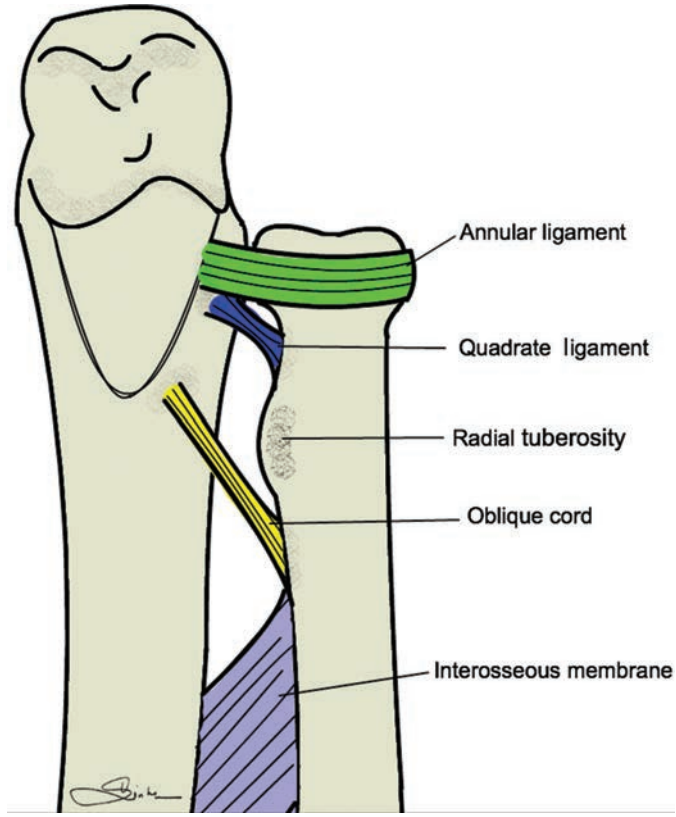

Figure 33.10 The PRUJ and its ligaments

b. The posterior band, which is shaped like a fan, is attached above by its apex to the lower and posterior part of the medial epicondyle and below to the medial margin of the olecranon
c. The transverse/oblique portion, which joins the anterior and posterior portions also called ligament of Cooper

Proximal radioulnar joint (PRUJ) (Figure 33.10)

1. Annular ligament: runs from the posterior to the anterior margins of radial notch on the ulna and encircles the head of radius
2. Quadrate ligament: a fibrous band from the inferior border of the radial notch on the ulna to the neck of the radius
3. Oblique cord: runs from the lateral side of the ulnar tuberosity at the base of the coronoid process to the radius a little below the radial tuberosity

Distal radioulnar joint (DRUJ)

Radioulnar ligaments (RULs) are the principal stabilisers of the DRUJ. There are two RULs, the palmar and dorsal radioulnar ligaments.

The PRUJ, DRUJ and the interosseous membrane coordinate the pronation and supination movements of forearm.

Wrist joint

Ligaments

The ligaments of wrist joint can be divided into extrinsic and intrinsic ligaments.

1. Extrinsic ligaments: Bridge carpal bones to the radius and ulna or metacarpals and include *volar* (stronger) and *dorsal* ligaments
   a. Volar radiocarpal ligaments (Figure 33.11)
      - The radial collateral ligament extends from the tip of the radial styloid to the radial side of the scaphoid and some fibres extend to the lateral side of the trapezium. It limits ulnar deviation at the wrist
      - Radioscaphocapitate ligament
        The radioscaphocapitate (RSC) ligament passes beneath the waist of the scaphoid, and inserts onto the capitate, creating a sling to support the scaphoid
      - Long radiolunate ligament
      - Short radiolunate ligament
      - Radioscapholunate ligament
        The radioscapholunate ligament (RSL/ligament of Testut) is not considered a true ligament, because it is composed of vascular and neural elements and no ligamentous tissue
        The *space of Poirier* (Figure 33.11) represents an area of weakness between the long radiolunate and radioscaphocapitate ligaments at the level of the mid carpal joint. In lunate dislocations, the lunate escapes into this space
   b. Volar ulnocarpal ligaments (Figure 33.11)
      - Ulnotriquetral ligament
      - Ulnolunate ligament
      - Ulnocapitate ligament
        The ulnolunate and ulnocapitate ligaments prevent dorsal migration of the distal ulna
   c. Dorsal ligaments (Figure 33.12)
      - Radiotriquetral
      - Dorsal intercarpal (DIC)
      - Radiolunate
      - Radioscaphoid
2. Intrinsic ligaments: originate and insert on carpal bones
   a. Scapholunate interosseous ligament
      The scapholunate ligament is an intra-articular ligament binding the scaphoid and lunate bones of the wrist together. It is divided into three areas, dorsal, proximal and palmar, with the dorsal segment being the strongest part. It is the main stabiliser of the scaphoid
   b. Lunotriquetral interosseous ligament
      In contrast to the scapholunate ligament, the lunotriquetral ligament is more prominent on the palmar side
   c. Trapeziotrapezoid ligament
   d. Capitotrapezoid ligament
   e. Capitohamate ligament

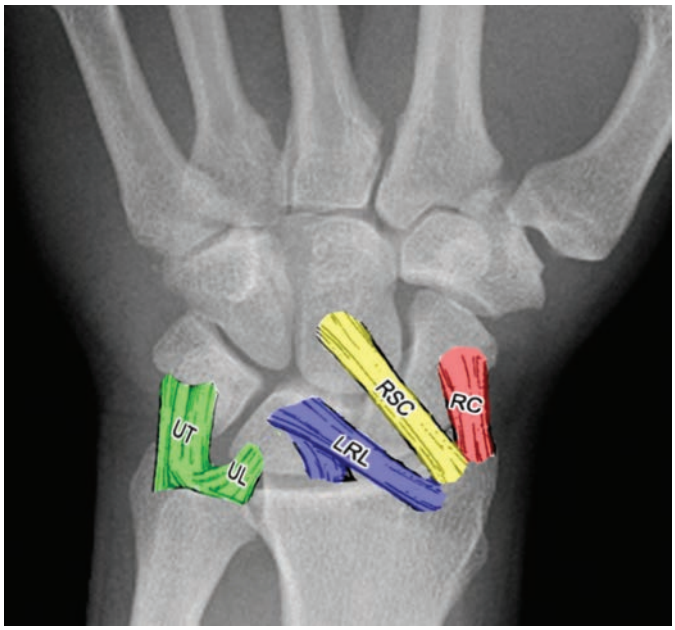

Figure 33.11 Extrinsic volar wrist ligaments. LRL, long radiolunate ligament; RC, radial collateral; RSC, radioscaphocapitate; UL, ulnolunate ligament; UT, ulnotriquetral ligament

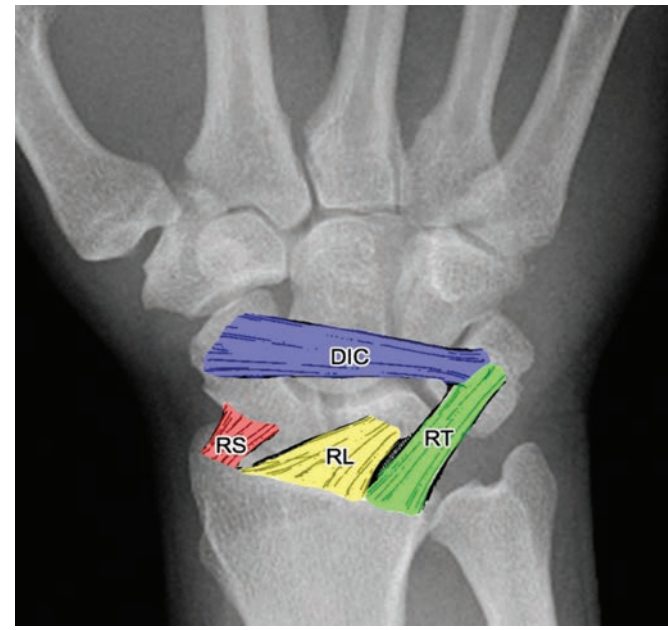

Figure 33.12 Extrinsic dorsal wrist ligaments. DIC, dorsal intercarpal; RL, radiolunate; RS, radioscaphoid; RT, radiotriquetral

Triangular fibrocartilage complex (TFCC)

This is formed by the

1. Triangular fibrocartilage (TFC)
2. Ulnocarpal ligaments (volar ulnolunate and ulnotriquetral ligaments)
3. Meniscal homologue
4. Extensor carpi ulnaris (ECU) sheath

Its origin is from the medial border of the distal radius and it inserts into the base of the ulnar styloid. TFCC is a main stabiliser of the radioulnar joint.

The TFC is an articular discus that lies on the pole of the distal ulna. It has a triangular shape and a biconcave body; the periphery is thicker than its center. The central portion of the TFC is thin and consists of chondroid fibrocartilage. This type of tissue is often seen in structures that can bear compressive loads. The central area is often so thin that it is translucent and in some cases it is even absent. The peripheral portion of the TFC is well vascularized, while the central portion has no blood supply.

This discus is attached by thick tissue to the base of the ulnar styloid and by thinner tissue to the edge of the radius just proximal to the radiocarpal articular surface.

Hand

Extensor tendons/dorsal digital expansion (Figure 33.13)

The dorsal digital expansion/dorsal hood is the special connective attachment by which the extensor tendons insert into the phalanges.

Distal to the metacarpophalangeal (MCP) joint, the extensor digitorum tendon flattens to form extensor expansions. The extensor expansion (hood or dorsal expansion) is a triangular, tendinous expansion that wraps around the dorsum and sides of the metacarpals and proximal phalanx.

The expansion then divides into three parts which progress further as two lateral slips on either side and a central slip in middle.

The *lateral slips* join the deep head of the interosseous and lumbrical muscles to become the conjoined lateral band. The two lateral bands converge and join over the middle phalanx and go on to insert on the base of the dorsum of the distal phalanx and extend the distal interphalangeal joint (DIPJ).

The *central slip* inserts into base of the dorsum of middle phalanx and extends the proximal interphalangeal joint (PIPJ).

The *retinacular ligaments* help to retain and position common extensor mechanism during PIP and DIP flexion.

- Transverse retinacular ligament arises from the flexor tendon sheath and inserts onto the lateral border of the conjoined lateral bands. It prevents excessive dorsal translation of lateral bands
- Oblique band (oblique retinacular ligament of Landsmeer) links the motion of the DIPJ to the PIPJ. It arises from the flexor tendon sheath and inserts onto the terminal tendon of the extensor mechanism and extends the DIPJ when the PIPJ is extended

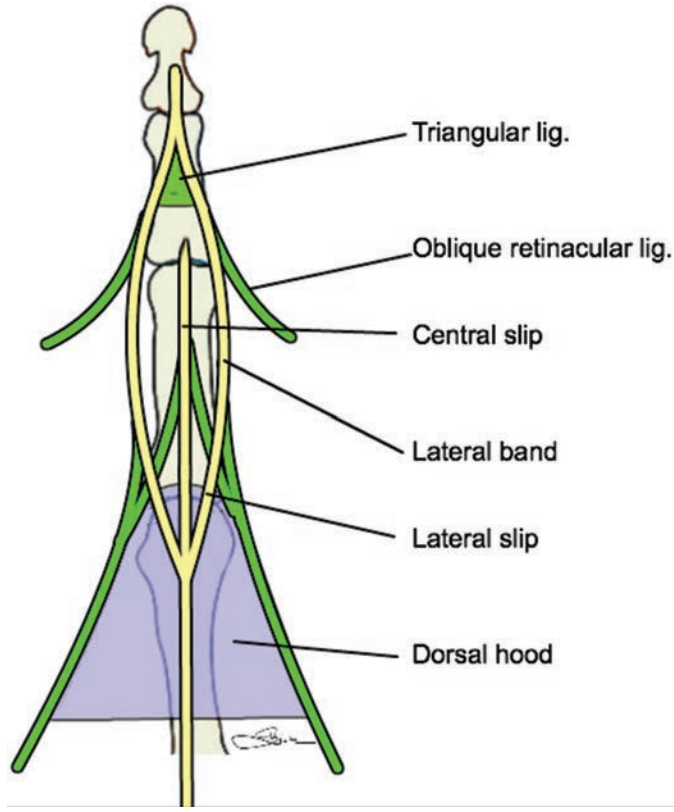

Figure 33.13 The extensor tendon and dorsal digital expansion

The *triangular ligament* joins the two lateral bands before they join together to become the terminal tendon. They prevent volar subluxation of the lateral bands.

The extensor tendon is held centrally by the *sagittal bands* at the level of the MCP joint. They arise from the volar plate and intermetacarpal ligaments volarly and attach to the extensor tendon dorsally

Other important ligaments in the hand

Deep transverse metacarpal ligament: This interconnects the second to fifth metacarpal heads at the volar plate and prevent them from splaying apart (abduction)

Natatory ligament (superficial transverse metacarpal ligament): This runs from the distal metacarpal to proximal phalanx and resists abduction

Triangular ligament: This is present between the two lateral bands over the dorsal side distal to the PIPJ and counteracts the pull of the oblique retinacular ligament, preventing lateral subluxation of the common extensor mechanism (Figure 33.13)

Volar plate: This is a thickening of the joint capsule volar to the MCP joint and the PIPJ. It prevents hyperextension of the joint

Table 33.5 Sequence of pulleys in finger

A1	A2	C1	A3	C2	A4	C3	A5
MCP joint	Proximal phalanx		PIP joint		Middle phalanx		DIP joint

Anterior oblique ligament: This is a primary stabiliser of the trapeziometacarpal joint and is taut in abduction, extension and pronation. It originates from the tuberosity of the trapezium and inserts on the volar ulnar edge of the thumb metacarpal base

Ulnar collateral ligament of thumb: This runs along the ulnar side of the MCP joint of the thumb and is on the radial side of the wrist joint but the ulnar side of the thumb. It originates from the metacarpal head and inserts into the medial aspect and the base of the proximal phalanx of the thumb

Digital cutaneous ligaments

Grayson's ligament

These are thin ligaments that originate from the volar aspect of the tendon sheath and insert into the skin. Grayson's ligament prevents bowstringing of the neurovascular bundle during finger flexion.

Cleland's ligaments

These are firm fascial bands that run from the osseous flexor tendon gutter at the PIPJs and pass dorsal to the neurovascular bundle to attach to the digital fascia. They serve as a window through which the retinacular ligament passes and act to retain different parts of the integument in position.

Cleland's ligaments work with Grayson's ligaments to hold the skin in position during flexion and extension of the fingers.

Flexor pulley system in fingers

The flexor pulley system (Table 33.5) helps to prevent bowstringing of flexor tendon

There are two types of pulleys, annular and cruciate.

Annular: There are five annular pulleys – A1 to A5 from proximal to distal. The odd-numbered pulleys A1, A3 and A5 overlie the MCP, PIP and DIP joints respectively and originate from volar plate

The even-numbered pulleys, A2 and A4, arise from the periosteum of the proximal and middle phalanx and are critical in preventing bowstringing of the flexor tendons. The A1 pulley is involved in trigger finger

Cruciate: Cruciate pulleys are three in total and are placed in-between the annular pulleys from A2 onwards (no cruciate pulley between A1 and A2). The cruciate pulley's orientation of fibres allows them to collapse, preventing crimpling of the tendon sheath with finger flexion

Table 33.6 Flexor pulley system in the thumb

A1	Oblique pulley	A2
MCP joint	Proximal phalanx	Distal proximal phalanx

Flexor pulley system in the thumb (Table 33.6)

The thumb has two annular and one oblique pulley. The A1 annular pulley overlies the MCP joint and the A2 over the distal part of the proximal phalanx.

The oblique pulley, which is the most important and prevents bowstringing of flexor pollicis longus tendon is between the A1 and A2 pulleys, centred over the proximal phalanx and is in continuity with the adductor pollicis insertion.

Palmar aponeurosis pulley

The fingers have a palmar aponeurosis pulley, which is a condensation of palmar fascia proximal to the A1 pulley. This pulley becomes mechanically important if the other pulleys are injured or non-functional.

Flexor tendons

Flexor digitorum superficialis (FDS) enters the fibrous flexor sheath on the palmar surface of the flexor digitorum profundus (FDP). It divides into two halves which spiral around the FDP tendon to insert onto the sides of the middle phalanx of the medial four fingers.

The FDP enters the fibrous flexor sheath deep to FDS, then lies superficial to the partial decussation of the FDS before attaching to the base of the distal phalanx.

The blood supply is via short and long vincula to both FDS and FDP.

Carpal tunnel

This is the route by which major structures pass into the palm of the hand from the forearm (Figure 33.14). The volar surface of the carpal bones form a concavity and this concavity is roofed over by a thick fibrous band, the flexor retinaculum converting the concavity into a tunnel through which structures pass.

Flexor retinaculum

This is a distal continuation of two sheets of fascia: The deep fascia of the forearm and fascia over flexor digitorum superficialis. These two sheets come together and later become continuous with the palmar aponeurosis.

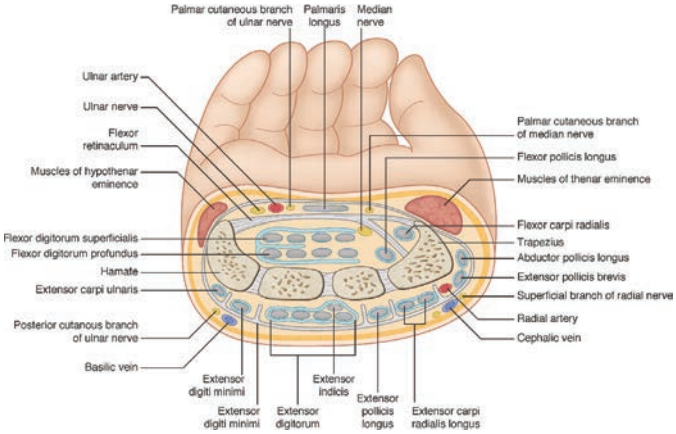

Figure 33.14 Carpal tunnel anatomy

The attachments are:

- Medial: Pisiform, hook of hamate
- Lateral: Tubercle of scaphoid, ridge of trapezium
- There is also a deep slip, which is attached to the medial lip of a groove on the trapezium

Structures passing superficial to the retinaculum (medial to lateral):

- Tendon of the flexor carpi ulnaris (FCU) (attached to pisiform bone)
- Ulnar nerve and vessels (close to pisiform bone)
- Palmar cutaneous branches of the ulnar nerve
- Tendon of palmaris longus (middle of retinaculum)
- Palmar cutaneous branch of the median nerve

Structures passing through the carpal tunnel:

- The four tendons of the FDS pass through in two layers, with the middle and ring lying in front of index and little fingers
- The four tendons of FDP all lie on the same plane deep on the carpal bones
- The tendon of FCR passes down to the base of metacarpals 2 and 3
- The median nerve passes beneath flexor retinaculum on the lateral side of FDS (middle finger tendon), between it and FCR
- The tendon of FPL lies in its own synovial sheath as it passes through the radial side of the tunnel

Functions of the retinaculum:

- Acts as a protracting and restraining device which is essential to stop bowstringing of the long tendons
- It gives partial insertion to some muscles (palmaris longus (PL), FCU)
- It gives partial origin to some muscles (thenar and hypothenar muscles)

Anatomical snuffbox

The anatomical snuffbox is a triangular deepening on the dorso-radial aspect of the hand at the level of the carpal bones.

Table 33.7 Extensor zones

Zone	Anatomical location	Clinical significance
Zone I	At or distal to DIPJ	Mallet finger
Zone II	Middle phalanx	Lacerations over phalanx
Zone III	PIPJ	Boutonnière lesion
Zone IV	Proximal phalanx	Adhesion formation is a common complication which may require tenolysis
Zone V	MCP joint	Human bites
Zone VI	Metacarpal	Most frequently injured zone, Associated lacerations of superficial sensory branches of the radial or ulnar nerves
Zone VII	Wrist joint	
Zone VIII	Distal forearm	Musculotendinous junction
Zone IX	Proximal forearm	Laceration may injure posterior interosseous nerve (PIN)

The boundaries and contents have been frequently tested in the FRCS Orth MCQs and EMQs.

Boundaries

Dorsal: Extensor hallucis longus (EHL)

Volar: Abductor pollicis longus and extensor pollicis brevis (APL and EPB)

Proximal: Radial styloid

Distal: Apex of the triangle where dorsal and volar borders meet

Floor: Scaphoid and trapezium

Contents

- Radial artery
- Cephalic vein
- Dorsal cutaneous br. of radial nerve

Tendons in the hand

Extensor zones

Zones are used to describe the location of tendon injuries.

There are nine zones of injury as listed in Table 33.7. Extensor compartments of the wrist (Figure 33.15: Ext ret wrist): The extensor tendons of the wrist course through six fibro-osseous compartments (Table 33.9). These have been tested on many occasions in FRCS section 1 as EMQs.

Flexor zones (Verdan's zone) (Table 33.8)

Table 33.8 Flexor zones

Zone	Anatomical location	Clinical significance
Zone I	Distal to FDS insertion	'Rugger jersey finger' Classified by Leddy
Zone II	Zone I and metacarpal neck	'No man's land' Two tendons in Zone II'
Zone III	Between distal palmar crease and the distal end of the transverse carpal ligament	The Lumbrical muscles originate from the radial aspect of the FDP tendon in this zone
Zone IV	The carpal tunnel	Tendons can be injured in various combinations along with median and/or ulnar nerve
Zone V	proximal edge of the transverse carpal ligament and the musculotendinous junction flexors	Spaghetti wrist

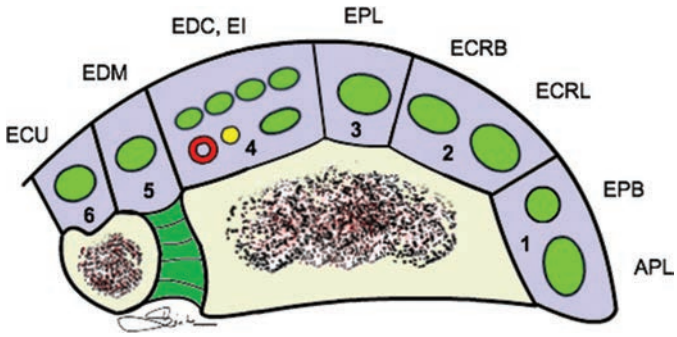

Figure 33.15 Dorsal wrist compartments and contents. APL, abductor pollicis longus; ECRB, extensor carpi radialis brevis; ECRL, extensor carpi radialis longus; ECU, extensor carpi ulnaris; EDC, extensor digitorum communis; EDM, extensor digiti minimi; EI, extensor indicis; EPB, extensor pollicis brevis; EPL, extensor pollicis longus

Table 33.9 Extensor compartments of the wrist

I	Abductor pollicis longus and extensor pollicis brevis	First compartment for tendons of first finger thumb
II	ECRL and brevis ECRL (second MC base) ECRB (third MC base)	Second compartment for radial wrist extensors ECRL and ECRB
III	EPL	Third EPL around Lister's tubercle
IV	Extensor digitorum, EIP The posterior interosseous artery and nerve located on the floor radial side of fourth compartment	Fouth compartment for extensors of four fingers
V	Extensor digiti minimi	Fifth compartment for fifth finger extensor
VI	ECU	Sixth compartment for ulnar wrist extensors ECU

ECRB, extensor carpi radialis brevis; ECRL, extensor carpi radialis longus; ECU, extensor carpi ulnaris; EIP, extensor indicis proprius; EPL, extensor pollicis longus; MC, metacarpal.

Table 33.10 Blood supply of scaphoid

Radial artery	Dorsal	Dorsal carpal artery branches	Dorsal ridge of scaphoid	70–80% distal to proximal
	Volar	Superficial palmar arch branches	Tubercle of scaphoid	20–30% distal to proximal

Scaphoid

The radial surface is a convex area that extends well onto the dorsal surface. The tubercle is a blunt prominence to the thumb side of the distal surface and is palpable. It provides attachment for the flexor retinaculum and abductor pollicis brevis. The narrow non-articular waist is palpable in the snuff-box distal to the radial styloid. It is perforated, especially on its dorsal surface, by vascular foramina. There is a flat surface medially for the lunate, and a large concavity distomedially for the capitate. The triangular convex distal surface articulates with the trapezium and trapezoid.

Blood supply of scaphoid (Table 33.10)

The major blood supply of the scaphoid is derived from dorsal carpal branches of the radial artery entering the dorsal ridge by vascular foramina from ligamentous and capsular attachments. This supplies the 70–80% of distal scaphoid in a retrograde fashion including the proximal pole.

A second major group, palmar and superficial palmar branches of the radial artery enters the scaphoid tubercle volarly and perfuses only the distal 20–30% of bone.

The proximal pole is the region with the most tenuous blood supply owing to the distal to proximal (retrograde) vascular supply. There are no anastomoses between dorsal and palmar vessels. Fractures across the waist can result in avascular necrosis of the proximal fragment.

Upper limb muscles (Tables 33.11–33.13)

Rotator cuff muscles (Table 33.14)

The rotator cuff is composed of four muscles originating form the scapula and inserting on humeral tuberosities.

Table 33.11 Anterior group muscles connecting upper limb to trunk

Muscle	Origin	Insertion	Action	Innervation	Notes
Pectoralis major	Clavicle head: Med. half clavicle. Sternocostal head: Lat manubrium and sternum, upper six costal cartilages and ext. oblique aponeurosis	Lat. lip of bicipital groove of humerus and ant. lip of deltoid tuberosity	Clavicle head: Flexes and adducts arm. Sternal head: Adducts and med. rotates arm	Med. pectoral nerve (from med. cord) and lat. pectoral nerve (from lat. Cord)(C6, C7, C8)	
Pectoralis minor	Ribs 3–5	Med. and upper surface of coracoid process of scapula	Protracts scapula (assists serratus ant.), elevates ribs if scapula fixed (accessory muscles of respiration)	Med. pectoral nerve (from med. Cord) and lat. pectoral nerve (from lat. cord) (C6, C7, C8)	Important landmark for underlying axillary artery and cords of brachial plexus
Serratus anterior	Upper eight ribs and ant. intercostal membranes from midclavicular line. Lower four interdigitate with external oblique	Inner med. border scapula.	Lat. rotates and protracts scapula	Long thoracic nerve (roots of brachial plexus C5, C6, C7)	Forms medial wall axilla

Table 33.12 Posterior group muscles connecting upper limb to trunk

Muscle	Origin	Insertion	Action	Innervation	Notes
Trapezius	Med. third superior nuchal line, ligamentum nuchae, spinous process and supraspinous ligaments, all thoracic vertebrae	Upper fibres to lat. third of post. border of clavicle, lower to med. acromion and sup. lip of spine of scapula to deltoid tubercle	Lat. rotates, elevates and retracts scapula. If scapula is fixed, extends and lat. flexes neck	Spinal part of accessory nerve (C1–5)	Fibres divided into three parts: Descending, middle and ascending
Latissimus dorsi	Spine T7, spinous process and supraspinous ligaments of all lower thoracic, lumbar and sacral vertebrae, lumbar fascia, post. third iliac crest, last four ribs (interdigitating with ext. oblique abdominis) and inf. angle of scapula	Floor of bicipital groove of humerus after spiralling around teres major. (Mnemonic for insertion: The lady and the two majors)	Extends, adducts and med. rotates arm	Thoracodorsal nerve (C6, C7, C8) (from post. cord)	Very wide origin, very narrow insertion. Forms lower border post. axillary fold
Levator scapulae	Post. tubercles of transverse process of C1–C4	Upper part of med. border of scapula	Raises med. border of scapula	Ant. primary rami of C3, C4 and dorsal scapular nerve (C5)	Strap-like muscle in the floor post. Triangle neck
Rhomboid major	Spines of T2–T5 and supraspinous ligaments	Lower half of posteromedial border of scapula, from angle to upper part of triangular area at base of scapula spine	Retracts scapula	Dorsal scapular nerve (C5) (from root)	
Rhomboid minor	Lower ligamentum nuchae, spines of C7 and T1	Posteromedial border of scapula at level of spine, below levator scapula	Retracts scapula	Dorsal scapular nerve (C5) (from root)	Narrow band parallel with above

Table 33.13 Scapulohumeral muscles

Muscle	Origin	Insertion	Action	Innervation	Notes
Deltoid	Lat. third clavicle, acromion and spine of scapula	Lat. surface humerus (deltoid tuberosity)	Abducts the arm, ant. fibres flex and internally rotate, post. fibres extend and laterally rotate	Axillary nerve (C5, C6) (from post. cord	
Teres major	Oval area (lower third) of lat. side of inf. angle of scapula below teres minor	Med. lip of bicipital groove of humerus	Med. rotates and adducts arm. Stabilises shoulder joint	Lower subscapular nerve (C6, C7)	Offspring of subscapularis which has migrated to dorsal surface scapula

Table 33.14 Rotator cuff muscles

Muscle	Origin	Insertion	Action	Innervation	Notes
Supraspinatus	Med. three-quarters of supraspinous fossa of scapula and upper surface spine scapula	Superior facet of greater tuberosity humerus and capsule of shoulder joint	Stabilises shoulder joint and helps in shoulder abduction	Suprascapular nerve (C5, C6) (from upper trunk)	Suprascapular nerve passes beneath superior transverse ligament of scapula and the artery goes over it
Infraspinatus	Med. three-quarters of infraspinous fossa of scapula	Middle facet of greater tuberosity of humerus and capsule of shoulder joint	External rotation of the arm	Suprascapular nerve (C5, C6) from upper trunk	Partly covered by deltoid and trapezius
Teres minor	Middle third lateral border of scapula	Inferior facet of greater tuberosity of humerus (below infraspinatus) and capsule of shoulder joint	External rotation of the arm and stabilises shoulder joint	Axillary nerve (C5, C6) (from post. cord)	Often not clearly differentiated from the infraspinatus. Hidden by deltoid
Subscapularis	Med. two-thirds of costal surface of scapula	Lesser tuberosity, upper med. lip bicipital groove and capsule of shoulder joint	Internal rotation of the arm and stabilises shoulder joint	Upper and lower subscapular nerve (C6, C7) (from post. cord)	Forms part of post. wall of axilla. Tendon fuses with capsule of shoulder joint

These muscles combine at the shoulder to form a thick cuff over the joint.

Functions include:

- Stabilising and centralizing the humeral head against the glenoid
- During the arc of shoulder motion cuff muscles maintain the humeral head within the centre glenoid cavity
- Concavity compression: During abduction of the arm, the rotator cuff compresses the humeral head into the centre of glenoid and stabilises the glenohumeral joint (GHJ). Without this effect contraction of deltoid will result in superior migration of humeral head rather than abduction and will reduce its efficiency (features seen in cuff tear arthropathy)
- Rotating the arm: The subscapularis helps in internal and the infraspinatus and teres minor help on external rotation of the arm

The long head of biceps could be regarded as a functional part of the rotator cuff. It inserts to the supraglenoid tubercle.

Muscles of the arm (Tables 33.15 and 33.16)

The anterior (flexor) muscle group is twice as strong as the posterior (extensor) group. Medial head of triceps would be better called deep head and the long head is actually medial. The radial nerve runs with the profunda brachii artery in the radial grove of the humerus between medial and lateral heads of triceps.

Muscles of the anterior compartment of the forearm (Figure 33.16 and Table 33.17)

These are arranged in two groups: Superficial and deep. The flexor digitorum superficialis (FDS) is frequently described as being in an intermediate layer. The flexor compartment is more bulky than extensor compartment.

Table 33.15 Anterior group muscles of arm

Muscle	Origin	Insertion	Action	Innervation	Notes
Coracobrachialis	Coracoid process of scapula with biceps brachii	Upper half of med. border of humerus	Flexes and weakly adducts arm	Musculocutaneous nerve (C6, C7)	Median nerve or brachial artery or both may run beneath it and be susceptible to compression. Musculocutaneous nerve passes through it.
Biceps	Long head: Supraglenoid tubercle of scapula. Short head: Coracoid process of scapula with coracobrachialis	Post. border of bicipital tuberosity of radius and bicipital aponeurosis to deep fascia and subcutaneous ulna	Supinates forearm, flexes elbow, weak flexor shoulder	Musculocutaneous nerve (C6, C7)	No attachment to the humerus
Brachialis	Ant. lower half of humerus and med. and lat. intermuscular septa	Coronoid process and tuberosity of humerus	Flexes elbow	Musculocutaneous nerve (C5, C6)	Lies posterior (deep) to biceps. Distal part covers ant. aspect of elbow. Workhorse of elbow flexors

Table 33.16 Posterior group muscles of arm

Muscle	Origin	Insertion	Action	Innervation	Notes
Triceps	Long head: Infraglenoid tubercle of scapula. Lat. head: Upper half post. humerus (linear origin). Med. head: Lower half post. humerus inferomedial to spiral groove and both intermuscular septa	Post. part of upper surface of olecranon process of ulna and post. capsule	Extends elbow. Long head stabilises shoulder joint. Med. head retracts capsule of elbow joint on extension	Radial nerve (C7, C8) (from post. cord) four branches	

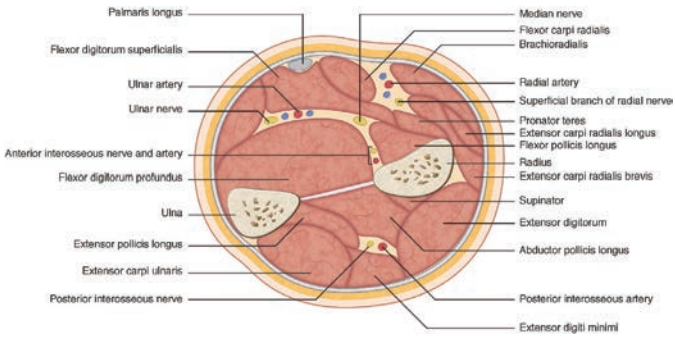

Figure 33.16 Cross-section of mid forearm

Muscles of the posterior compartment of the forearm (Table 33.18)

The muscles can be arranged into three functional groups:

- Muscles that extend and adduct or abduct the hand at the wrist joint (ECRL, ECRB, ECU)
- Muscles that extend the four fingers (extensor digitorum (ED), extensor indicis (EI) and extensor digiti minimi (EDM))
- Muscles that extend or abduct the thumb (APL, EPB and EPL)

They are organized anatomically into superficial and deep groups. Four of the superficial extensors are attached proximally by a common extensor tendon to the lateral epicondyle. The proximal attachment of the two other muscles is to the lateral supraepicondylar ridge of the humerus and lateral intermuscular septum.

Muscles of the hand and wrist (Table 33.19)

The thenar eminence consists of three short thumb muscles, which arise from the flexor retinaculum. The hypothenar eminence is on the ulnar (medial) side of the palm. There is a similar arrangement to the thenar eminence.

Upper limb nerves

Spinal accessory nerve (C1–C4)

The accessory nerve is formed by the fibres from the

- *Cranial root* from medulla oblongata
- *Spinal root* from cervical level C1–C4

The cranial part really belongs to the vagus nerve and gives all its fibres to it for the supply of the skeletal muscles of the pharynx and palate.

Table 33.17 Muscles of the anterior compartment of the forearm

Muscle	Origin	Insertion	Action	Innervation	Notes
Superficial flexors	The five muscles are attached to the common flexor. Origin is anterior aspect of the medial epicondyle of the humerus All cross the elbow; the three muscles of the deep group do not				
Pronator teres	Humeral head: Med. epicondyle, med. supracondylar ridge and med. intermuscular septum. Ulnar head: Med. border of coronoid process	Middle of lat. convex surface of the radius	Pronates forearm and flexes elbow	Median nerve (C6, C7)	Median nerve passes between its two heads
Flexor carpi radialis	Common flexor origin of med. epicondyle of humerus	Bases of second and third MCs via groove in trapezium and slip to scaphoid	Flexes and abducts wrist	Median nerve (C6, C7) (from median and lateral cords)	Radial artery lies lateral and median nerve medial
Palmaris longus	Common flexor origin of med. epicondyle of humerus	Flexor retinaculum and palmar aponeurosis	Flexes the hand and tightens palmar aponeurosis	Median nerve (C6, C7) (from medial and lateral cords)	Vestigial muscle often absent. Short belly and long tendon absent in 12%. Tendon slightly overlaps median nerve from the ulnar side
Flexor carpi ulnaris	Humeral head: Common flexor origin of med. epicondyle. Ulnar head: Aponeurosis from med. olecranon and upper three-quarters of subcutaneous border of ulna	Pisiform, hook of hamate, base of fifth MC via pisohamate and pisometacarpal ligaments	Flexes and adducts wrist	Ulnar nerve (C6, C7)	Ulnar nerve enters flexor compartment of forearm by passing between humeral and ulnar heads muscle
Flexor digitorum superficialis	Humeral head: Common flexor origin of med. epicondyle of humerus, med. ligament of elbow. Ulnar head: Sublime tubercle (med. border of coronoid process) and fibrous arch. Radial head: Whole length of ant. oblique line	Tendons split to insert onto sides of middle phalanges of med. Four fingers	Flexes prox. IP joints and, secondarily, MCP joints and wrist	Median nerve (C6, C7) (from median and lateral cords)	Middle and ring finger superficial to index and little finger beneath flexor retinaculum. In the forearm median nerve plastered to its deep surface
Deep flexors					
Flexor pollicis longus	Ant. surface of radius below ant. oblique line and adjacent interosseous membrane	Base of distal phalanx thumb	Flexes DIP joint of thumb	Ant. Interosseous nerve (C7, C8)	Long flexor of the thumb. Lies lateral to the FDP where it clothes ant. aspect of radius distal to supinator. Passes deep to the flexor retinaculum within its own synovial sheath
Flexor digitorum profundus	Med. olecranon, upper three-quarters of ant. and med. surfaces of ulnar as far round as subcutaneous border and narrow strip of interosseous membrane	Distal phalanges of med. Four fingers	Flexes distal IP joints. Secondarily flexes prox. IP and MCP joints and wrist	Median nerve (ant. Interosseous) (C6, C7)/ulnar nerve (C7, C8)	covers ant. aspect of the ulna. Bulkiest of the forearm muscles. The 2:2 distribution between median and ulnar nerves occurs in only 60%
Pronator quadratus	Lower quarter of anterolateral shaft of radius and some interosseous membrane	Lower quarter of anterolateral shaft of ulna and some interosseous membrane	Pronates forearm and maintains ulna and radius opposed	Ant. Interosseous nerve (C8)	Ant. interosseous artery pierces the interosseous membrane at the upper border of pronator quadratus. Only muscle to attach to radius at one end and ulna at the other end

Table 33.18 Muscles of the posterior compartment of the forearm

Muscle	Origin	Insertion	Action	Innervation	Notes
Superficial extensors					
Brachioradialis	Upper two-thirds of lat. supracondylar ridge of humerus and lat. intermuscular septum	Base of styloid process of radius	Flexes arm at elbow and brings forearm into midprone position	Radial nerve (C6, C7)	Middle of forearm: Superficial branch of the radial nerve and radial artery are under cover of brachioradialis
Extensor carpi radialis longus	Lower third of lat. supracondylar ridge of humerus and lat. intermuscular septum	Dorsal surface base of second MC	Extends and abducts hand at the wrist	Radial nerve (C6, C7)	Tendon crossed by abductor pollicis brevis and extensor pollicis brevis
Extensor carpi radialis brevis	Common extensor origin on ant. aspect of lat. epicondyle of humerus	Dorsal surface of base of third MC	Extends and abducts hand at the wrist	Radial nerve (C6, C7)	Shorter than ECRL as arises more distally. Both pass under the retinaculum along with ECR
Extensor digitorum	Common extensor origin on ant. aspect of lat. epicondyle of humerus	Extensor expansion to middle and distal phalanges by four tendons	Extends wrist. Extension of all finger joints	Post interosseous nerve (C7, C8)	Occupies much of the post. surface of forearm. At distal ends of the MCs the four tendons flatten to form extensor expansions
Extensor digiti minimi	Common extensor origin on ant. aspect of lat. epicondyle of humerus	Extensor expansion of little finger – Usually double tendon joined by slip from extensor digitorum at MCP joint	Extends all joints of little finger	Post interosseous nerve (C7, C8)	A partially detached part of extensor digitorum. Runs through a separate compartment of the extensor retinaculum. Divides into two slips
Extensor carpi ulnaris	Common extensor origin on ant. aspect of lat. epicondyle of humerus	Base of fifth MC via groove by ulnar styloid	Extends and adducts hand at the wrist	Post interosseous nerve (C7, C8)	Long fusiform muscle located on the med. border of the forearm. Lies in a groove beside the ulnar styloid
Deep extensors					
Supinator	Deep part (horizontal): Supinator crest and ulnar fossa. Superficial part (downwards): Lat. epicondyle and lat. ligaments of elbow and annular ligament	Neck and shaft of radius between ant. and post. oblique lines	Supinates forearm	Post interosseous nerve (C7, C8)	Two parts with different origins passing in different directions. PIN passes between these parts. Biceps principal supinator with rapid forceful supination against resistance (using a screw driver). Supinator is a slow prime mover with the elbow extended
Abductor pollicis longus	Upper post. surface of ulnar and middle third of post. surface of radius and interosseous membrane	Over tendons of radial extensors and brachioradialis to base of first MC and trapezium	Abducts and extends thumb at CMC	Post interosseous nerve (C7, C8)	Spirals around the radial extensors of the wrist and brachioradialis to reach the base of the first MC
Extensor pollicis brevis	Lower third of post. shaft of radius and adjacent interosseous membrane	Base of prox. phalanx of thumb	Extends MCP joint thumb	Post interosseous nerve (C7, C8)	Forms radial border of snuffbox

Table 33.18 (*cont.*)

Muscle	Origin	Insertion	Action	Innervation	Notes
Extensor pollicis longus	Middle third of post. ulna (below abductor pollicis longus) and adjacent interosseous membrane	Base of distal phalanx thumb via Lister's tubercle	Extends IP and MCP joints of thumb	Post interosseous nerve (C7, C8)	Forms ulnar border of snuffbox. Its long tendon changes direction, hooking around dorsal tubercle radius (Lister's tubercle). Extends higher into the forearm and more distally into thumb than EPB
Extensor indicis proprius	Lower post. shaft of ulnar (below EPL) and adjacent interosseous membrane	Extensor expansion of index finger (tendon lies on ulnar side of ED tendon)	Extends all joints of index finger	Post interosseous nerve (C7, C8)	Tendon passes across lower end radius covered up by extensor digitorum. The indicis tendon is on the ulnar side of the digitorum tendon

CMC, carpometacarpal; ECR, extensor carpi radialis; ECRL, extensor carpi radialis longus; ED, extensor digitorum; EPL, extensor pollicis longus; IP, interphalangeal; MCP, metacarpophalangeal; PIN, posterior interosseous nerve.

Table 33.19 Muscles of the hand and wrist

Muscle	Origin	Insertion	Action	Innervation	Notes
Thenar muscles					
Abductor pollicis brevis	Tubercle of scaphoid and flexor retinaculum	Radial sesamoid of prox. phalanx of thumb and tendon of extensor pollicis longus	Abducts thumb at MCP and CMC joint	Recurrent (muscular branch of median nerve) (C8, T1), occasionally ulnar nerve	Most radial. Forms anterolateral part thenar eminence. Short abductor of the thumb
Opponens pollicis	Flexor retinaculum and tubercle of trapezium	Whole of radial border of first MC	Opposes (med. rotates and flexes) CMC and MCP joints of thumb	Recurrent (muscular branch of median nerve) (C8, T1), occasionally deep branch of ulnar nerve (T1)	Lies deep to APB and lat. to FPB
Flexor pollicis brevis	Flexor retinaculum and tubercle of trapezium	Base of prox. phalanx of thumb (via radial sesamoid)	Flexes MCP joint of thumb	Recurrent (muscular branch of median nerve) (C8, T1), occasionally deep branch of ulnar nerve (T1)	Lies to the ulnar (medial) side of APB
Adductor compartment					
Adductor pollicis	Oblique head: Base of second and third MCs, trapezoid and capitate. Transverse head: Palmar border and shaft of third MC	Ulnar sesamoid then ulnar side of base of proximal phalanx and tendon of extensor pollicis longus	Adducts CMC joint of thumb. Approximates the thumb to the index	Deep branch of ulnar nerve (T1)	Not classified as part of the thenar eminence. Lies deep in the palm

Table 33.19 (*cont.*)

Muscle	Origin	Insertion	Action	Innervation	Notes
Hypothenar muscles					
Abductor digiti minimi	Pisiform bone, pisohamate ligament and flexor retinaculum	Ulnar side of base of prox. phalanx of little finger and extensor expansion	Abducts little finger at MCP joint	Deep branch of ulnar nerve (T1)	Most superficial and lies medial
Hypothenar muscles					
Flexor digiti minimi brevis	Flexor retinaculum and hook of hamate	Ulnar side of base of prox. phalanx of little finger	Flexes MCP joint of little finger	Deep branch of ulnar nerve (C8, T1)	Lies lateral to ADM
Opponens digiti minimi	Flexor retinaculum and hook of hamate	Ulnar border of shaft of fifth MC	Opposes (flexes and lat. rotates) CMC and MCP joints of little finger	Deep branch of ulnar nerve (T1)	Lies deep. Acts exclusively on the CMC joint of the thumb
Palmaris brevis	Flexor retinaculum and palmar aponeurosis	Skin of palm into dermis	Steadies and corrugates skin of palm to help with grip	Superficial branch of ulnar nerve (C8, T1)	It is not part of the hypothenar eminence. Covers and protects ulna nerve and artery
Intrinsic muscles					
Lumbricals	FDP. Radial 2: Radial side only (unipennate). Ulnar 2: Cleft between tendons (bipennate)	Extensor expansion fingers 2–5	Flexes MCP joints and extends IP joints fingers	Lat 2: Median. Med 2: Deep branch of ulnar nerve	
Four dorsal interossei	Bipennate from adjacent MC shafts of the space where they arise	Prox. phalanx and dorsal extensor expansion on radial side of index and middle fingers and ulnar side of middle and ring fingers	Abducts from axis of middle finger. Flexes MCP joint whilst extending IP joint (DAB)	Deep branch ulnar nerve (T1)	Located between the metacarpals. More powerful than the palmar
Three palmar interossei	Unipennate from the middle finger side of index, ring and little finger MCs	Insert into the same side of the prox. phalanx and dorsal extensor expansion of each finger	Adducts to axis of middle finger. Flexes MCP joint whilst extending IP joint (palmar adducts)	Deep branch ulnar nerve (T1)	The middle finger has no palmar interosseous, it can not be adducted towards itself. Some books describe four palmar interossei, a few fibres passing from the base of thumb MC to base PP thumb

ADM, abductor digiti minimi; APB, abductor pollicis brevis; CMC, carpometacarpal; DAB, dorsal abducts; FDP, flexor digitorum profundus; FPB, flexor pollicis brevis; IP, interphalangeal; MC, metacarpal; MCP, metacarpophalangeal; PP, proximal phalanx.

The spinal accessory nerve supplies two muscles of the neck: The sternocleidomastoid and trapezius. It runs through the posterior triangle of the neck and can be injured during surgery at this location (lymph node biopsy) and causes medial winging of the scapula due to weakness of trapezius. It can also be injured together with injuries to the trunks of the brachial plexus.

Brachial plexus (Figures 33.17 and 33.18)

The brachial plexus is a network of nerves, running from the spine, formed by the ventral rami of the lower four cervical nerves and first thoracic nerve roots (C5–T1). After emerging from the spine it proceeds through the neck, the axilla and into the arm.

The brachial plexus can be divided into parts as shown below.

(Mnemonic: **R**ob **T**aylor **D**rinks **C**old **B**eer.)

- **Roots**

  The five roots are the five anterior rami of the spinal nerves
  The roots pass through the gap between anterior and middle scalene muscle
- **Trunks**

  The five roots merge to form three trunks arranged from top to bottom and, hence, their name. They lie in the posterior triangle of the neck
  1. Superior or upper trunk (C5–C6)
  2. Middle trunk (C7)
  3.Inferior or lower trunk (C8, T1)
- **Divisions**
  - Each trunk then splits in two (anterior and posterior divisions) to form a total of six divisions. Trunks lie behind the clavicle
- **Cords**

  These six divisions re-group to become the three cords. The cords are named by their position with respect to the axillary artery
  1. Lateral,
  2. Medial,
  3. Posterior (formed by all three posterior divisions)
- **Branches**

  Branches arise from the roots, trunks and cords. The cords themselves end as terminal branches

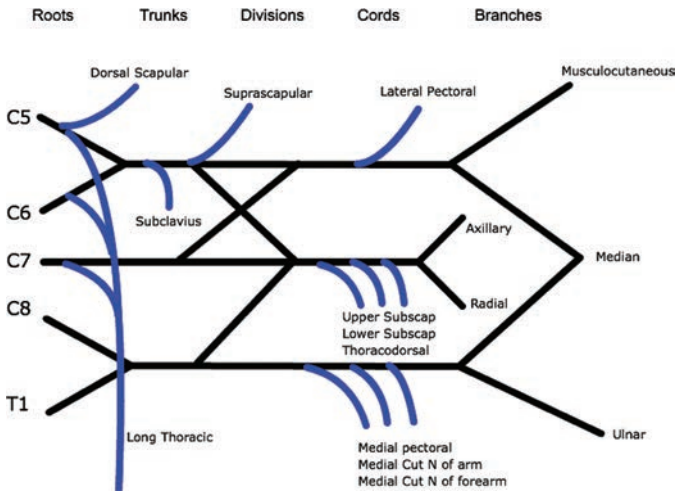

Figure 33.17 Brachial plexus

Supraclavicular branches (from roots and upper trunk) (Table 33.20)

From the roots

1. *Dorsal scapular nerve* (C5): Supplies rhomboid muscles and levator scapulae
2. *Long thoracic nerve* (C5, C6, C7): Supplies serratus anterior muscle (nerve damage causes medial scapula winging). Enters the axilla by passing down over the lateral border of the first rib behind the axillary vessels. Descends over the superficial surface of serratus anterior muscle
3. *Suprascapular nerve* (C5, C6): Supplies supraspinatus and infraspinatus muscles. Arises in the posterior triangle of the neck and runs downwards and laterally inferior to trapezius and omohyoid. Enters the suprascapular fossa by

Table 33.20 Supraclavicular branches (from roots and upper trunk)

ROOTS	TRUNKS	DIVISIONS	CORDS	BRANCHES
Dorsal scapular (C5)	Nerve to subclavius (C5, C6) Suprascapular nerve (C5, C6)		LATERAL CORD	Lateral pectoral nerve (C5, C6, C7) Musculocutaneous nerve (C5, C6, C7) Lateral head of median nerve
Long thoracic nerve (C5, C6, C7)			POSTERIOR CORD	Upper subscapular nerve (C5) Lower subscapular nerve (C6) Thoracodorsal nerve (C6, C7, C8) Axillary nerve (C5, C6) Radial nerve (C5, C6, C7, C8, T1)
			MEDIAL CORD	Medial pectoral nerve (C8, T1) Medial cutaneous nerve of arm (C8, T1) Medial cutaneous nerve of forearm (C8, T1) Medial root of median nerve (C8, T1) Ulnar nerve (C8, T1)

passing through the suprascapular notch beneath the superior transverse scapular ligament

Compression in the suprascapular notch causes wasting of supraspinatus and infraspinatus muscles. The suprascapular nerve also sends some filaments to supply the shoulder joint and capsule

From the upper trunk

1. *Nerve to subclavius* (C5, C6): Supplies subclavius. Descends posterior to clavicle and anterior to the brachial plexus and subclavian artery. May give a contribution to the phrenic nerve (C5), named the accessory phrenic nerve if present

Infraclavicular branches

From the lateral cord

1. *Lateral pectoral nerve* (C5, C6, C7): Supplies pectoralis major muscle
2. *Musculocutaneous nerve* (C5, C6, C7): Terminal branch of the lateral cord. It arises obliquely behind the lower fibres of pectoralis minor, lying lateral to the axillary artery and passes between the two conjoined heads of coracobrachialis. It runs laterally between biceps and brachialis adherent to the deep surface of biceps. It pierces the deep fascia, continuing on as the lateral cutaneous nerve of the forearm (lateral to the cephalic vein) supplying skin on the lateral aspect of the forearm

   Motor supply is to coracobrachialis, biceps brachii and brachialis. It is protected during the anterior (Henry's) approach to the shoulder by avoiding vigorous retraction of the conjoined tendon and avoiding dissection medial to the coracobrachialis muscle
3. *Lateral head of median nerve* (see median nerve)

From the posterior cord

1. *Upper subscapular nerve* (C5): Supplies the upper part of subscapularis
2. *Lower subscapular nerve* (C6): Supplies the lower part of subscapularis and teres major
3. *Thoracodorsal nerve* (C6, C7, C8): Supplies the latissimus dorsi muscle. It runs downwards on subscapularis accompanied by the subscapular vessels
4. *Axillary nerve* (C5, C6): Terminal branch of the posterior cord. The axillary nerve is just inferior to the shoulder capsule and it is protected during shoulder surgery by adduction and external rotation of the arm. The nerve is prone to injury during anterior dislocation of the shoulder owing to its close relationship with the inferior capsule. At the lower border of subscapularis it turns backwards and passes through the quadrangular space and then winds around the surgical neck of humerus with the posterior circumflex humeral vessels. After giving off a branch to the shoulder joint it divides into anterior and posterior branches. The anterior branch runs forward around the humerus in contact with the periosteum to enter the deep surface of deltoid. The posterior branch supplies teres minor and winds around the posterior border of deltoid. It ends as the upper lateral cutaneous nerve of the arm supplying the skin over the inferior half of the deltoid (regimental badge area)
5. *Radial nerve* (C5, C6, C7, C8, T1): The larger terminal branch of the posterior cord (largest branch of the brachial plexus). It crosses the lower border of the posterior axillary wall, lying on latissimus dorsi and teres major. It passes through a triangular space. It leaves the axilla and descends in the posterior compartment of the arm. It spirals around the humerus (medial to lateral) in the spiral groove between medial and lateral heads of triceps along with the profunda brachii artery. It pierces the lateral intermuscular septum at the midpoint of the humerus to reach the anterior compartment between the brachialis and brachioradialis. It crosses the anterior aspect of the lateral epicondyle (where it supplies anconeus) and enters the forearm, dividing into deep and superficial branches. At the elbow the radial nerve lies on the elbow capsule at the mid portion of the capitellum, making it prone to injury during arthroscopic capsular release

   It supplies:

   In axilla –

   - Long, medial and lateral heads of triceps
   - Posterior cutaneous nerve of arm (posterior upper arm skin)
   - Lower lateral cutaneous nerve of arm (lower lateral arm skin)

   In posterior compartment of the arm –

   - Motor to brachioradialis, brachialis (small supply), extensor carpi radialis longus
   - Posterior cutaneous nerve of forearm (posterior forearm skin)

   The radial nerve divides into a deep (posterior interosseous nerve – PIN) and superficial branch:

   - *Posterior interosseous nerve:* The PIN does not have a cutaneous branch and passes between the two heads of supinator to enter the posterior compartment. It reaches the interosseous membrane and ends on the back of the carpus. It supplies all the extensor muscles except extensor carpi radialis longus
   - *Superficial branch of radial nerve:* This runs over supinator, pronator teres and flexor digitorum superficialis (FDS) and lies under brachioradialis, running with the radial artery on its medial side from about half way down the forearm. It passes deep to brachialis, proximal to the radial styloid and over the tendons of the snuffbox to reach the dorsal radial aspect of the hand

From the medial cord

1. *Medial pectoral nerve* (C8, T1): arises from the medial cord behind the first part of the axillary artery and enters the deep surface of pectoralis minor. It perforates pectoralis minor to enter the pectoralis major muscle
2. *Medial cutaneous nerve of arm* (C8, T1): supplies anterior and medial aspects of the arm
3. *Medial cutaneous nerve of forearm* (C8, T1): supplies the medial aspect of the forearm
4. *Medial root of median nerve* (C8, T1) (see median nerve): crosses the axillary artery to join the lateral head
5. *Median nerve (C6, C7, C8, T1):* this arises in the lower axilla by two roots which clasp the axillary artery. The nerve initially lies anterior to the axillary artery then lateral to it. At the level of the mid-humerus the median nerve crosses the brachial artery, usually anteriorly to lie medial to the artery in the cubital fossa

   It lies on coracobrachialis and then brachialis. It passes beneath the bicipital aponeurosis at the elbow, leaving the cubital fossa between the two heads of pronator teres. It descends deep to the flexor digitorum superficialis on flexor digitorum profundus. Near the wrist it becomes superficial, passing between FDS and flexor carpi radialis. It travels deep to the palmaris longus tendon and enters the carpal tunnel where it divides into a lateral and medial branch. The lateral branch gives off the recurrent (muscular) branch and then breaks up into three palmar digital nerves, two for the thumb and one for the index, the last one also supplying the first lumbrical. The medial branch divides into two common palmar digital nerves for the index, middle and radial half of ring finger and the last one also supplies the second lumbrical. Palmar digital nerves supply not only the whole palmar aspect of the finger but also the distal half of the dorsal aspect of each finger as well

   The median nerve does not give off any branches in the arm

   It supplies all the flexor muscles of the forearm except flexor carpi ulnaris and the medial half of flexor digitorum profundus (ulnar nerve). It also supplies the thenar eminence and the first and second lumbrical muscles

   - *Anterior interosseous nerve:* This arises just below the two heads of pronator teres to run on the interosseous membrane between flexor digitorum profundus and flexor pollicis longus to reach pronator quadratus. It supplies these muscles, except for the medial half of flexor digitorum profundus. It also sends an articular branch to the wrist joint. This nerve does not have a cutaneous branch. Anterior interosseus nerve palsy presents principally as weakness of the thumb and index finger (the 'OK' sign)
   - *Palmar cutaneous branch of the median nerve:* This arises just proximal to flexor retinaculum and becomes cutaneous between palmaris longus and flexor carpi radialis. It passes superficial to the flexor retinaculum to supply the lateral aspect of the palm (skin). It helps to determine the level of a median nerve injury; numbness over the thenar eminence may indicate a high lesion, whilst intact sensation with loss of function in the recurrent and palmar digital branches may indicate a more distal lesion, e.g. carpal tunnel (this is not entirely reliable owing to the anatomical variations)
   - *Recurrent motor (thenar) branch of the median nerve:* This supplies abductor pollicis brevis, flexor pollicis brevis and opponens pollicis. It loops around the distal border of flexor retinaculum and enters the thenar muscles
6. *Ulnar nerve (C8, T1):* The ulnar nerve arises medial to the axillary artery and continues medial to the brachial artery, lying on coracobrachialis to the midpoint of the humerus where it leaves the anterior compartment by passing posteriorly through the medial intermuscular septum with the superior ulnar collateral artery to enter the posterior compartment of the arm. It runs anterior to the medial head of triceps, passing posterior to the medial humeral epicondyle to enter the forearm between the two heads of flexor carpi ulnaris

   It runs deep to flexor carpi ulnaris on the flexor digitorum profundus with the ulnar artery on its lateral side from one-third of the way down the forearm

   In the distal forearm it becomes superficially covered only by fascia and skin passing superficial to the flexor retinaculum with the ulnar artery. It then passes lateral to the pisiform and grooves the hook of the hamate. The deep branch of the ulnar nerve pierces between abductor digiti minimi and the flexor digiti minimi brevis

Branches of the ulnar nerve:

- No branches in the arm
- Articular branches to elbow joint
- Flexor carpi ulnaris and medial half of flexor digitorum profundus
- Palmar cutaneous branch (skin over hypothenar eminence)
- Dorsal cutaneous branch (medial skin dorsum of hand and 1½ digits)
- Dorsal cutaneous branch arises 5 cm proximal to the wrist, passes deep to flexor carpi ulnaris (FCU) onto the medial aspect of the dorsum of the hand
- Superficial branch of the ulnar nerve: Supplies palmaris brevis and then carries on as the superficial palmar branch to innervate the palmar 1½ digits
- Deep branch of the ulnar nerve: Supplies wrist joint, flexor digiti minimi, abductor digiti minimi, opponens digiti minimi, palmar and dorsal interossei, two medial lumbricals, adductor pollicis and deep head of flexor pollicis brevis

Anomalies

Martin Gruber anastomosis (~17% individuals).

Branches from the median to the ulnar nerve in the forearm: Two patterns:

1. Median nerve in proximal forearm to ulnar nerve in middle/distal third of forearm
2. Anterior interosseus nerve to ulnar nerve

   This results in the median nerve innervating a variable number of intrinsic muscles of the hand:

   - First dorsal interosseus: Most common
   - Others: Adductor pollicis, abductor digiti minimi

   Often bilateral: 60–70%:

   Riche–Cannieu anastomosis

   Connections between deep ulnar and median nerves in the hand

Guyon's canal

Fibrosseous tunnel approximately 4 cm long. Boundries:

- Proximal: Proximal edge of transverse carpal ligament at pisiform bone
- Distal: Aponeurotic arch of origin of hypothenar muscles
- Medial wall: Pisiform and fibrous attachments of pisohamate ligament
- Lateral wall: Hook of hamate
- Roof: Volar carpal ligament

The ulnar nerve divides within the canal into superficial (more ulnar) and deep (more radial) branches at the level of the pisiform.

There are four sites of compression:

1. Guyon's canal – Sensory loss of the palmar 1½ digits. Spared dorsal medial hand and fingers (dorsal cutaneous branch) and proximal ulnar palm (palmar cutaneous branch). Motor: All ulnar hand muscles
2. Distal to Guyon's canal (proximal deep terminal branch) (pure motor). Motor: All ulnar hand muscles. Sensory: Normal
3. Hook of hamate (distal deep terminal branch) (pure motor). Motor: Spares hypothenar muscles. Sensory: Normal
4. Superficial terminal branch – Sensory loss of the palmar 1½ digits. Motor: Normal

Upper limb arteries

Subclavian artery

The subclavian vessels follow an oblique course along the length of the clavicle, beginning posterosuperiorly to it at its medial end and eventually dipping down into the axilla inferior to it at the lateral end. Upon crossing the lateral border of the first rib the vessels continue onward as the axillary vessels.

The risk of iatrogenic injury to the subclavian vessels during the use of a plate and screws during osteosynthesis of the clavicle is well known.

The clavicle has been clinically divided into three zones based on the proximity and orientation of vessels in relation to clavicle[1].

In the medial third part of clavicle the subclavian vessels are situated behind the clavicle. The vein is intimately related to the clavicle, whereas the artery is somewhat protected by the intervening scalenus anterior muscle. In the middle third the subclavian vessels pass laterally and begin their descent towards the axilla, taking up a position posteroinferior to the clavicle.

At the lateral end of the clavicle the vessels descend more acutely within the axilla and are placed inferior and pass underneath the coracoid process making it safer.

Scapular anastomosis

The dorsal scapular, suprascapular and descending branch of the superficial cervical artery arise from the first part of the subclavian artery. The subscapular and its dorsal branch the circumflex scapular artery form the third part of the axillary artery. This arterial anastomosis provides a collateral circulation in case of obstruction of the subclavian artery. There are various sites of anastomosis including the infraspinous fossa, medial border of the scapula, surgical neck of the humerus, thoracic walls and acromion.

The circumflex humeral arteries encircle the surgical neck of the humerus, anastomosing with each other. The smaller anterior circumflex humeral artery passes laterally, deep to coracobrachialis and biceps. The larger posterior circumflex humeral artery passes medially through the posterior wall of the axilla in the quadrangular space with the axillary nerve.

Axillary artery

This is a continuation of the subclavian artery. It begins at the lateral border of the first rib and ends at the inferior border of teres major to become the brachial artery. It is divided into three parts by pectoralis minor:

- First part: Between the lateral border of the first rib and the medial border of pectoralis minor
- Second part: Lies posterior to pectoralis minor
- Third part: Lateral border of pectoralis minor to the inferior border teres major, three branches
  - Subscapular artery,
  - Anterior circumflex humeral artery,
  - Posterior circumflex humeral artery

Brachial artery

This begins as a continuation of the axillary artery at the lower border of teres major and ends just distal to the elbow joint, dividing into radial and ulnar arteries. In the proximal arm the brachial artery lies medially but spirals laterally to take up a position midway between both epicondyles of the humerus. It crosses anterior to the elbow joint medial to biceps tendon.

It is superficial throughout its whole course, accompanied by venae comitantes. It is crossed obliquely from lateral to medial by the median nerve in the mid arm. Medial and posterior to the artery is the ulnar nerve, which leaves it in the lower part of the arm to pass through the medial intermuscular septum. The artery can be exposed in the groove at the medial border of biceps between biceps and triceps.

Branches:

- Profunda brachii
- Muscular
- Nutrient to the humerus
- Superior ulnar collateral
- Inferior ulnar collateral: Divides into anterior and posterior branches

Radial artery

This is the terminal branch of the brachial artery. It crosses anterior to the biceps tendon to lie on supinator. It then passes down the lateral side of the forearm, lying on pronator teres, radial head of FDS, FPL and pronator quadratus deep to brachioradialis and FCR. The radial artery then passes beneath abductor pollicis longus and extensor pollicis brevis to enter the anatomical snuffbox.

It passes over the floor of the anatomical snuffbox and between the two heads of the first dorsal interosseous muscle and then between the two heads of adductor pollicis to enter the deep plane of the palm and form the *deep palmar arch* with the deep branch of the ulnar artery.

Two arteries, the princeps pollicis artery and the radialis indicis artery, arise from the radial artery between the first dorsal interosseous and adductor pollicis. The princeps pollicis artery is the major blood supply to the thumb and the radialis indicis artery supplies the lateral side of the index finger.

The deep palmar branch gives off:

- Three palmar metacarpal arteries which, at the metacarpal heads, join the common palmar digital arteries from the superficial palmar arch
- Three perforating branches, which anastomose with the dorsal metacarpal arteries from the ulnar artery

Ulnar artery

This is the terminal branch of the brachial artery, arising in the cubital fossa. It leaves the fossa deep to the deep head of pronator teres and deep to the fibrous arch of FDS just lateral to the median nerve to cross beneath the nerve, before travelling down the medial side of the forearm.

The artery enters the wrist lateral to the ulnar nerve and the pisiform bone. Distally, the artery is medial to the hook of hamate and swings laterally across the palm, forming the *superficial palmar arch*. The arch is superficial to everything in the palmar compartment in contact with the deep surface of the palmar aponeurosis. When complete, it communicates with the superficial palmar branch of the radial artery.

Hand

The blood supply of the hand is from the radial and ulnar arteries, which form two, interconnected vascular arches (superficial and deep) in the palm. The deep palmar arch lies 1 cm proximal to the superficial palmar arch.

Lower limb osteology and joints

Pelvis

The two hip bones are joined anteriorly at the pubic symphysis by a fibrous cartilage covered by a hyaline cartilage, the interpubic disk, within which a non-synovial cavity might be present. Two ligaments, the superior and inferior pubic ligaments, reinforce the symphysis.

Both sacroiliac joints, formed between the auricular surfaces of the sacrum and the two hip bones are amphiarthroses; almost immobile joints enclosed by very taut joint capsules. This capsule is strengthened by the ventral, interosseous, and dorsal sacroiliac ligaments. The most important accessory ligaments of the sacroiliac joint are the sacrospinous and sacrotuberous ligaments which stabilise the hip bone on the sacrum and prevent the promonotory from tilting forward.

Additionally, these two ligaments transform the greater and lesser sciatic notches into the greater and lesser foramina, a pair of important pelvic openings. The iliolumbar ligament is a strong ligament which connects the tip of the transverse process of the fifth lumbar vertebra to the posterior part of the inner lip of the iliac crest. It can be thought of as the lower border of the thoracolumbar fascia.

1. Anterior sacroiliac ligament
   This is a thin band that connects the anterior surface of the lateral part of the sacrum to the auricular surface of the ilium and preauricular sulcus.
2. Posterior sacroiliac ligaments
   These are very strong ligaments forming the main bond of union between bones. They are divided into short (intrinsic) and long (extrinsic) ligaments. The short posterior sacroiliac ligament passes from the first and second transverse tubercles of the sacrum to the tuberosity of the ilium. The long posterior sacroiliac ligament, which is oblique in direction, attaches from the third transverse tubercle of the sacrum to the posterior superior iliac spine (PSIS).
3. Sacrotuberous ligament
   This is flat and triangular, attaches to posterior border of the ilium and the posterior superior and posterior inferior iliac spines, to the transverse tubercles of the sacrum below the articular surface, and to the upper part of the coccyx. Passing obliquely downward, forward and lateral, it becomes narrow and thick, to insert into the thinner margin of the ischial tuberosity.

Table 33.21 Ligaments of hip joint

Ligaments	Function
Iliofemoral, (Y-shaped, strongest)	Prevents excessive adduction and internal rotation. Prevents the trunk from falling backward without the need for muscular activity in standing position
Ischiofemoral,	Restricts internal rotation
Pubofemoral	Restricts abduction and internal rotation
Ligamentum teres (intra-articular)	Blood supply through foveal artery

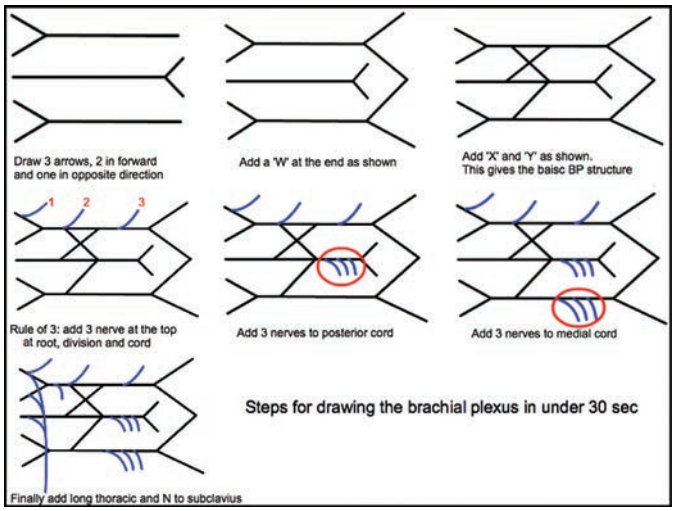

Figure 33.18 Steps to draw brachial plexus

4. Sacrospinous ligament
   This lies on the pelvic aspect of the sacrotuberous ligament. It attaches to the side of the lower part of the sacrum and the upper part of the coccyx. The apex is attached to the spine of the ischium.
5. Sacrotuberous and sacrospinous ligaments
   The sacrotuberous and sacrospinous ligaments, with the lesser sciatic notch of the ischium, enclose the lesser sciatic foramen, whose lateral part is occupied by the obturator internus.
6. Iliolumbar ligament
   This is a V-shaped ligament. The apex is attached to the transverse process of the L5 vertebra. The upper band of the base passes to the iliac crest and the lower band runs laterally and downwards to blend with the front of the anterior sacroiliac ligament.

Hip joint (Figures 33.19 and 33.20; Table 33.21)

The hip joint is a synovial joint formed by the articulation of a roughly spherical femoral head contained in the acetabulum.

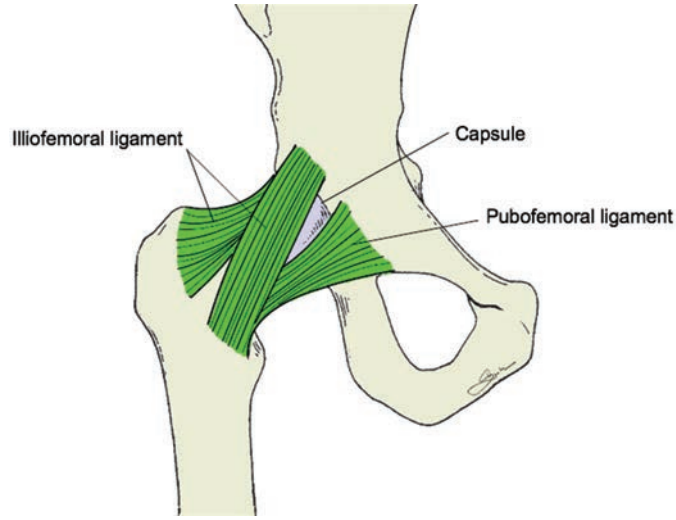

Figure 33.19 The hip joint ligaments

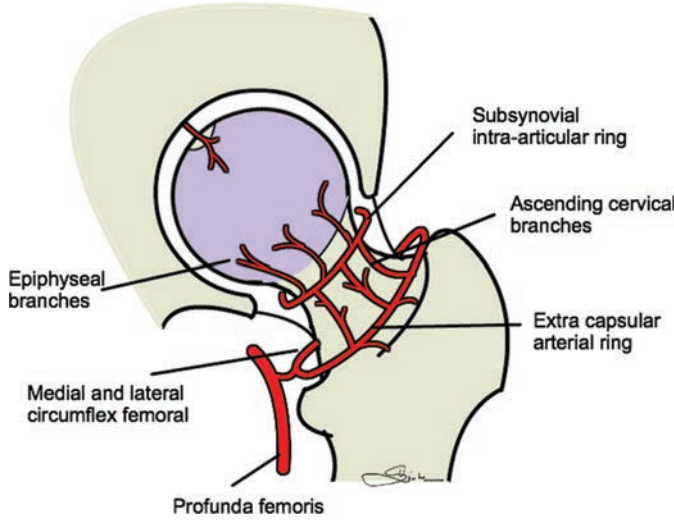

Figure 33.20 The blood supply of femoral head

The acetabular labrum further extends beyond the equator increasing this containment.

The capsule of hip joint is attached to the acetabulum outside the labrum making it an intra-articular structure. It is attached to the femoral neck and allows for large range of movement in the joint.

The circular fibers of the capsule form a collar around the femoral neck called the zona orbicularis. The longitudinal retinacular fibers in the capsule travel along the neck and carry blood vessels.

Ligaments of hip joint (Figure 33.19)

Blood supply to femoral head (Figure 33.20)

The blood supply to the femoral head comes from the medial and lateral circumflex femoral arteries (MCF and LCF) which are both branches of the deep/profunda femoris artery (branch of femoral artery). There are minor contributions from the superior and inferior gluteal arteries.

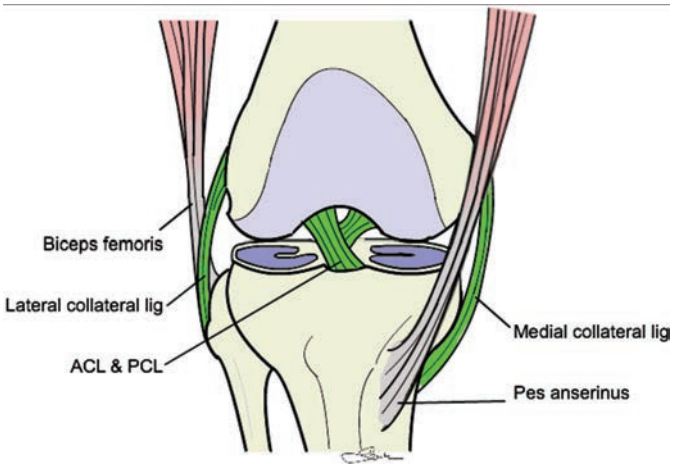

Figure 33.21 The ligaments of knee joint and pes anserinus

Branches from the MCF and LCF anastomose to form an extra capsular arterial ring. This gives rise to ascending cervical branches, which travel up the capsule of hip joint as retinacular arteries. The retinacular arteries anastomose again to form a subsynovial intra articular ring, which gives off epiphyseal arterial branches that pierce and supply the femoral head.

There are minor contributions from the artery of ligamentum teres (foveal artery), a branch of obturator artery.

Knee joint (Figure 33.21)

The knee is the largest synovial joint in the body. It is enclosed by a capsule which attaches about 15 mm distal to the articular surface of the tibia. The patella that articulates with the femoral condyle is the largest sesamoid bone in the body.

The vascular supply to the knee is mainly through an anastomosis formed by:

- The descending and genicular branches of the femoral, popliteal and lateral circumflex femoral arteries from above (the thigh region)
- Circumflex fibular artery and branches of the anterior tibial artery from below (the leg)

It is innervated by branches from the obturator, femoral, tibial and common fibular nerves.

Menisci

The menisci serve to deepen the concavity of the tibial side of the joint and, therefore, provide some stability. Other functions include protection and assisting in rotation of the knee. They are avascular except in the peripheral third.

Medial meniscus:

This is a 'C'shaped structure which is anchored to the tibia via the meniscotibial (coronary) ligaments.

- The anterior part is attached just below the tibial articular surface. Anteriorly it also connects with the lateral meniscus via a transverse ligament
- In the mid portion it is attached to the deep fibres of the MCL
- Posteriorly, it is anchored to the posterior intercondylar area

Lateral meniscus:

Compared to the medial meniscus the lateral meniscus is more 'O' shaped, is smaller and more mobile. There is no attachment to the adjacent LCL.

Posteriorly it is attached in the intercondylar area to the medial femoral condyle via the ligaments of Humphry (anterior menisco-femoral ligament) and Wrisberg (posterior menisco-femoral ligament).

Stability of the knee joint

Stability is provided mainly by the cruciate and collateral ligaments. The patella stability is mainly through the medial patellofemoral ligament.

1. Medial collateral ligament (MCL)
   This is a flat triangular band composed of two portions, superficial and deep.
   The *superficial MCL* proximally is attached to the posterior aspect of the medial femoral condyle. This attachment is a few millimeters proximal and posterior to the medial epicondyle. Distally it is attached to the metaphyseal region of the tibia, about four fingers-breath below the joint, lying beneath the pes anserinus. The superficial MCL can be divided into anterior and posterior portions. The posterior part forms the posterior oblique ligament.
   The superficial MCL is the primary restraint to valgus force.
   The *deep MCL* is divided into menisco-femoral and menisco-tibial ligaments and inserts directly into the edge of the tibial plateau and meniscus
2. Lateral collateral ligament (LCL)
   This is cord-like, attached superiorly to the lateral femoral epicondyle (below the lateral head of gastrocnemius and above the popliteus). Inferiorly, it attaches to the head of the fibula and is overlapped by the biceps femoris. It lies free from the capsule and lateral meniscus, being separated from the meniscus by the tendon of popliteus inside the joint and the inferior lateral genicular vessels outside the joint.
   The cruciate ligaments lie within the capsule (but not synovial membrane) of the knee joint.
3. The anterior cruciate ligament (ACL)
   Attaches to the anterior part of the tibial plateau between the attachment of anterior horns of medial and lateral meniscus. The ACL ascends posterolaterally, twisting on itself, to attach to the posteromedial aspect of the lateral femoral condyle. The ACL is made up two bundles , the anteromedial and posterolateral.
   The anteromedial bundle is tight in flexion and the posterolateral is tight in extension
4. The posterior cruciate ligament (PCL)
   Compared to the ACL, the PCL is stronger, shorter, broader and less oblique. It attaches to a small impression

at the posterior part of the intercondylar area of the tibia. It lies between the ligaments of Humphrey and Wisberg. It ascends anteromedially to attach to the anterolateral aspect of the medial femoral condyle. The PCL has an anterolateral portion that is tight in flexion and a posteromedial portion that is tight in extension.

5. Posterolateral corner (PLC)
   This PLC complex comprises of static and dynamic components. The static stabilisers are: The arcuate ligament, popliteus tendon, posterolateral capsule, lateral collateral ligament, the fabello-fibular ligament and the politeofibular ligament. The dynamic component consists of the iliotibial band, the biceps femoris and the lateral head of the gastrocnemius.
6. Anterolateral ligament (ALL)
   This is a term used to describe the soft-tissue stabilising structures in the anterolateral region of the knee. Recent studies have described this structure as passing anterodistally from an attachment proximal and posterior to the lateral femoral epicondyle to the margin of the lateral tibial plateau, approximately midway between Gerdy's tubercle and the head of the fibula[2]. The ALL has been has been implicated as being the cause of the 'segond fracture'. It is also thought to be injured frequently together with the ACL.
7. Medial patellofemoral ligament (MPFL)
   The medial patellar retinaculum inserts into the upper two-thirds of the medial margin of the patella. Within the middle layer out of three, two distinct condensations of fibres are classically described. The medial patellotibial ligament(MPTL) which inserts into the medial meniscus and tibia and, more importantly, the MPFL, which inserts into a point 10 mm posterior and proximal to the medial femoral epicondyle (although some authors say it attaches to the medial epicondyle). From here it attaches to the upper third of the patella. On the patella the midpoint is located 40% of the distance from the proximal tip of the patella. The upper extent can be identified by the way it blends with the distal insertion of the VMO into the patella. The lower extent of the MPFL is a distinct thickening within the fascial layer[3].

   The MPFL provides the major restraining force to lateral patellar dislocation when the knee is between 0° and 30° of flexion.

Pes anserinus (Figure 33.21)

Pes anserinus refers to the conjoined tendons of three muscles sartorius, gracilis and semitendinosus that insert onto the anteromedial surface of the proximal tibia. The appearance of the insertion looks like 'goose foot' and, hence, the name.

Popliteal fossa

A diamond-shaped depression behind the knee joint.

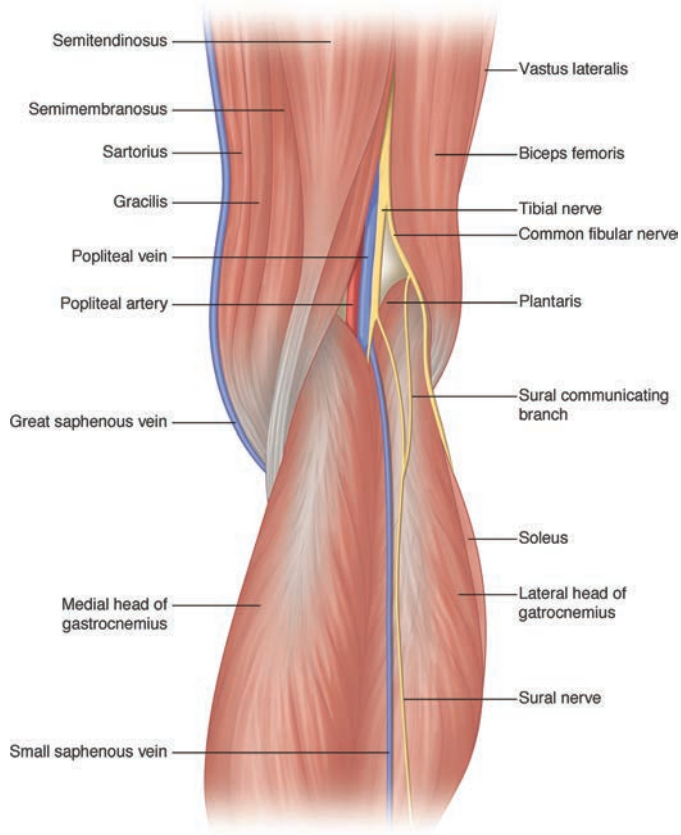

Figure 33.22 Popliteal fossa

Boundaries (Figure 33.22)

- Superior/lateral – Biceps femoris; superior/medial – Semitendinosus and semimembranosus; inferior/medial and lateral – Medial and lateral heads of gastrocnemius
- Roof: Deep fascia of the thigh
- Floor: From above downwards – Popliteal surface of femur, posterior capsule of the knee joint and popliteus muscle

Contents

Two nerves (tibial and common peroneal nerve), one vein (popliteal vein) and one artery (popliteal artery) pass through the fossa. The tibial nerve is most superficial and the popliteal artery deepest, with the popliteal vein in between.

The tibial nerve runs vertically down the middle of the fossa and disappears by passing between the heads of gastrocnemius. The nerve enters the calf by passing beneath the fibrous arch in the origin of soleus. The common peroneal nerve runs downwards and laterally, medial to the biceps tendon, and disappears into the substance of peroneus longus to lie on the neck of the fibula. It ends by dividing into deep and superficial peroneal nerves.

Ankle joint

This is a synovial joint which is usually described as a hinge joint but its movements are not quite that of a hinge as the axis

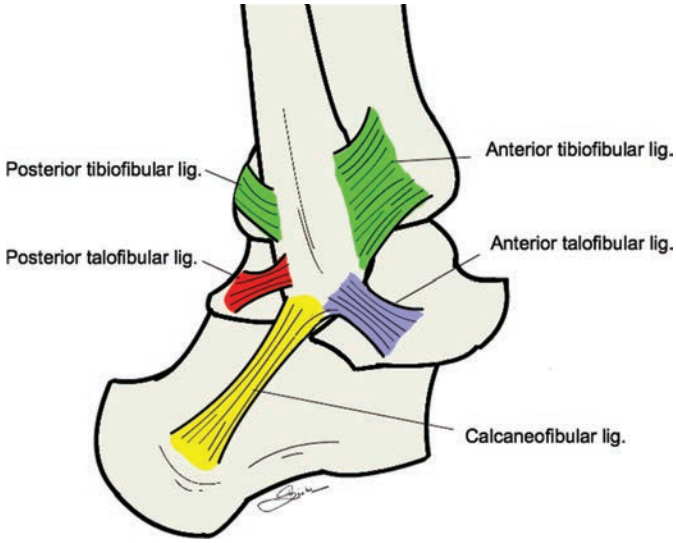

Figure 33.23 The lateral ligament complex of ankle joint

of rotation is not fixed but changes between the extremes of plantar flexion and dorsiflexion.

The stabilising surfaces are those of the medial and lateral malleoli, which grip the sides of the talus. The capsule is attached to the articular margins off all three bones except the anterior part of the talus where it is fixed some distance away from the articular margin at the neck of the talus.

Ankle ligaments (Figures 33.23 and 33.24)

The *deltoid (medial) ligament* consists of two layers:

- The deep part is a narrow rectangular band extending from the medial malleolus to the side of the talus (anterior and posterior tibiotalar ligaments)
- The superficial part is triangular-shaped and fans downwards from the borders of the medial malleolus to a continuous attachment on the sustentaculum tali and spring ligament (very strong) to the tuberosity of the navicular (weak).(tibionavicular and tibiocalcaneal)

The *lateral ligament* consists of three separate bands radiating from the lateral malleolus. The anterior (anterior talofibular ligament, ATFL) and posterior bands (posterior talofibular ligament) pass to the talus, and the intermediate band to the calcaneus (calcaneofibular).

The ATFL is the weakest and is intracapsular.

Foot

Osteology and blood supply of the talus

The head has a large distal articular surface, which is ovoid, convex and directed forwards and medially. Its inferior surface has two facets. The medial is for the plantar calcaneonavicular ligament. The lateral facet articulates with the corresponding facet on the upper surface of the anterior part of the calcaneus.

The inferior surface of the body has two articular surfaces, which are separated by a smooth ridge. The posterior calcaneal

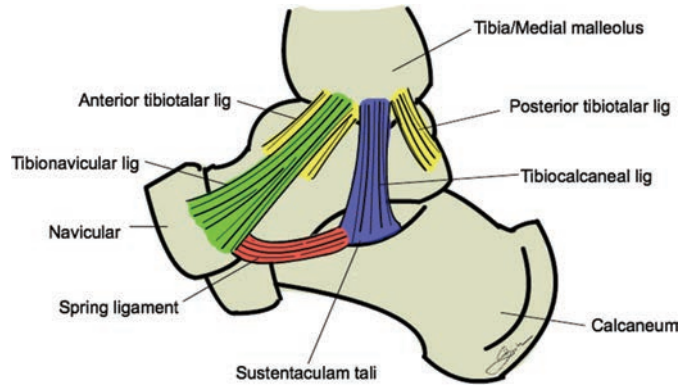

Figure 33.24 The medial ligament complex of ankle joint

surface is large and oval, articulating with the corresponding facet on the upper surface of the calcaneus. The middle calcaneal surface is small, oval and slightly convex, resting on the sustentaculum tali of the calcaneum.

The upper surface of the body carries an articular area, the trochlea, that is convex from front to back but with a shallow central groove. The trochlea is broad in front and narrow behind. The trochlear surface is continued down over each side of the body for articulation with the malleoli. Behind the trochlea the talus is projected into a posterior process, which is grooved, by flexor hallucis longus.

The inferior surface of the neck is grooved to fit over the corresponding groove on the calcaneum to make the tarsal sinus and gives attachment to the talocalcaneum ligament. Laterally the neck gives attachment to the cervical ligament and the anterior talofibular ligament. The neck of the talus is very short and is directed forwards and medially.

The talar *blood supply* is from intraosseous and extraosseous sources.

The main blood supply is the 'artery of the tarsal canal' arising from the posterior tibial artery 1 cm proximal to the division into the medial and lateral plantar arteries. It passes anteriorly between FDL and FHL to enter the tarsal canal and continues to the lateral part of the tarsus sinus, where it anastomoses with the artery of the tarsal sinus, forming a vascular sling under the talar neck.

A branch of the artery of the tarsal canal known as the deltoid branch passes deep to the deltoid ligament and supplies the medial aspect of the talar body. Occasionally this artery arises from the posterior tibial artery or medial plantar artery. In talar fractures it may be the only remaining arterial supply to the talus.

The dorsalis pedis supplies the superior aspect of the talar neck and gives off the artery of the tarsal sinus.

The peroneal artery gives off small branches, which form a plexus posteriorly with branches of the posterior tibial artery.

The intraosseous blood supply of the talar head comes medially from the dorsalis pedis artery and laterally from the anastomoses between the artery of the tarsal canal and tarsal sinus. The middle and lateral third of the talar body is supplied

Table 33.22 Ligaments in the foot

Ligament	Usual name	Origin	Insertion
Interosseous talocalcaneal	Cervical	Talus	Calcaneum
Calcaneocuboid/calcaneonavicular	Bifurcate	Calcaneum	Cuboid and navicular
Plantar calcaneocuboid	Short plantar	Calcaneus	Cuboid
Calcaneocuboid–metatarsal	Long plantar	Calcaneum	Cuboid and first to fifth metatarsal bones
Plantar calcaneonavicular	Spring	Sustentaculum tali	Navicular
Tarsometatarsal	Lisfranc	Medial cuneiform	second metatarsal base

Table 33.23 Flexors of the hip

Muscle	Origin	Insertion	Action	Innervation	Notes
Iliacus	Hollow of the iliac fossa	Lowermost surface of lesser trochanter of femur	Flexes and med. rotates hip	Femoral nerve (L2, L3) in the iliac fossa	Enters thigh beneath lat. part inguinal ligament where femoral nerve lies on it
Psoas	Transverse processes of L1–5, bodies of T12–L5 and inververtebral discs below bodies of T12–L4	Middle surface of lesser trochanter of femur	Flexes and med. rotates hip	Ant. Primary rami of L1, L2, L3 (mainly L2)	Enters thigh beneath middle part inguinal ligament where femoral artery lies on it
Pectineus	Pectineal line of pubis and narrow area of sup. pubic ramus below it	A vertical line between spiral line and gluteal crest below lesser trochanter of femur	Flexes, adducts and medially rotates hip	Ant. division of femoral nerve (L2, L3). May receive branch from obturator nerve	
Sartorius	Immediately below ASIS	Upper med. surface of shaft of tibia	Flexes, abducts, lat. rotates thigh at hip. Flexes, med. rotates leg at knee	Ant. division of femoral nerve (L2–L4)	A long strap-like muscle which in its upper part forms lat. boundary of femoral triangle. The oblique line of sartorius passing downwards and medially separates the adductor muscles of the hip superomedially from the extensor muscles of the knee inferolaterally

by this anastomotic sling in the tarsal canal. The medial third of the talar body is supplied by the deltoid artery, a branch of the artery of the tarsal canal.

Sinus tarsi

The sinus tarsi is an anatomical space bounded by the talus and calcaneum, the talocalcaneonavicular joint anteriorly and posterior facet of the subtalar joint posteriorly. It is medially continuous with the much narrower tarsal canal. The sinus tarsi contains the cervical ligament and the three roots of the inferior extensor retinaculum.

The tarsal canal contains the interosseous talocalcaneal ligament and the deep and intermediate roots of the inferior extensor retinaculum. Both the sinus and the canal contain nerves and blood vessels, which are important for the nutrition of the talus. The extensor digitorum brevis and bifurcate ligament lie anterior to the sinus tarsi.

Ligaments in the foot

Stability between the bones in the foot is provided by the shape of the bones articulating with each other as well as specific strong ligaments. The more important ones are listed in Table 33.22.

Lower limb muscles

Pelvis and thigh muscles (Table 33.23)

Piriformis is the key muscle of the gluteal region. A number of structures pass from the pelvis through the greater sciatic

notch into the gluteal region either above or below the piriformis muscle. Above the upper border emerge the superior gluteal nerve and vessels. Below the lower border emerge the inferior gluteal nerve and vessels, pudendal nerve and vessels, nerve to obturator internus, sciatic nerve with the posterior femoral cutaneous nerve on its surface, and the nerve to quadratus femoris deep to it.

Gluteal muscles/abductors and external rotators of the hip (Table 33.24)

The muscles of the gluteal region are arranged into superficial and deep groups. (Figure 33.25). The superficial group consists of three large overlapping glutei (maximus, medius and minimus) and tensor fascia lata. These muscles have proximal attachments to the external surface and margins of the ilium. The deep group consist of smaller muscles (piriformis, obturator internus, superior and inferior gemelli, and quadratus femoris) covered by the inferior half of gluteus maximus. They have distal attachments on or nearby the intertrochanteric crest of the femur.

The obturator internus and gemelli form a tricipital (three-headed) muscle (triceps of the hip), which occupies the gap between the piriformis and quadriceps femoris. The combined tendon lies horizontal in the buttock as it passes to the greater trochanter.

Medial thigh muscles/adductors of hip (Table 33.25)

These are arranged in three vertical layers: Anteriorly, pectineus and adductor longus are in front of adductor brevis, which separates them from the posteriorly placed adductor magnus. Gracilis lies superficial to these on the medial side of the thigh.

In general they attach to the anteroinferior surface of the bony pelvis and distally to the linea aspera of the femur.

Anterior thigh muscles/extensors of the knee (Table 33.26)

The quadriceps femoris (four-headed femoral muscle) forms the main bulk of the anterior thigh muscle. The tendons of the four parts unite in the distal part of the thigh to form the quadriceps tendon.

Rectus femoris acts at both the hip and knee joint, whilst the vasteralis muscles act only at the knee joint.

Posterior thigh muscles (Table 33.27)

The hamstring muscles share common features:

- A proximal attachment to the ischial tuberosity deep to the gluteus maximus
- A distal attachment to the bones of the leg
- Span two joints, producing extension at the hip joint and flexion at the knee joint

The two semi muscles are inserted medially and the two heads of biceps laterally into the upper part of the leg.

Muscles of the compartments of the leg (Figure 33.26)

Anterior

This is located anterior to the interosseous membrane between the lateral surface of the tibial shaft and the medial surface of the fibula shaft and anterior to the intermuscular septum that connects them.

The tendons along with the neurovascular bundle pass under the extensor retinaculum on the dorsum of ankle and their anatomical location and relationships have been tested frequently (Figure 33.27).

Lateral (Table 33.29)

The smallest (narrowest) of the leg compartments. It is bounded by the lateral surface of the fibula, the anterior and posterior intermuscular septa and the deep fascia of the leg. The compartment ends inferiorly at the superior fibular retinaculum. It contains two evertors of the foot (peroneus longus and peroneus brevis).

Superficial posterior (Table 33.30)

The posterior compartment of the leg is subdivided by the transverse intermuscular septum into superficial and deep compartments. Gastrocnemius and soleus make up the three-headed triceps surae and share a common insertion into the calcaneum.

Deep posterior (Table 33.31)

This consists of four muscles. The popliteus acts on the knee joint, whereas the other muscles plantarflex the ankle, with two continuing on to flex the toes. The two muscles that pass to the toes are criss-crossed; the muscle attaching to the great toe, flexor hallucis longus (FHL), arises laterally from the fibula while the muscle attaching to the lateral four toes, flexor digitorum longus (FDL), arises medially.

Muscles of the ankle and foot (Table 33.32)

There are two neurovascular planes on the plantar aspect of the foot, a superficial one between the first and second layers (plantar arteries and nerves) and a deep one between the third and fourth layers. Despite individual actions, the intrinsic muscles of the sole of the foot are to maintain the arch of the foot.

The second foot layer consists of the long flexor tendons and their connections in the sole. The third layer consists of three muscles like the first layer but they are broader, shorter, and confined to the metatarsal (MT) region of the foot. Two act on the big toe, one on the little toe. The fourth layer consists of the interossei in the intermetatarsal spaces. Interossei have a similar arrangement to the hand but the longitudinal axis of the foot has shifted preaxially and lies along the second MT bone.

Table 33.24 Gluteal muscles/abductors and external rotators of the hip

Muscle	Origin	Insertion	Action	Innervation	Notes
Gluteus maximus	Gluteal (outer) surface of ilium behind post. gluteal line and post. third of iliac crest, sacrum and coccyx, sacrotuberous ligament	Upper fibres: Ant. surface lat. condyle tibia; lowermost fibres : Gluteal tuberosity of femur	Extends and laterally rotates hip. Maintains knee extended via iliotibial band. Assists in rising from a sitting position	Inferior gluteal nerve (L5, S1, S2)	Largest, heaviest muscle in body. Large flat quadrilateral mass forming prominence of buttock
Piriformis	Middle three costotransverse bars of the ant. sacrum. Few fibres from sup. border of greater sciatic notch	Ant. part of med. aspect of greater trochanter of femur	Lat. rotates and stabilises the hip	Ant. primary rami of S1–S2	Underneath gluteus max
Obturator internus	Inner surface of obturator membrane and rim of pubis and ischium	Middle part of med. aspect of greater trochanter of femur	Lat. rotates and stabilises the hip	Nerve to obturator internus (L5, S1, S2)	
Superior gemellus	Spine of ischium	Middle part of med. aspect of greater trochanter of femur	Lat. rotates and stabilises the hip	Nerve to obturator internus (L5, S1, S2)	
Inferior gemellus	Upper border of ischial tuberosity	Middle part of med. aspect of greater trochanter of femur	Lat. rotates and stabilises the hip	Nerve to quadratus femoris (L4, L5, S1)	
Quadratus femoris	Lat. border of ischial tuberosity	Quadrate tubercle of femur and vertical line below this to lesser trochanter	Lat. rotates and stabilises the hip	Nerve to quadratus femoris (L4, L5, S1)	
Gluteus medius	Gluteal (outer) surface of the ilium between posterior and middle gluteal line	Posterolateral surface of greater trochanter of femur	Abducts and medially rotates hip	Superior gluteal nerve (L4, L5, S1)	
Gluteus minimus	Outer surface of ilium between anterior and inferior gluteal line	Ant. surface of the greater trochanter of femur	Abducts and medially rotates hip	Superior gluteal nerve (L4, L5, S1)	
Tensor fasciae latae	Outer surface of ant. iliac crest between tubercle of the iliac crest and ASIS	Iliotibial tract (ant. surface of lat. condyle of tibia)	Maintains knee extension (assists gluteus maximus and abducts hip). Its action is to pull on the iliotibial tract	Superior gluteal nerve (L4, L5, S1)	Ant. border lies edge to edge with sartorius at the ASIS. Below this two muscles diverge, allowing rectus femoris to emerge between them
Iliotibial band	Begins at level of greater trochanter with ¾ gluteus maximus and tensor fasciae latae inserting into it	Inserts smooth circular facet on the ant. surface of lat. condyle of tibia	Maintains knee in hyperextended position. Assists gluteus maximus in hip abduction	Superior gluteal nerve (L4, L5, S1)	When the knee is straight, the ITB passes in front of the axis of flexion. Maintains knee in hyperextended position. It is not an extensor of the flexed knee

Table 33.25 Medial thigh muscles/adductors of hip

Muscle	Origin	Insertion	Action	Innervation	Notes
Adductor magnus	Adductor portion: Ischiopubic ramus. Hamstring portion: Lower outer quadrant of post. surface of ischial tuberosity	Adductor portion: Lower gluteal line and linea aspera. Hamstring portion: Adductor tubercle	Adductor portion: Adducts and med. rotates hip. Hamstring portion: Extends hip	Adductor portion: Post. Division of obturator nerve (L2–L4). Hamstring portion: Tibial portion of sciatic nerve (L4–S3)	Largest, most powerful and most posterior muscle in the adductor group
Adductor brevis	Inf. ramus and body of pubis	Upper third of linea aspera	Adducts hip	Ant. division of obturator nerve (L2, L3)	The two divisions of the obturator nerve pass ant. and post. to adductor brevis
Adductor longus	Body of pubis inf. and med. to pubic tubercle	Lower two-thirds of med. linea aspera	Adducts and med. rotates hip	Ant. division of obturator nerve (L2, L3)	Most anteriorly placed
Gracilis	Outer surface of ischiopubic ramus	Upper med. shaft of tibia below sartorius	Adducts hip. Flexes knee and med. rotates flexed knee	Ant. division of obturator nerve (L2, L3)	Long, strap-like muscle
Obturator externus	Outer obturator membrane, rim of pubis and ischium bordering it	Trochanteric fossa on med. surface of greater trochanter	Laterally rotates hip	Post. division of obturator nerve (L2, L3, L4)	Flat, small fan-shaped muscle deeply placed in the superomedial part of the thigh
Pectineus	Pectineal line of pubis and narrow area of sup. pubic ramus below it	A vertical line between spiral line and gluteal crest below lesser trochanter of femur	Flexes, adducts and medially rotates hip	Ant. division of femoral nerve (L2, L3). May receive branch from obturator nerve	Included in both the flexor and adductor group of muscles

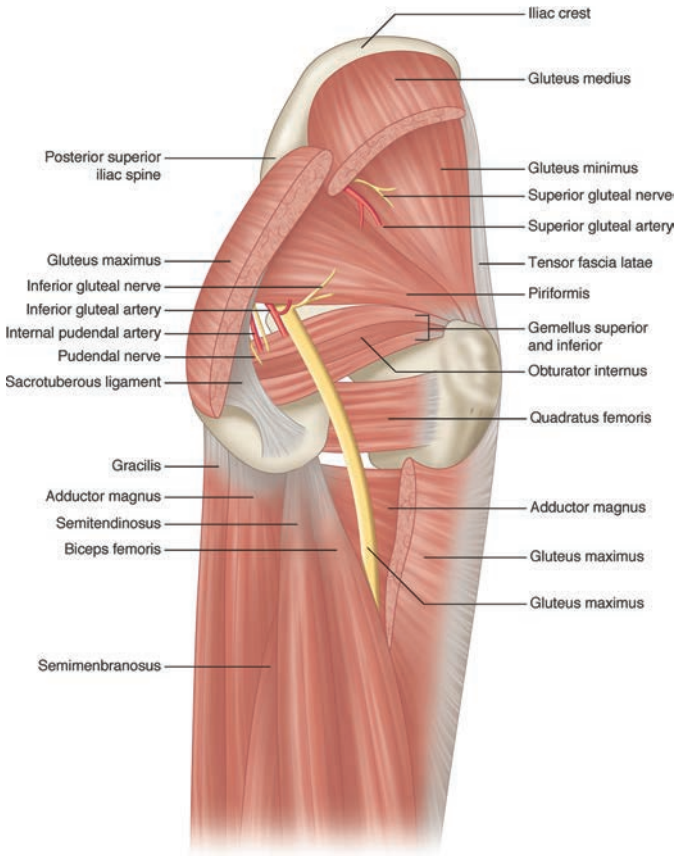

Figure 33.25 Gluteal area of the hip

Tarsal tunnel (Figure 33.28)

The tarsal tunnel is a fibro-osseous space situated posterior to the medial malleolus lying deep to the flexor retinaculum.

Boundary

- Floor: Medial surface of talus, sustentaculum tali and medial wall of the calcaneus

Contents

- Tibialis posterior
- Flexor digitorum longus
- Posterior tibial artery
- Posterior tibial vein
- Tibial nerve
- Flexor hallucis longus

A useful mnemonic to remember the contents is Tom, Dick (**a**nd very **n**aughty) Harry if artery, vein and nerve are included.

The tibial nerve gives rise to the medial calcaneal nerve, the medial plantar nerve and the lateral plantar nerve. The nerve anatomy can be variable.

Lower limb nerves

Obturator nerve (L2, L3, L4)

This emerges from the medial border of psoas major on the ala of the sacrum to pass behind the common iliac vessels. It runs

Table 33.26 Anterior thigh muscles/extensors of the knee

Muscle	Origin	Insertion	Action	Innervation	Notes
Rectus femoris	Straight head: Ant. inf. iliac spine. Reflected head: Ilium above acetabulum	Via quadriceps tendon to patella	Extends leg at knee. Flexes thigh at hip	Post. division of femoral nerve (L3, L4)	Crosses two joints so can flex the thigh at the hip joint and extend the leg at the knee joint. Susceptible to avulsion from the AIIS; hence, the name kicking muscle
Vastus lateralis	Upper intertrochanteric line, base of greater trochanter, lat. linea aspera, lat. supracondylar ridge and lat. intermuscular septum	Lat. quadriceps tendon to lat. patella blending with fibres of rectus femoris	Extends knee	Post. division of femoral nerve (L3, L4)	
Vastus medialis	Lower intertrochanteric line, spiral line, linea aspera and med. intermuscular septum	Med. quadriceps tendon to med. patella and via ligament patellae into tibial tubercle	Extends knee. Stabilises patella	Post. division of femoral nerve (L3, L4)	
Vastus intermedius	Ant. and lat. shaft of femur to one hand's breadth above condyles	Via quadriceps tendon to upper border of patella deep to other muscles	Extends knee	Post. division of femoral nerve (L3, L4)	

Table 33.27 Posterior thigh muscles

Muscle	Origin	Insertion	Action	Innervation	Notes
Biceps (long head)	Upper inner quadrant of post. surface of ischial tuberosity	The two heads join and form a single tendon attached to styloid process of the head of fibula, lat. collateral ligament and lat. tibial condyle	Flexes and lat. rotates knee. Extends hip	Tibial portion of sciatic nerve (L5, S1)	Crosses and protects sciatic nerve. When sciatic nerve divides common peroneal nerve continues this relationship running with the biceps tendon
Biceps (short head)	Middle third of linea aspera, lat. supracondylar ridge of femur	As above	Flexes and lat. rotates knee	Common peroneal portion of sciatic nerve (L5, S1)	In the inferior part of the thigh the long head becomes tendinous and is joined by short head
Semitendinosus	Upper inner quadrant of post. surface of ischial tuberosity	Upper med. shaft of tibia below gracilis	Flexes and med. rotates knee. Extends hip	Tibial portion of sciatic nerve (L5, S1)	Halfway down the thigh becomes cord-like, lying in reciprocal gutter on surface of semimembranosus
Semimembranosus	Upper outer quadrant of post. surface of ischial tuberosity	Post. surface med. condyle of tibia below articular margin, fascia over popliteus and oblique popliteal ligament	Flexes and med. rotates knee. Extends hip	Tibial portion of sciatic nerve (L5, S1)	Sciatic nerve lies on adductor magnus a finger's breadth from the lateral margin of semimembranosus, deep to the long head of biceps

over the pelvic brim on the lateral wall of the sacrum to enter the thigh through the obturator foramen. It has anterior and posterior branches, which lie between the adductor brevis.

- *Anterior branch:* Runs on the anterior aspect of adductor brevis deep to pectineus and then deep to adductor longus to end by becoming subcutaneous at the lower border of adductor longus

Table 33.28 Anterior muscles of the compartments of the leg

Muscle	Origin	Insertion	Action	Innervation	Notes
Tibialis anterior	Upper half of lat. shaft of tibia and interosseous membrane	Inferomedial aspect of med. cuneiform and base of first MT	Extends and inverts foot at ankle. Supports medial longitudinal arch of foot	Deep peroneal nerve (L4, L5)	Lies against the lat. surface of tibia. The long tendon of TA begins halfway down the leg along the ant. surface of tibia
Extensor hallucis longus	Middle half of ant. shaft of fibula	Base of distal phalanx of great toe	Extends big toe and foot. Inverts foot and tightens subtalar joints	Deep peroneal nerve (L4, L5)	The most lateral of the ant. leg muscles
Extensor digitorum longus	Upper two-thirds of ant. shaft of fibula, interosseous membrane and sup. tibiofibular joint	Extensor expansion of lat. four toes	Extends toes and extends foot at ankle	Deep peroneal nerve (L4, L5)	Thin muscle that lies deeply between TA and EDL
Peroneus tertius	Third quarter of ant. shaft of fibula	Shaft and base of 5fth MT	Extends and inverts foot	Deep peroneal nerve (L4, L5)	

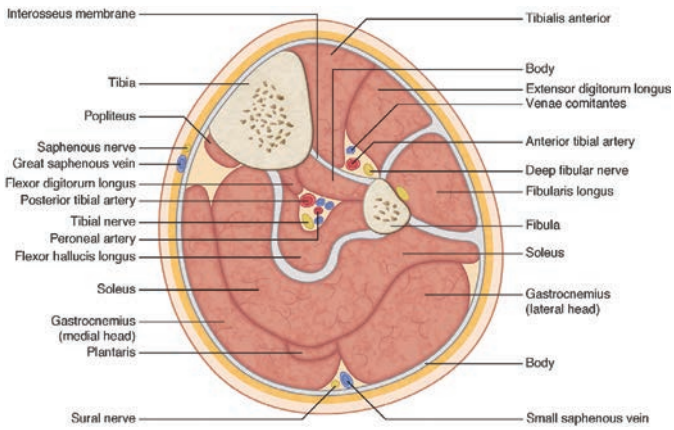

Figure 33.26 Cross-section of mid calf

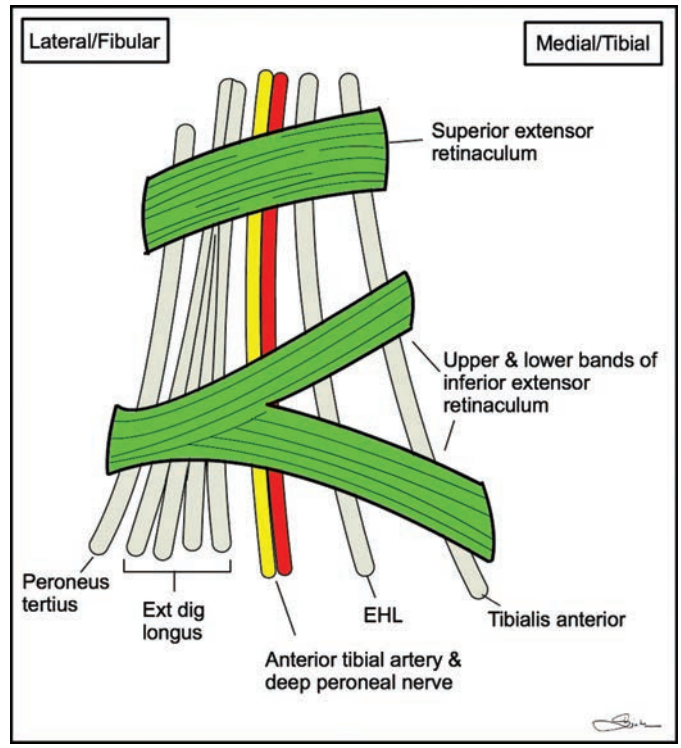

Figure 33.27 Dorsum of foot – Extensor retinaculum and anterior compartment muscles and neurovascular bundle

- Motor to adductor longus, adductor brevis and gracilis. Occasional twig to pectineus
- Sensory medial side of the thigh
- *Posterior branch*: Runs deep to adductor on adductor magnus
- Motor to obturator externus, part of adductor magnus (adductor portion), adductor brevis (variable)

The obturator nerve can be damaged during hip and acetabular surgery (screw placement in the anteroinferior quadrant), leading to loss of hip adduction and decreased sensation of the medial thigh.

Lateral cutaneous nerve of the thigh (L2, L3)

This emerges from the lateral border of psoas major below the iliac crest, crosses the iliacus muscle obliquely and runs towards the ASIS where it passes under the inguinal ligament into the thigh. In the thigh it divides into anterior and posterior branches about 10 cm below the inguinal ligament. Branches from the anterior division distribute and supply the skin of the anterior and lateral parts of the thigh, as far as the knee joint. The posterior branch pierces fascia lata and supplies skin from the level of the greater trochanter to the middle of the thigh.

Bernhardt–Roth syndrome (meralgia paraesthetica) is a condition characterized by numbness and paraesthesia along the distribution of the lateral cutaneous nerve caused by entrapment of the nerve during its course into the thigh.

Table 33.29 Lateral muscles of the compartments of the leg

Muscle	Origin	Insertion	Action	Innervation	Notes
Peroneus longus	Upper two-thirds of lat. shaft of fibula, head of fibula and sup. tibiofibular joint	Plantar aspect of base of first metatarsal and med. cuneiform	Plantarflexes and everts foot. Supports lat. longitudinal and transverse arches	Superficial peroneal nerve (L5, S1)	Arising more superiorly on fibular shaft than peroneus brevis. Exits compartment within common synovial sheath deep to sup. fibular retinaculum. Crosses sole of foot obliquely
Peroneus brevis	Lower two-thirds of lat. shaft of fibula	Tuberosity of base of fifth metatarsal	Dorsiflexes and everts foot. Supports lat. longitudinal arch	Superficial peroneal nerve (L5, S1)	Shorter and deeper than peroneus longus

Table 33.30 Superficial posterior

Muscle	Origin	Insertion	Action	Innervation	Notes
Gastrocnemius	Lat head: Post. surface of lat. condyle of femur and highest three facets on lat. condyle. Med head: Posterior surface of femur above med. condyle	Tendo calcaneus to middle of three facets on post. aspect of calcaneus	Plantarflexes foot. Flexes knee	Tibial nerve (S1, S2)	Fusiform, two-headed, two-jointed muscle. Med. head slightly larger and extending more distally than lateral part
Soleus	Soleal line and middle third of post. border of tibia and upper quarter of post. shaft of fibula, including neck	Tendo calcaneus to middle of three facets on post. surface of calcaneus	Plantarflexes foot	Tibial nerve (S1, S2)	Named as similar to sole-fish. Inverted U attachment to fibula, tibia and tendinous arch of soleus
Plantaris	Lat. supracondylar ridge of femur above lat. head of gastrocnemius	Tendo calcaneus (med. side, deep to gastrocnemius tendon)	Plantarflexes foot and flexes knee	Tibial nerve (S1, S2)	Short muscle, short belly, long tendon. Vestigial muscle absent in 5–10%. Runs distally between gastrocnemius and soleus Can be removed for grafting without causing disability. Long slender tendon easily mistaken for nerve

Table 33.31 Deep posterior

Muscle	Origin	Insertion	Action	Innervation	Notes
Popliteus	Post. part off tibia above soleal line and below tibial condyles	A facet on lat. surface of lat. condyle of femur posteroinferior to epicondyle. Tendon passes into capsule of knee to post. part of lat. meniscus	Unlocks the extended knee by lateral rotation of femur on tibia. Pulls back lat. meniscus	Tibial nerve (L4, L5)	Forms inferior part of the floor of the popliteal fossa. During knee flexion pulls the lat. meniscus posteriorly
Flexor hallucis longus	Lower two-thirds of post. fibula, lower intermuscular septum and aponeurosis of flexor digitorum longus	Base of distal phalanx of big toe and slips to med. two tendons of FDS	Flexes distal phalanx of big toe, flexes foot at ankle, supports med. longitudinal arch	Tibial nerve (L4, L5)	Occupies a shallow groove on the post. surface of talus. Crosses deep to FDL in the sole of the foot
Flexor digitorum longus	Post. shaft of fibula below soleal line and by broad aponeurosis from fibula	Base of distal phalanges of lat. four toes and foot at ankle. Supports lat. longitudinal arch	Flexes distal phalanges of lat. four toes and foot at ankle. Supports lat. longitudinal arch	Tibial nerve (S1, S2)	Its direction of pull is realigned by quadratus plantae
Tibialis posterior	Upper half of post. shaft of tibia and upper half of fibula between medial crest and interosseous border	Tuberosity of navicular bone and all tarsal bones (except talus) and spring ligament	Plantarflexes and inverts foot. Supports med. Longitudinal arch of foot	Tibial nerve (L4, L5)	Deepest muscle in post. compartment. Lies between FDL and FHL in the same plane as the tibia/fibula

Table 33.32 Muscles of the ankle and foot

Muscle	Origin	Insertion	Action	Innervation	Notes
Dorsal layer					
Extensor digitorum brevis	Sup. surface of ant. calcaneus	Four tendons into prox. phalanx of big toe and long extensor tendons to toes 2, 3 and 4	Extends toes when foot fully dorsiflexed	Deep peroneal nerve (L5, S1)	
First plantar layer					
Abductor hallucis	Med. process of post. calcaneal tuberosity and flexor retinaculum	Med. aspect of base of prox. phalanx of big toe via med. sesamoid	Flexes and abducts big toe. Supports med. longitudinal arch	Med. plantar nerve (S1, S2)	
Flexor digitorum brevis	Med. process of post. calcaneal tuberosity	Four tendons to four lat. toes inserted into sides of middle phalanx. Tendons of FDL pass through them	Flexes lat. four toes. Supports med. and lat. longitudinal arches	Med. plantar nerve (S1, S2)	Central muscle. Equivalent to FDS in the upper limb and gives off four tendons
Abductor digiti minimi	Med. and lat. processes of post. calcaneal tuberosity	Lat. side of base of prox. phalanx of fifth toe and fifth MT	Flexes and abducts fifth toe. Supports lat. longitudinal arch	Lat. plantar nerve (S2, S3)	
Second layer					
Quadratus plantae (flexor accessorius)	Med. and lat. sides of calcaneus	Tendons of FDL	Assists FDL to flex lat. four toes, especially when ankle is plantarflexed	Lat. plantar nerve (S2, S3)	
Lumbricals	Lat. 3: Bipennate origin from cleft between the four tendons of FDL. Med. 1: Unipennate origin from med. aspect of first tendon	Med. side of dorsal extensor expansion and base of prox. phalanges	Extends toes at IP joints and flexes MTP joints	First: Med plantar nerve (L4, L5). 2–4: Deep branch of lat. plantar nerve (S2, S3)	Pass forwards on the med. (tibial) side of the MCP joints of the lateral four toes. Lie on the deep transverse ligaments of MC heads
Flexor hallucis longus	Lower two-thirds of post. fibula between median crest and post. border, lower intermuscular septum and aponeurosis of FDL	Base of distal phalanx of big toe and slips to med. two tendons of FDS	Flexes distal phalanx of big toe, flexes foot at ankle, supports med. longitudinal arch	Tibial nerve (L4, L5)	Runs forward below sustentaculum tali and crosses deep to FDL
Flexor digitorum longus	Post. shaft of fibula below soleal line and by broad aponeurosis from fibula	Base of distal phalanges of lat. four toes and foot at ankle. Supports lat. longitudinal arch	Flexes distal phalanges of lat. four toes and foot at ankle. Supports lat. longitudinal arch	Tibial nerve (L4, L5)	Similar insertion to FDP in the hand
Third layer					
Flexor hallucis brevis	Cuboid, lat. cuneiform and tibialis posterior insertion over the two remaining cuneiforms	Med. tendon to med. side of base of prox. phalanx of big toe. Lat. tendon to lat. side of same, both via sesamoids	Flexes MTP joint of big toe. Supports med. longitudinal arch	Med. plantar nerve (S2, S3)	

Table 33.32 (*cont.*)

Muscle	Origin	Insertion	Action	Innervation	Notes
Adductor hallucis	Oblique head: Base of 2, 3, 4 MT's. Transverse head: Plantar MT ligaments and deep transverse ligament	Lat. side of base of prox. phalanx of big toe and lat. sesamoid	Adducts and flexes MTP joint of big toe. Supports transverse arch	Deep branch of lat. plantar nerve (S2, S3)	
Flexor digiti minimi brevis	Base of fifth MT and sheath of peroneus longus	Lat. side of base of prox. phalanx of little toe	Flexes MTP joint of little toe	Superficial branch of lateral plantar nerve	
Fourth layer					
Dorsal interosseous	Bipennate from inner aspects of shafts of all MTs	Bases of prox. phalanges and dorsal extensor expansions of med. side of second toe and lat. sides of 2–4 toes	Abducts 2–4 toes from axis of second toe. Assists lumbricals in extending IP joints whilst flexing MTP joints	Lat. plantar nerve (1–3 deep branches; 4: Superficial branch) (S2, S3)	Four dorsal interossei arises by two heads from the two MTs between which it lies
Plantar interosseous	Inferomedial shafts of 3–5 MTs (single heads)	Med. Sides of bases of prox. phalanges with slips to dorsal extensor expansions of 3–5 toes	Adduct 3–5 toes to axis of second toe. Assists lumbrical in extending IP joints whilst flexing MTP joints	Deep branch of lat. plantar nerve (S2, S3)	Three plantar interossei arise from the MT bone of its own toe
Peroneus longus	Upper two-thirds of lat. shaft of fibula, head of fibula and sup. tibiofibular joint	Plantar aspect of base of first MT and med. cuneiform, passing deep to long plantar ligament	Plantarflexes and everts foot. Supports lat. longitudinal and transverse arches	Superficial peroneal nerve (L5, S1)	Passes posterior to the lateral malleolus, superficial to peroneus brevis. Peroneus brevis lies on the bone
Tibialis posterior	Upper half of post. shaft of tibia and upper half of fibula between medial crest and interosseous border, and interosseous membrane	Tuberosity of navicular bone and all tarsal bones (except talus) and spring ligament	Plantarflexes and inverts foot. Supported med. longitudinal arch of foot	Tibial nerve (L4, L5)	

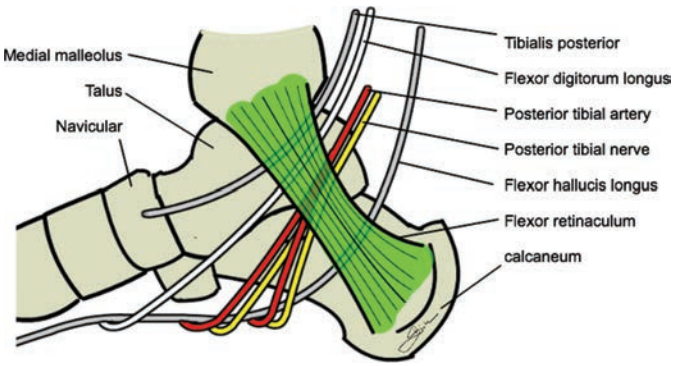

Figure 33.28 Tarsal tunnel

Femoral nerve (L2, L3, L4)

The femoral nerve arises from posterior divisions of the ventral rami of L2, L3 and L4. It is formed within psoas major and emerges from its lateral border lying in the iliac fossa. It reaches the thigh by running beneath the inguinal ligament lateral to the femoral artery lying on the psoas and iliacus tendon.

As it enters the femoral triangle it divides into a superficial and deep group.

Superficial group: This comprises of two cutaneous and two muscular branches.

- Intermediate femoral cutaneous nerve (anterior thigh skin)
- Medial femoral cutaneous nerve (medial thigh skin)

There is a nerve to pectineus and a nerve to sartorius.

Deep group: There are four muscular nerves and one cutaneous nerve.

The saphenous nerve supplies the skin of the medial knee, medial calf and shin, and medial forefoot. It descends in the femoral triangle to reach the adductor canal where it spirals over the femoral artery to lie medial to it. It pierces the deep fascia on the medial side of the knee after emerging between

sartorius and gracilis. It continues down the medial side of the leg with the long saphenous vein. It passes in front of the medial malleolus and along the medial border of the foot to the big toe.

The four muscular nerves are nerves to rectus femoris, vastus medialis, vastus lateralis and vastus intermedius.

Sciatic nerve (L4–S3)

The sciatic nerve arises from the ventral rami of L4–S3 and descends through the greater sciatic foramen to enter the gluteal region, emerging from below the piriformis muscle more laterally than the inferior gluteal and pudendal vessels.

It descends through the posterior compartment of the thigh and ends by dividing into tibial and common peroneal nerves, usually just above the popliteal fossa. Occasionally there is a high division at the piriformis muscle. The nerve lies on the ischium, superior gemellus, obturator internus, inferior gemellus, quadriceps femoris and adductor magnus. It is overlaid by the long head of biceps. This nerve does not supply any structure in the gluteal region.

The common peroneal portion has one innervation in the thigh – The short head of biceps femoris. The short head of biceps femoris was developed in the extensor compartment but migrated to the flexor compartment for functional reasons, retaining its nerve supply.

The tibial portion supplies the hamstring muscles (semimembranous, semitendinosus, biceps) as well as the hamstring part of the adductor magnus.

Nerve to quadratus femoris (L4, L5, S1)

This arises from the anterior surface of the sciatic nerve. It leaves the pelvis through the lower part of the greater sciatic notch and runs deep to obturator internus and inferior gemelli before entering quadratus femoris.

Nerve to obturator internus (L5, S1, S2)

This arises from the anterior surface of the sciatic nerve. It leaves the pelvis through the greater sciatic notch below piriformis and medial to the sciatic nerve. It passes over the ischial spine and sends a branch to the superior gemellus before turning forward to pass through the lesser sciatic notch and then penetrating and supplying obturator internus.

Common peroneal nerve (L4–S2)

This is the smaller terminal branch of the sciatic nerve. It begins just above the apex of the popliteal fossa and descends underneath the medial border of biceps femoris muscle. It crosses plantaris, lateral head of gastrocnemius, popliteus tendon inside the knee joint capsule and the fibular origin of soleus. It leaves the fossa, passing into peroneus longus where it divides into superficial and deep peroneal nerves. Before dividing, the nerve gives off five branches (three cutaneous and two articular):

- Lateral sural cutaneous nerve (upper lateral calf skin)
- Peroneal communicating nerve
- Recurrent genicular nerve (skin over patella)
- Superior and inferior genicular nerves

The common peroneal itself supplies no muscles. It is a nerve of the extensor compartment. It is vulnerable to injury where it lies on the neck of the fibula.

Deep peroneal nerve (L4–S2)

This begins within peroneus longus and winds around the fibular neck deep to extensor digitorum longus. It enters the anterior compartment by piercing the interosseous membrane and descends between extensor digitorum longus (EDL) and tibialis anterior (TA) lateral to the anterior tibial vessels.

In the middle of the leg the deep peroneal nerve lies on the interosseous membrane between TA and extensor halluces longus (EHL). At the lower part of the tibia, EHL crosses over the nerve so that two muscles lie on either side of the neurovascular bundle (TA, EHL medially and EDL, peroneus tertius laterally). It ends by supplying the skin of the first web space.

It supplies the muscles of the extensor compartment of the leg: Tibialis anterior, extensor digitorum longus, extensor hallucis longus, peroneus tertius and extensor digitorum brevis.

Superficial peroneal nerve

The superficial peroneal nerve begins in the substance of peroneus longus on the lateral side of the fibular neck. It descends first between peroneus longus and peroneus brevis, and then between peroneus longus and extensor digitorum longus. It supplies peroneus longus and brevis and pierces the deep fascia in the distal third of the leg. It divides into medial and lateral cutaneous branches.

The medial branch supplies skin on the distal part of anterior leg, skin on the dorsum of the foot (except the first web space), the medial side of the big toe and adjacent sides of the second and third toes.

The lateral branch also supplies skin of the dorsum of the foot, and adjacent sides of the third, fourth and fifth toes.

Tibial nerve (L4–S2)

This is the terminal branch of the sciatic nerve. It begins just above the popliteal fossa, descends almost vertically through the fossa, lying first on the lateral side of the popliteal artery, then posterior to it and finally medial to it. The popliteal vein lies between the nerve and artery throughout its course. At the lower border of the popliteus muscle, it passes deep to the tendinous arch of the soleus muscle accompanied by the tibial vessels. It runs straight down the midline of the calf, deep to soleus, lying on the fibular aponeurosis of flexor digitorum longus. The nerve accompanies the posterior tibial artery and lies first on its medial side, then crosses posterior to it and finally lies on its lateral side.

It leaves the posterior compartment of the leg under the flexor retinaculum behind the medial malleolus and then divides into the medial and lateral plantar nerves. The tibial nerve innervates all the muscles of the foot except the extensor digitorum brevis (supplied by the peroneal nerve).

In the popliteal space it gives off five muscular, three articular (genicular nerves) and one cutaneous nerve:

- Popliteus muscle, two heads of gastrocnemius, soleus and plantaris
- Upper and lower medial and middle genicular nerves
- Medial sural cutaneous nerve, which joins with the peroneal communicating branch of the common peroneal nerve to form the sural nerve

In the posterior compartment it supplies four muscles:

- Soleus, tibialis posterior, FDL and FHL

The nerve passes behind the medial malleolus between FDL and FHL under cover of the flexor retinaculum where it divides into medial and lateral plantar nerves.

Medial plantar nerve (counterpart of the median nerve in the hand): Runs forward deep to the abductor hallucis with the medial plantar artery on its medial side. Supplies abductor hallucis, flexor digitorum brevis, flexor hallucis brevis and the first lumbrical. The nerve gives off three sensory branches (common plantar digital nerves) that supply the medial 3½ digits, including the dorsal skin and nail bed of the distal phalanges

Lateral plantar nerve (counterpart of the ulnar nerve in the hand): Crosses the sole obliquely just deep to the first layer of muscles and supplies flexor accessorius and abductor digiti minimi. Near the base of the fifth metatarsal bone it divides into superficial and deep branches

- Main trunk: Motor to quadratus plantae and abductor digiti minimi
- The superficial branch supplies three muscles: Flexor digiti minimi brevis and the two interossei of the fourth space (third plantar and fourth dorsal)
- The deep branch supplies the remaining interossei, transverse head of adductor hallucis and the lateral three lumbricals

Sural nerve (S1, S2)

The sural nerve descends on the posterior surface of gastrocnemius and unites with the peroneal communicating nerve (communicating branch from the common peroneal nerve). It runs down alongside the saphenous vein behind the lateral malleolus and ends on the lateral side of the little toe. It is sensory to the posterolateral third of the leg, the lateral part of the foot and heel, and the lateral side of the little toe.

The nerve is often used for nerve grafting. It can be used as a guide to the tibial nerve; follow the sural nerve upwards to pierce the deep fascia and join the tibial nerve.

Saphenous nerve (L3, L4)

The saphenous nerve is the continuation of the femoral nerve from the thigh. In the leg it travels with the great saphenous vein and passes in front of the medial malleolus into the foot where it supplies sensation over the medial border of the leg and foot as far as the ball of the big toe. The nerve can be damaged during knee surgery (especially the infrapatellar branch) as it becomes subcutaneous on the medial side of the knee between the sartorius and the gracilis.

Lower limb arteries

Femoral artery

The femoral artery is a continuation of the external iliac artery, beginning posterior to the inguinal ligament at the mid-inguinal point (halfway between ASIS and pubic symphysis). It ends as it passes through the adductor hiatus in the adductor magnus to become the popliteal artery. It emerges from under the inguinal ligament with the femoral vein medial to it, both within the femoral sheath.

It lies on psoas major.

As the artery enters the adductor canal it lies on adductor longus then adductor magnus.

Four branches arise from the artery in the femoral triangle: Superficial epigastric artery, superficial circumflex iliac artery, superficial external pudendal artery and deep external pudendal artery.

The profunda femoris artery is the main branch of the femoral artery and is given off posterolaterally just below the femoral sheath. It is the main blood supply to the thigh. It runs posteriorly between pectineus and adductor longus. It gives off medial and lateral circumflex femoral branches.

Popliteal artery

The popliteal artery begins as the continuation of the femoral artery as it passes through the adductor magnus hiatus and ends as it passes under the fibrous arch of soleus where it divides into anterior and posterior tibial arteries. The popliteal artery is the deepest neurovascular structure in the popliteal fossa, lying with only fat between it and the popliteal surface of the femur. Biceps femoris is medial and semimembranosus medial. Lower down it lies between the two heads of gastrocnemius. At all levels the popliteal vein lies between the artery and the tibial nerve. It is crossed laterally to medially by the tibial nerve.

Anterior tibial artery

This supplies structures in the extensor compartment of the lower leg. It descends on the interosseous membrane and crosses the lower tibia at the ankle joint midway between the malleoli. Initially it lies between tibialis anterior (medially) and EDL (laterally), then between tibialis anterior and EHL. The deep peroneal nerve is initially lateral to the artery but passes anterior to it before again becoming lateral.

Posterior tibial artery

The posterior tibial artery travels downwards on tibialis posterior, FDL, the tibia and the ankle joint. It lies deep to gastocnemius, soleus, flexor retinaculum and abductor hallucis. It ends under the flexor retinaculum by dividing into medial and lateral plantar arteries.

Dorsalis pedis artery

This is a direct continuation of the anterior tibial artery. It begins midway between the malleoli and runs anteromedially, deep to the inferior extensor retinaculum between EHL and EDL tendons.

Spine osteology and joints

The osteology of the spine involves the cervical, the thoracic, the lumbar and the sacral spinal vertebrae. The typical spinal vertebrae are very similar and have almost identical features in comparison to the other vertebrae of the same region of the spine. The atypical vertebrae differ from the other vertebrae in a few key components.

Cervical vertebrae

Typical cervical vertebra C3–C6

The features of the cervical vertebra are as follows.

Shape

- The body of the vertebra is small and is broader from side to side in comparison from front to back
- The spinous processes are short and bifid
- The vertebral foramen is large and triangular in shape

Articulations

- The superior and inferior articulating facets form articulation between the vertebrae
- These articular facets are flat and oval in shape
- The orientation of the superior facets is facing backward, upward and slightly medially
- The corresponding orientation of the inferior facets in order to help articulate with the superior facets of the next vertebrae is facing forward, downward and slightly laterally

Distinguishing features

The transverse processes of these vertebrae have a foramen transversarium, which allows passage of the vertebral artery and the vertebral vein along with a plexus of sympathetic nerves

The anterior tubercle of the sixth cervical vertebrae is known as the carotid tubercle or the *Chassaignac tubercle*. This tubercle is significant in separating the carotid artery from the vertebral artery. The carotid artery is massaged against this tubercle for relieving the signs of supraventricular tachycardia. This tubercle is also used as a landmark for anesthesia of the brachial or the cervical plexus of nerves

The cervical nerves emerge above the designated vertebrae. For example the third cervical nerve C3, passes above the third cervical vertebrae

Atypical vertebra atlas/C1

The first cervical vertebra is unique in the following aspects.

Shape

The shape of the vertebra is similar to a ring. This ring comprises of the following parts:

- The anterior arch – Forming the anterior wall of the ring
- The posterior arch – Forming the posterior wall of the ring
- The lateral masses – Forming the lateral walls
- The transverse processes on either side housing the transverse foramen – These foramens transmit the vertebral artery and the vertebral vein along with a plexus of sympathetic nerves
- There is no body of this vertebra. The dens of the second vertebra is embryologically derived from the body of the first vertebra

Ligaments

- The central vertebral foramen is divided into two parts by the transverse atlantal ligament (TAL). The anterior part is smaller and houses the odontoid process (dens) of the second cervical vertebra. The posterior part is larger and houses the spinal cord and its membranes
- This transverse atlantal ligament is prone to inflammatory degeneration in patients with rheumatoid arthritis causing atlanto-axial instability. Attempts at intubating these patients can result in damage to the spinal cord

Articulations

- The lateral masses have two articular facets
- The superior articular facets are large and articulate with the corresponding condyles of the occipital bone
- The inferior facets are slightly flattened to convex and articulate with the axis/C2 vertebra

Atypical vertebra axis/C2

The second cervical vertebra is unique in the following aspects.

The dens

- This is also referred to as the odontoid process and is shaped like a peg
- The anterior surface articulates with the anterior arch of the atlas bone
- The posterior surface has a transverse groove for the TAL. This ligament stabilises the dens and limits it from compressing the spinal cord
- The apex of the dens gives attachment to the apical odontoid ligament and the alar ligaments on either side. These ligamentous connections join the dens to the occipital bone

Some of the other distinguishing features

- The central foramen is larger than the typical cervical vertebrae. The lamina are very thick and strong

- The transverse processes are very small and have a perforation on either side or the vertebral foramen
- The spinous process is large, very strong and has a bifurcated tip

Atypical vertebra C7

Unique features of the C7 vertebra

- The spinous process is long and prominent but is not bifid
- The vertebral artery passes anterior to the transverse process rather than inside the transverse foramen, but variations are known to occur

Typical thoracic vertebrae T2–T8

Shape

- The bodies of the vertebrae are heart shaped
- The laminae are broad and thick and overlap the adjacent laminae like tiles on a roof
- The pedicels are directed backward and slightly upward
- The spinous process is long and directed downwards. The cross-section of the process is triangular
- The transverse processes are thick, strong and long. They are directed obliquely backward and lateral. The ends if the processes are clubbed. These processes have a small concave surface close to the clubbed ends for **articulation of the ribs**

Articulations

Typical lumbar vertebrae

- The superior articular processes are thin plates of bone projecting upward from the junctions of the pedicles and laminae
- These superior articular facets are practically flat, and are directed backward and a little laterally and upward
- The inferior articular facets are fused to an extent with the laminae. Their facets are directed forward, downward and medial
- These vertebrae constitute the largest segment of the vertebral column
- The main components of these vertebrae are:
  - **Vertebral body**
    - This is large and wider from side to side
    - It is thicker in front than the back
    - It is flattened or slightly concave from above and below
    - It is concave on the behind and deeply constricted in the front and the sides
  - **Vertebral arch – This vertebral arch consists of:**
    - A pair of pedicels – Which are strong and directed backward from the upper body. These pedicels change in morphology as we progress from upper lumbar to lower lumbar
      - The sagittal width increases
      - The angulation in axial plane increases from 10° to 20°
    - A pair of laminae – That are broad short and strong
      - They form the posterior portion of the vertebral arch
      - The laminae connect the spinous process to the pedicels
  - **Vertebral foramen – Is within the arch and is triangular in shape**
    - This foramen is smaller than the cervical vertebrae, but larger than the thoracic vertebrae
  - **The spinous processes:**
    - These spinous processes are thick, broad and quadrilateral in cross-section
    - It is projected backwards and ends in a rough border
    - It has well-defined superior and inferior articular facets

Important ligaments

Anterior longitudinal ligament

This extends from the basiocciput of the skull and anterior tubercle of the atlas to the front of the upper part of the sacrum. It is firmly attached to the periosteum of the vertebral bodies but less so over the intervertebral discs.

Posterior longitudinal ligament

This extends from the back of the vertebral body of the axis to the anterior wall of the upper sacral canal. It is broader and more firmly attached over the intervertebral discs but narrower and more loosely attached to the vertebral bodies.

Ligamentum flavum

These are a pair of yellowish (high content of elastic tissue) ligaments that join the contiguous borders of adjacent lamina. They are attached above to the front of the upper lamina and below to the back of the lower lamina.

They are long, thin and broad in the cervical region, thicker in the thoracic region and thickest in the lumbar region.

Supraspinous ligaments

These join the tips of the adjacent spinous processes from C7 to the sacrum. They are strong cord-like bands of white fibrous tissue. At the back of the neck they merge with the ligamentum nuchae.

The ligament nuchae is a strong broad triangular septum of fibroelastic tissue attached to the external occipital crest, bifid spines of the cervical vertebrae and the investing layer of deep cervical fascia, which encloses the trapezius muscles.

Interspinous ligaments

These unite the spinous processes of the vertebrae along their adjacent borders. They are well developed only in the lumbar region and they fuse with the supraspinous ligaments.

Intertransverse ligaments

Weak sheets of fibrous tissue joining the transverse processes of vertebrae along their adjacent borders.

Apical ligament

This joins the apex of the dens to the anterior margin of the foramen magnum, and is a fibrous remnant of the notochord.

Alar ligaments

The alar ligaments lie obliquely on either side of the apical ligament. They diverge from the side of the dens to the foramen magnum. They are very strong ligaments and limit rotation of the head.

Spinal cord

The spinal cord is the main pathway for information connecting the brain and peripheral nervous system. The length of the spinal cord is much shorter than the length of the vertebral column.

It extends from the foramen magnum and continues through to the conus medullaris near the second lumbar vertebra, terminating in a fibrous extension known as the filum terminale, which anchors the spinal cord to the coccyx. The cauda equina ('horse's tail') is the name for the collection of nerves in the vertebral column that continue to travel through the vertebral column below the conus medullaris.

The spinal cord has two enlargements:

- Cervical enlargement – Corresponds roughly to the brachial plexus nerves, which innervate the upper limb. It includes spinal cord segments from about C4 to T1. The vertebral levels of the enlargement are roughly the same (C4 to T1)
- Lumbosacral enlargement – Corresponds to the lumbosacral plexus nerves, which innervate the lower limb. It comprises the spinal cord segments from L2 to S3 and is found about the vertebral levels of T9 to T12

The spinal cord is protected by three spinal meninges:

- The *duramater* is the outermost layer, and it forms a tough protective coating. The space between the vertebral column and the duramater is called the epidural space, which contains adipose tissue and blood vessels. The space between duramater and arachnoid mater is called subdural space
- The *arachnoid mater* is the middle protective layer. The space between the arachnoid and the underlying piamater is called the subarachnoid space. The subarachnoid space contains cerebrospinal fluid (CSF)
- The *piamater* is the innermost protective layer. It is very delicate and it is tightly associated with the surface of the spinal cord

The cord is stabilised within the dura mater by the connecting denticulate ligaments, which extend from the piamater laterally between the dorsal and ventral roots. The dural sac ends at the vertebral level of the second sacral vertebra.

In cross-section, the peripheral region of the cord contains neuronal white matter tracts containing sensory and motor neurons. Internal to this peripheral region is the grey matter, which surrounds the central canal, which is an extension of the fourth ventricle and contains CSF.

The white matter consists almost totally of myelinated motor and sensory axons. The grey matter is shaped like a butterfly and consists of cell bodies of interneurons, motor neurons, neuroglia cells and unmyelinated axons.

The spinal cord has a shape that is compressed dorsoventrally, giving it an elliptical shape. The cord has grooves in the dorsal (posterior median sulcus) and ventral sides (anterior median fissure).

Spinal cord segments

Spinal cord is divided into 31 segments. At every segment, right and left pairs of spinal nerves (mixed, sensory and motor) emerge.

Motor nerve rootlets emerge from the ventro lateral sulci and combine to form the motor nerve root.

The sensory nerve rootlets arise form the dorso lateral sulci and form sensory nerve roots. Each segment of the spinal cord is associated with a pair of ganglia, called dorsal root ganglia, which are situated just outside the spinal cord. These ganglia contain cell bodies of sensory neurons.

The ventral (motor) and dorsal (sensory) roots combine to form spinal nerves one on each side of the spinal cord and exit from the intervertebral foramen.

In the upper part of the vertebral column, spinal nerves exit directly from the spinal cord, whereas in the lower part of the vertebral column the nerves pass further down the column before exiting.

Blood supply of spinal cord

The spinal cord is supplied by three arteries that run along its length starting in the brain. There are also many arteries that approach it through the sides of the spinal column.

The three longitudinal arteries are called the *anterior spinal artery* and the *right and left posterior spinal arteries*.

These travel in the subarachnoid space and send branches into the spinal cord. They form anastomoses via the anterior and posterior *segmental medullary arteries*, which enter the spinal cord at various points along its length.

The actual blood flow through these arteries, derived from the posterior cerebral circulation, is inadequate to maintain the spinal cord below the cervical segments.

The major contribution to the arterial blood supply of the spinal cord below the cervical region comes from the radially arranged *posterior and anterior radicular arteries*, which run into the spinal cord alongside the dorsal and ventral nerve roots. These intercostal and lumbar radicular arteries arise

from the aorta, provide major anastomoses and supplement the blood flow to the spinal cord.

The largest of the anterior radicular arteries is known as the *artery of Adamkiewicz*, or *anterior radicularis magna (ARM) artery*, which usually arises between L1 and L2, but can arise anywhere from T9 to L5. Impaired blood flow through these critical radicular arteries, especially during surgical procedures that involve abrupt disruption of blood flow through the aorta, for example during aortic aneursym repair, can result in spinal cord infarction and paraplegia.

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Chapter 34

SAS doctors and the FRCS (Tr & Orth) exam

Ramnadh S. Pulavarti and Kevin P. Sherman

Although the FRCS (Tr & Orth) examination is not mandatory, it is often considered as a strong point when making an application for the Certificate of Eligibility for Specialist Registration (CESR) and the Specialty Advisory Committee (SAC) can request it.

Besides helping with any application for Certificate of Completion of Training (CCT) through the CESR route, the preparation required for the FRCS (Tr & Orth) diploma will also help in many other ways:

- Getting up-to-date with knowledge, understanding and clinical skills
- Improving the understand of the basic sciences behind daily clinical practice
- Improving confidence both in clinical and academic circles
- Increasing awareness of the best available evidence to support your clinical practice
- Improving the counselling of patients, both in clinics and preoperatively
- Helping the teaching and training of junior colleagues
- Generally helping with continuing medical education (CME) and career progression

However, the examination can be a particularly daunting experience for doctors in the non-training grades. As with all other examinations, planning well ahead and intense preparation are essential for success.

SAS doctors have a lower pass rate in the FRCS (Tr & Orth) examination than those taking the examination from formal numbered training posts

Pass rates for doctors who are not in formal structured training are much lower than those for doctors in such formal training; the overall pass rate may be as low as one-third that of those in numbered training posts. This may seem surprising at first sight given the great experience of some specialty and associate specialist (SAS) doctors.

There are a number of reasons why pass rates for SAS doctors tend to be low and an understanding of some of these reasons will help to improve your chances of success.

- **Insufficient breadth of recent experience**

  Although SAS doctors often have a lot of clinical experience, their recent experience is frequently in the relatively narrow area of Orthopaedics. The examination tests knowledge, understanding and skills across the breadth of the curriculum and it is important to ensure that there are not significant gaps in expertise and knowledge
- **Lack of objective feedback**

  Trainees in structured training programmes are subjected to regular formative assessments that provide continuous feedback on their performance and progression. SAS doctors have frequently spent many years working largely in isolation without having anyone carry out formative assessments of their day-to-day clinical skills. Bad habits and 'short cuts' can creep in over time. As a result they may find that when they take the examination they do not fail because of lack of knowledge about complex topics but instead fail on simple 'everyday' clinical skills such as basic examination of joints or taking a history. This is a similar problem to taking a driving test; many 'experienced' drivers would fail a driving test because they would not demonstrate adequately to the examiner that they consistently follow safe and comprehensive processes and procedures

  It can be difficult for an experienced SAS doctor to ask someone to watch them perform a simple examination of a joint and then critically appraise the performance, but for success it is essential to do so and for the person doing the watching to provide constructive feedback
- **Lack of confidence**

  This is connected with the lack of objective feedback. When someone who has not been observed doing something suddenly has to do it in front of an 'audience' (the examiners) their performance may be much poorer than normal due to lack of confidence or self consciousness; so called 'exam nerves'. The best way to counter exam nerves is to repeatedly practise performing in front of others. This practice must, however, be accompanied by objective and constructive feedback, otherwise there will be an increase in confidence without an improvement in performance

  Lack of confidence due to the absence of formative assessments in the workplace is exacerbated by the fact that

many SAS doctors have not taken an examination for many years, which makes it all the more daunting, and courageous, to subject themselves to examination once again. The answer to this problem is again regular practice

- **Lack of understanding of the standard required**
  Some examinees take the examination without a sound understanding of the standard required; this is not only an expensive waste of money but also unwise. Although some may think that it would be worth taking the examination to gain an understanding of the standard required there is usually a vain hope that they might actually pass. The failure that usually follows taking an examination without proper preparation can damage self confidence and lead to poorer subsequent performance on a re-take
- **Lack of practice at using the correct Orthopaedic language and terminologies**
  The French sociologist Pierre Bordieu developed the concepts of 'Habitus' and 'Capital'. Without wishing to go into the complexities of sociology, it is worth understanding the relevance of these concepts; namely that people acquire 'Habitus' through their interactions with a society or a particular group within society and that their Habitus dictates the way they think, feel and act. 'Capital' is acquired by individuals and is used to attain position within the society or group, and this concept applies not only to material capital but also to social and cultural capital. The cultural capital involves, amongst other things language, judgments and values. The relevance of this to the FRCS (Tr & Orth) examination is that a candidate who is used to using the appropriate language (i.e. 'professional' language) and making judgments in the 'correct' way, based on the prevailing values of the body to which he or she wishes to join is more likely to prove successful. The appropriate terminologies and judgments can only be acquired by regular discussions with those already in the group that the examinee wishes to join. It follows that regular professional discussions with Consultants about how judgments and decisions are made are an essential component of preparation for the examination

  The above argument applies particularly to discussing the basic sciences that underpin clinical practice; SAS doctors are often very good at making clinical decisions based on extensive empirical experience but find it difficult to explain the underlying basic science on which such decisions are made. Understanding basic science is, however, important to ensure that decision making is continuously updated on a rational basis rather than being based solely on past experience
- **Lack of practice at completing tasks on time**
  In clinics there may be time pressures but a longer time spent with one patient can often be made up on subsequent consultations. Similarly when discussing orthopaedic topics in the coffee room or in the operating theatre there are no absolute time pressures. Although many parts of the examination mimic clinical practice fairly closely, the one essential difference is that there are absolute time limits on each interaction; whatever is not discussed or demonstrated in each relevant time slot (usually 5 min) will not count. It is, therefore, important to set aside time to practice discussing topics, developing ideas or performing clinical examinations within a fixed (short) timescale and then to reflect (with an observer) what has been missed out or covered inadequately

Planning for the examination

The eligibility criteria for FRCS Tr & Orth examination are given in the Joint Committee on Intercollegiate Examinations (JCIE) website. The applicant must have held a recognised medical qualification for at least 6 years. The applicant must also provide evidence of having reached the standard of clinical competencies defined in the Intercollegiate Surgical Curriculum. The required standard may have been achieved through training or qualifications, and experience considered all together. The passing of the Intercollegiate Specialty Examination alone does not imply that the CCT will be automatic, the Examination will form only part of the evidence required. This evidence must include three structured references in the format prescribed by the JCIE. These references must be completed by the appropriate senior colleagues with direct experience of the applicant's current clinical practice.

Placement, gaining experience and competencies in various specialties including Paediatric Orthopaedic Surgery and Spinal Surgery over a period of at least 6 years is important. It necessitates careful planning with your supervising Consultant and rota coordinator. Regular annual appraisals highlighting the competencies reached at every stage and development plan for the year ahead will be of great benefit. This is particularly true for those doctors in SAS grade positions and other non-training positions. It is important to collect evidence of achieving competencies in an objective manner such as PBAs, DOPS, etc, and maintaining a log of operative and academic activities (this will also help with examination preparation as described above).

Choosing three referees who have direct experience with your clinical practice and willing to provide a structured reference is the next important step. One of these should be your current clinical director if you are a SAS doctor.

Before asking your consultant for a reference, it is important to ask yourself the following questions:

- Have you demonstrated an interest in and a potential for career progression?
- Have you demonstrated an intent to learn and change? (reflective practice)
- Have you been attending instructional courses and workshops regionally and nationally?
- Have you been participating actively in trauma and clinical meetings?

- Have you been presenting audits, interesting cases and participating in a journal club?
- Have you been keeping your log-book up to date?
- Have you had regular annual appraisals and personal development plans over a period of at least 5 years?
- Have you had at least 4 years of active middle grade experience?
- Has your log-book got the necessary numbers and case mix?

If your answer to all the above questions is 'yes', then it is *very likely* that the consultant will sign your form and give you a good reference.

Preparation for the examination

It is not easy for SAS doctors to form a study group. Experience shows, however, that those who revise alone have a much lower chance of passing than those who revise in groups. It is quite possible for non-training grade doctors in a region to team up and prepare together. Similarly, you should, if possible, join the local Specialist Registrars in their discussion and practice sessions. Careful planning of courses including basic sciences, viva and clinical examination at the zenith of your preparation would help in quick and useful revision of subjects and in presentation skills.

Acquiring knowledge and polishing up your examination and communication skills are the essential steps for most SAS doctors.

Right through your preparation leading to the examination, ask yourself the following questions in your routine clinical practice:

- Am I thorough with my history and examination techniques? Can I finish the history taking and examination in the given time frame required in the examination?
- What investigations are required and why? What is the evidence?
- What antibiotics and other medications are needed? What is the evidence?
- What implants should surgeons use and what is the rationale behind these decisions? What is the evidence?
- Risks and benefits of various common operations including the evidence?
- Postoperative physiotherapy and some details about the rationale of using certain types of splints and other orthotics, etc?
- Be aware of situations and try to have some answers handy for: Consenting a child; consenting a patient who lacks capacity in elective and emergency situation; Jehovah's witness patient; surgery on patients on anticoagulants; immunosuppressive agents in emergency and elective situations; previous surgeries with multiple scars/flaps and dealing with clinical errors

The above questions may come in various situations and may not be a direct question; therefore, some lateral thinking may be necessary. Often, these situations can be practiced during mock sessions. Therefore, the more you do it, the better.

Not all SAS doctors have had the opportunity to work for a consultant with a special interest in paediatric orthopaedic, hand or spinal surgery. You could use your free time or SPA time to fill these gaps. Try to make arrangement to do a clinical attachment in a regional specialist unit such as Paediatric Orthopaedics and Orthopaedic Oncology to gain some working knowledge and experience over a period of 6–8 months. Often, your Consultants are supportive of these ideas and help you to acquire such placements locally.

Such placements will help in observing and acquiring certain specialty specific skills.

Paediatrics

- How to take a history and assess children
- Clubfoot assessment and management
- Developmental dysplasia of the hip (DDH)– How to apply a Pavlik harness, closed and open reductions
- Management of slipped upper femoral epiphysis (SUFE)
- Cerebral palsy
- Congenital deformities
- Inherited disorders (e.g. osteogenesis imperfecta, Marfan's, achondroplasia, etc)
- How to communicate with parents and children and obtain consent

Hand surgery

- Classification and management of swan-neck and boutonnière deformities
- Management of the paralytic hand
- Management of wrist instability
- Congenital deformities and various associated syndromes
- Different hand splints

Spinal surgery

- Segmental anatomy
- Examination of the spine in a given time frame
- Principles of management of spinal fractures
- Cauda equina syndrome
- Different surgical approaches to the spine and spinal decompression

Group study

SAS doctors often work and gain extensive experience in a particular subspecialty over a long period of time. Therefore, there is certainly an opportunity for such SAS doctors with varied subspecialty exposure to group together and share their knowledge and experience whilst preparing for the

examination. An essential component of all group study and practice is positive critiquing; it is no help to a colleague just to say that their performance was good – There is always a way performance could be improved, or if the performance is indeed exceptional to discuss the reasons why such is the case.

Reading core topics and practicing viva/clinical examination

Certain useful tips

- Read all core topics until you understand them very well. There is a difference between being able to reproduce something from a book and understanding the topic – This should ideally be brought out in group discussions
- Discuss these topics with a colleague who is also preparing for the examination or those trainees who have recently passed the examination – The aim is to ensure understanding rather than simple reproduction of facts or drawings (an experienced examiner can spot the difference almost immediately)
- Find out the gaps in your knowledge and skills
- Make sure you have read the curriculum – Some topics on the curriculum, such as orthotics and gait are regularly underprepared by candidates
- Initially topic-based vivas followed by random vivas with emphasis on drawings is helpful
- Read again either the same book or different sources until your understanding improves and makes you feel confident
- When practicing clinical examination, make sure you time yourself and have a check list to ensure you have covered every aspect of relevant history taking and examination
- Working in pairs whilst examining patients helps
- Keep up to date with topics of current interest; read journals and be able to describe any papers either by author, journal and year, or by the centre where the study was done
- Read at least the summary of the National Joint Registry annual report

Delivery

This is a most important step. Your answers to the questions should be clear, crisp and to the point. Take a moment, think about the question asked and frame your answer in a structured way. Practice, practice and practice; this is the key. Practice regularly with either a Consultant you are working for or other helpful colleagues.

If you do not know the answer when practicing, then learn to say 'I don't know' rather than making up answers. You can then go and read about what you didn't know.

In the examination itself never assume you have failed just because a topic went badly; always treat each question as a separate event and do not dwell on the previous one. Many candidates think they have done badly on questions when they have in fact performed satisfactorily, but they can then do badly on the following questions because they are still thinking about the earlier topic and have given up because they think they have already failed.

Final thoughts

What to do if your consultants are unhelpful with either supporting your application or your preparation?

- Reflective practice: Work on a positive note: Discuss the reasons behind the refusal and try to address them
- Then ask the same Consultant or another consultant after a period of reflective practice
- Highlight any deficiencies in your requirements for FRCS (Tr & Orth) examination during your appraisal. Have a personal development plan (PDP) and try and incorporate this in your job plan
- Speak to the Clinical Lead, Clinical Director, Postgraduate Tutor, etc

It is extremely unlikely that your Consultant will be against you taking the exam, unless there is a good reason. If there is a good reason, find out what it is and work on it. Discuss the eligibility criteria for the examination with your consultant and identify any areas of concern or deficiencies highlighted by him/her. Ask for any advice as to how to fill the gaps identified and work positively. If you show an intent and demonstrate a positive attitude, generally the Consultants will extend their support.

The FRCS (Tr & Orth) examination is a very fair examination. The examiners do not know whether you are a formal trainee or SAS doctor. It is very important that you demonstrate the knowledge and clinical skills necessary to pass the examination in a confident manner. The examiner is only looking for a safe, competent and confident candidate with a potential to work as a year 1 Consultant.

Failures are common with FRCS (Tr & Orth) examination. Therefore, don't get disappointed. Reflect on the experiences gained, analyse the mark sheet, identify your weaknesses and strengths. Go for appropriate courses, plan your preparation and try again! When doing so, however, do not assume that you do not need to continue to work on the areas that you 'passed' the first time; there is inevitably some loss of knowledge and skill over time if there is no further practice and there is nothing worse than failing the examination in a different area each time.

If you put in sufficient work and study in a systematic and effective way you will pass one day. Be patient, work hard, believe in yourself. Taking this examination is not only nerve-racking but also very expensive. So, prepare well, keep calm and don't give up.

Chapter 35

Candidates' accounts of the examination

Jibu J. Joseph and Shariff Hazarika

General examination experiences and advice

Firstly, congratulations on reaching this stage in your Orthopaedic training where your Consultants, Training Programme Director feel you are ready to sit the FRCS (Tr & Orth) exam.

However, it is important to realize that you require a huge amount of knowledge to pass this exam and that takes preparation. Although initially buying lots of books can feel good, it will leave you immensely stressed prior to the exam when you have failed to read all of them. Instead, save your money and concentrate on a few core textbooks and know them well.

Studying for the exam takes a lot of time and so put any research projects, audits, other work aside and focus on the exam. All these things can and will wait until you have passed.

> 'The exam is about seeing if you are ready to be a Day 1 General Orthopaedic Consultant in a District General Hospital.'

On the day of your exam you know more about the generality of Orthopaedics than your examiner. The examiners are looking for candidates who have a solid grounding in the principles of Orthopaedic and Trauma surgery, and you are expected to convey to them that you are safe, sensible and logical. Keep this in mind when studying to keep things in perspective. You can't and won't be expected to know everything. The benchmark the examiners have begun referring to is if they would be happy for you to treat an immediate family member of theirs such as a mother or spouse.

> 'Your view of the exam is entirely dependent on how you feel it went and whether you pass. With this in mind, I can say that I felt that both the MCQ and clinicals/vivas were fair, with nothing unexpected.'

It is important to know what works for you when studying and give yourself plenty of time to prepare. For example, if you like to write notes from multiple sources then giving yourself 2 months of preparation time is not realistic as you will not be able to complete the syllabus in time. Also remember that the part 1 (written papers) and part 2 (clinicals and viva) are very different beasts and so your approach will need to be tailored to each.

> 'The part 1 is a pure test of knowledge and requires a different approach to the part 2. The part 2 requires you to take this knowledge and convey it in an organized manner appropriate to the specific situation encountered. Similarly to the part 1, the only way to succeed is to practice.'
>
> 'I studied individually for the part 1 but for the part 2 found it a lot better to study in a group. This allows you to assimilate other peoples knowledge in areas where you may be deficient, bounce ideas of each other, practice answering questions (the whole basis of the part 2) and to have people to take a break from studying with.'
>
> 'The examiners in the part 2 want to know that you can safely manage a case and seemed to concentrate on your approach and thinking rather than detailed knowledge. From my understanding they are meant to be testing higher order thinking rather than facts (which are assessed in part 1).'
>
> 'It's surprising how even describing the simple things can be difficult in the heat of the exam, and you begin to doubt yourself.'

If you are studying in a group for the viva, it is important to make your sessions as useful as possible. You should keep a timer during questioning, sit at a desk with the examiner on one side and the candidate on the other and you should give appropriate and constructive feedback. You are doing your colleagues a disservice if you tell them that their answers are good when they are not.

It is easier to study for the viva than it is to study for the Clinicals. It is a mistake to fail to practise for the Clinicals, as this is often the area that people struggle. If you have been a diligent trainee and gone to specialist clinics throughout your training then you are in a great position. I did not and so had to cram in these sessions when studying for the exam. The key is to prepare and practice basic examination on each other so it gets to the point where you don't have to think about your sequence. When you are in the exam you can then focus on identifying the pathology and demonstrating it. Remember you can only know what is 'abnormal' if you have examined lots of people who are 'normal'.

Don't get hung up on research. You need to score a 6 before you can score an 8 and so first learn the basics of each topic that would allow you to pass. Once you have done this you can then go on to learn 'key' papers that you wish to quote in your exam. If you do decide to quote research make sure you know the paper well and never fabricate research to support your argument. If you are asked about specific papers or research it is because you are doing well and they are trying to push you to a 7 or 8. If you do not know the papers they will not fail you.

You will have spent quite a bit of money by the time you get to part 2 of the exam. A few extra pounds won't kill you, so make sure you book a decent hotel well in advance and ensure it is somewhere quiet. The last thing you need is to be kept up all night by fleas, wedding parties and stag dos.

'Eat well before the exam but don't be too adventurous. If you haven't tasted a lamb vindaloo, I assure you the night before the clinical is not the time to try it.'

Some people like to spend the last minutes before an exam prepping certain topics and others do not. In my experience trying to cram in last minute studying just makes you more stressed but it is important to know what makes you happier in this situation and do that regardless of what the rest of the group you are sitting the exam with does.

'Do what the examiners say *exactly*; they *will* rush and interrupt you. Keep calm. They may hound you but they are simply trying to get you the points you need for the magic '6'. Remember, you can always make points up and no one knows your past scores, so keep your chin up and forget any mistakes as soon as you can. I truly believe they are out to pass you by finding out what you know, not what you don't. Say 'don't know' and they move immediately on.'

'Stick to principles of management for everything, especially when you feel like you don't have a clue and you'll have a fighting chance.'

'The most important piece of advice is to have faith in yourself. If you've done the work, you deserve to pass. Good luck!'

Section 1 – Written papers

The written section comprises a single best answer paper (SBA) paper first followed by an extended matching item (EMI) paper.

The SBA paper lasts 2 hours and 15 minutes and includes 110 multiple-choice questions with 1 correct answer from 5 options. The first 12 questions will be related to a published paper. Prior to starting the exam all candidates must be seated and are given 15 minutes to read the chosen published paper before being allowed to turn over the question booklet and begin answering.

The EMI paper will last 2 hours and 30 minutes. Each question has a theme, such as Biomaterials. You are then given a list of options, a lead in question and the question stems follow. For each question stem you need to select the correct answer from the list of options. Each answer option from the list can be used once, more than once or not at all. In total there are 135 questions, e.g. 40 questions with 3–4 question stems each.

A process of standard setting will be performed after each exam in order to set the pass mark.

Prior practice doing multiple choice questions is essential and there are various books and online resources available. This preparation helps you apply your knowledge and gets you used to answering questions under time pressure.

General comments

'For part 1 I relied heavily on Orthobullets. I exhausted the question bank once and then went back and did the questions I either got wrong or simply guessed correctly the first-time round. This meant I was reading or doing orthobullets for 4–5 months before part 1. Incidentally I found UKITE rather tougher and only scored 74% on it. With this amount of prep I scored 85% on part 1 of the FRCS.'

'Stay calm, watch the clock, read the question and fill in the answer sheet carefully.'

'Do not spend too long on the 12 questions relating to the published paper or on questions you do not know the answer to quickly. Note down questions you want to re-review, complete the paper and then come back to these questions if you have time. Each question is worth 1 mark so you are better to finish the paper and score as many points as possible rather than waste several minutes thinking about one question only to guess the answer and not have time to complete the paper.'

Section 2 – Clinicals and oral

Intermediate cases (15 min)

The candidate will be faced with two intermediate cases, lower limb including spine and upper limb including spine, both lasting 15 minutes. This includes time for history taking, examination and questions from the examiners. This is your chance to shine and show off your clinical skills, but be aware of the time. Fifteen minutes can pass very quickly! Pre-exam preparation is key and you should be able to take a thorough history and perform a relevant systematic clinical examination, which elicits signs that lead you to a diagnosis within 10 minutes. This should give you ample time for questions from the examiners. Always be courteous and polite to the patient and do your utmost to minimize any discomfort you cause to the patient during the examination.

'I found the intermediates quite enjoyable. I had prepared for them well but I think I was also fortunate with the patients I got. They were both relatively straightforward cases and I felt completely comfortable.'

'My examiner harassed me throughout my intermediate and this put me off but the patient and her mother were very encouraging and well versed at giving a relevant history. This helped me pull it together and gave me ample time to take a history, perform a relevant examination and answer questions.'

Painful, valgus knee in young man

'History: 40-year-old mechanic with 6 months' increasing aching in right knee. Multiple injuries aged 17 years old when hit by drunk driver. Open femoral shaft fracture (IM nail), tib/fib fracture (non-operative treatment), PCL repair (patient described an ACL repair – This threw me!). Had been fine for several years, slight limp only.'

'Examination: Multiple confusing scars!! Genu valgum when standing. Muscle wasting. Posterior knee scar – I had to ask what this was for. He said "ACL repair, as I told you before!" This made me feel a bit stupid in front of the examiners, but I politely remarked that I would have found it difficult to reconstruct an ACL through this approach (slight smile from the examiners – At least they realized that I knew the difference!). Reduced ROM, correctible valgus, lax ACL.'

'Discussion: Investigations – They had lost the x-rays (!) so instead I had to comment on a single sagittal MRI slice!'

'Management: Non-operative vs operative. Discussion on femoral/tibial osteotomies. I'm sure we would have gone on to talk about unicompartmental vs total knees but the bell went.'

Rheumatoid with elbow pain and ulnar nerve symptoms

'History: 50-year-old woman with long-standing, well controlled (methotrexate) rheumatoid in hands and wrists. Fell onto right elbow 6 months ago – Told it may be fractured, not sure. Treated in sling for 6 weeks. Since then has had an increasingly stiff, aching elbow with pins and needles along ulnar border of hand.'

'Examination: Very stiff, but not so painful, elbow. Explained I would start with neck and shoulder, etc, but told to concentrate on the elbow. Tinel's very sensitive over cubital tunnel. Neuro exam distally. Motor fine, altered ulnar nerve sensation. Talked through functional assessment of hands (grips, etc).'

'Discussion: Investigations: x-rays (very poor quality). They were pushing me towards nerve conduction studies but I said it was obvious where the pathology was. Diagnosis of tardy ulnar nerve palsy 2 years post injury. I said there was nothing to suggest it was due to cubitus valgus but, apparently, you can't comment on this if the elbow won't extend! Discussed surgical approach for decompression, pros/cons of transposition.'

Forty-four-year-old with femoral fracture

'Fall 9 months ago, bilateral femoral fractures, right IM nail, left LISS plate. Now painful unstable left knee.'

'Examination: Fixed varus painful left knee, stable.'

'X-ray: Varus mal-union/malreduction distal femur. Chat about axes, management, osteotomies, arthroplasty. Problems, bone graft, BMPs (very briefly!)'

Girdlestone arthroplasty

'This was a 49-year-old man with indolent long-term sepsis in Girdlestone after numerous procedures aged 13.'

'Routine history and examination: Poor historian – It took a long time to complete the history.'

'Apart from routine examination I was asked to demonstrate limb length discrepancy. Discussed management in detail up to staged modular tumour stem hip replacement and also what if there was no infection.'

Revision hip arthroplasty

'This was a 40-year-old woman. Bilateral hip problems since early 20s, with left THR and two revisions. GP note read pain in right hip and swelling on left thigh. Not the best historian (kept going on about an accident that is unlikely to have been relevant). Now pain on right. Denied childhood problems. There was ample time for a good history and the examiners faded away in the background, so I had time to get her social and functional history too.'

'Examination: Standing (on crutches); short right leg, flexed at hip and knee, scar from left hip (posterior approach) all way down over knee. No patella. Large fluid-filled swelling over lateral thigh. No infection in scar. Didn't walk her as she couldn't!'

'On bed – Tape to quantify LLD (it was lying on a table in full view – No tricks), swelling on left – Seroma. Thomas' test – Left hip fixed flexion deformity, rest of movements very limited.'

'Discussion: Diagnosis? OA probably due to dysplasia. Probably total femoral replacement on left with patellectomy. Not sure why. What next? X-ray showed dysplasia and severe OA. Spoke about acetabular index and centre edge angle. What operation? Cemented Exeter THR with acetabular augmentation – Spoke a bit about the various ways, e.g. bone graft/mesh, etc. Showed x-ray of left before revision – Describe, modes of failure, Gruen zones, why lysis? Cement properties, e.g. creep.

A 72-year-old man with unstable knee

'Current problem is painful and unstable left knee, worse over the past 6 months.'

'Had a fall 3 years ago while fell-walking and sustained the following injuries:

1. Open left tibia #
2. Closed left femoral condyle # (Hoffa type)
3. Closed right patella fracture
4. Significant head injury – Was in ITU for 3 days.'

'Has had multiple procedures, including skin grafting.'

'Examination: Multiple scars. Commented on significant acquired flat foot with an obvious midfoot break and forefoot abduction. Had a varus thrust while walking. Significant laxity of the medial collateral ligament. (Examiner was not happy with my term 'laxity' and wanted me to say instability – Although instability is a symptom not a sign, I agreed with the examiner.) Range of motion − 5 to 80°.'

'Discussion: Non-operative and operative. Asked me to expand on the operative treatment. He wanted to know if I would request a CT scan, which I did. We talked about ruling out infection – Bloods, aspiration and multiple synovial biopsy. Discussed arthroplasty. I mentioned this is not a straightforward TKR.'

A 45-year-old man with bilateral lower limb polio

'Entire right lower limb was flail. Left lower limb – Some power around hip. Pelvic obliquity. Quadriceps power 0/5. Multiple (five) knee operations. Very painful knee. Good muscle power around ankle and foot. Clearly this was a complex case, and the examiners were 'sympathetic' about it from the way they gave a briefing about the case. I just took quite a detailed history and started the examination with gait, description of deformities, scars and stuck to the look feel and move routine. Moved on to examination of power and specifically left knee, which was his main problem. He basically had a degenerate knee. Discussion went into the x-ray features and management options, which predominantly were non-operative.'

Hip fusion

'This was a 63-year-old woman, a good historian, who had TB ankylosed/fused hip with proximal femoral osteotomy, ipsilateral frame knee, contralateral osteoarthritis and degenerate spine.'

'The case centred round identifying the deformity, limb length discrepancy, fusion and discussion really only about appropriate treatment for osteoarthritis.'

Thoracic outlet

'History was of gradual declining hand function. Altered sensation medial forearm (T1). No pain. Median and ulnar intrinsic

weakness. Just took a medical student style history and did clinical exam of c-spine and upper limb neuro (as per Chesterfield handout). Adsons and Roos tests. Nothing too fancy. Asked for radiographs and NCS. Unfortunately they had the nerve studies! This was a good case because of textbook signs. Got onto talking about tendon transfers for abductor pollicis weakness.'

Rotator cuff arthropathy

'Seventy-one-year-old retired male. History of multiple operations after trauma. Complaining of shoulder pain.'

'Gentle examination as in a lot of pain. Examiner wanted me to elicit specific things, not perform a general shoulder examination.'

'Discussion on x-ray findings, Seebauer classification, treatment (reverse vs extended head, different types of reverse), risks and consent.'

Radial nerve injury following humeral fracture

'History: Young soldier involved with blast injury 6 months previous with open left humeral fracture and radial nerve injury treated with humeral ex fix and radial nerve interposition graft.'

'Examination: Pointed out the obvious fixator and scars. Performed brachial plexus type examination, which showed 4–5 power in ERCB/ERCL. Positive Tinel's sign over extensor wad.'

'Discussion: Discussion focused on management of nerve injuries and nerve recovery. Asked whether I thought there was any (most proximal radial nerve innervated muscles distal to grafting had power), duration of recovery and direction to test Tinel's. I just managed to get in discussion on radial nerve injury tendon transfers before the bell.'

Adolescent idiopathic scoliosis

'History: An 11-year old premenstrual girl with a painful thoracic scoliosis. Curve noticed previous year whilst on holiday (bikini/hair line uneven). Otherwise well. Maternal aunt had a history of scoliosis requiring operative intervention.'

'Examination: Routine scoliosis examination including: Adam's forward bend test, assessing if the curve was correctable with bending and sitting, walking the patient, looking for lower limb deformities (esp. unilateral foot) and neurological examination including abdominal reflexes.'

'Discussion: Shown radiographs and asked to comment. Then discussion focused on use of braces, risk factors for progressive curves, indications for surgery in this patient, risks of surgery, consenting the patient.'

'At the end I thanked the patient and her mother.'

Hip and knee OA with distal tibial mal-union

'History: GP letter on middle-aged lady with severe left knee pain and previous ankle fracture that the GP did not think was relevant. My history revealed that the lady's main complaint was of severe left knee pain disturbing her quality of life and giving symptoms at night. She also complained of stiffness in the left hip but little pain. She informed me that 30 years' ago she fractured her ankle, this was treated conservatively and has not given her any problems.'

'Examination: The cubicle was very small and so I asked the patient to step out into the corridor in order to see her walk. Immediately it became apparent that she had an external rotation and varus deformity of her tibia at the distal meta-diaphyseal junction and an antalgic gait. I then examined her spine, hip, knee, ankle and assessed the extent of the rotational deformity. Revealed a very stiff left hip, stiff and painful left knee and the aforementioned tibial deformity.'

'Radiographs: Demonstrated an arthritic hip, arthritic knee and a healed tibial fracture with a varus and external rotation deformity.'

'Discussion: Focused on which of her various pathologies I would treat and the order of correction. Asked about difficulties of doing a knee replacement in someone with tibial deformity and discussion on use of navigation in joint arthroplasty. Asked about methods of correcting distal tibial deformities and the risks/merits of the different methods.'

Short cases (5 min)

In the exam the short cases will be divided into two sections – Upper limb and lower limb, with each lasting 15 minutes. Two examiners will take you through each section. You will roughly spend 5 minutes with each case; this includes discussion and questions from the examiner. This realistically gives the candidate around 3 minutes to assess the patient and offer a diagnosis. Some cases will be straightforward and others a bit more complex. Usually the examiners will guide you into what sign to elicit or body part to examine, e.g. 'Please can you examine this lady's feet and tell me what you see; what do you think the diagnosis is?' However, candidates often feel bewildered after the short cases and not quite sure what the examiner was expecting. As stated earlier, if you feel things haven't gone well, clear your mind and focus on the next case.

'Short cases seemed a bit rushed and if I failed them I'm not sure what I would do differently – Probably be a bit more aggressive and proactive.'

'All seemed very simple – I kept feeling as though I was missing something. Keep in mind that the examiners want you to make sensible decisions and not to overcomplicate things. They are not trying to catch you out.'

'The short cases pass very quickly. I tried to keep it simple and described what I saw.'

'These are stressful. You will (and should) try to stick to "look, feel, move", but invariably they'll interrupt or ask you to do something specific. Listen to the examiner carefully and answer directly.'

'In the short cases, I tried to talk as much as I could about each case as I was examining, trying to demonstrate lateral thinking, e.g. mentioning osteophytes in OA knee, and how they would affect surgical planning.'

Upper limb short cases

Painful lump on ulnar border of little finger

'Forty-year-old woman with 6-month history of exquisitely sensitive lump on ulnar border of little finger. Trapped finger in a deckchair as a child. Brief history, examination – Very little to see.'
'Discussion of investigation (USS), management (excision), skin incisions and complications.'

Rotator cuff tear/arthropathy, AC joint arthritis

'Seventy-year-old woman with a painful shoulder. No history allowed. Difficult examination as the examiner kept interrupting me and asking questions about things I had not yet got round to examining. Felt sorry for the patient as she was clearly very uncomfortable and it was difficult not to cause pain. I felt rushed and pressurised. Did not get as far as discussing management.'

Distal radius mal-union with wrist pain

'Fifty-year-old woman with distal radius treated non-operatively 2 years ago. Brief history and basic examination. Asked what I thought was the problem. Fortunately, I got the right answer. Discussed initial x-rays and original management options. Discussed current x-rays and management options (corrective radial osteotomy).'

Rheumatoid hand

'Asked to describe deformities, examine, volar subluxed index MCP, differentiate tendon rupture, subluxation. Management based on function, MCP replacement, surgical goals.'

Rotator cuff tear

'Obvious massive cuff tear and biceps rupture. Discussion of investigation and management.'

Rheumatoid hand

'Description of deformity, scars, etc. Discussion of thumb deformity and pathogenesis.'

'Examination of thumb, classification of thumb deformities. What are different types of grips? Would MPJ arthrodesis help?'

Dupuytren's disease

'Bilateral extensive Dupuytren's in a diabetic 65-year-old man. Examination in detail showed boutonnière deformity of his little finger. Asked about diathesis and associations, risks and complications and outcome. Then examine and describe. Noted calluses on PIPJ (steel worker), not Garrod's pads and told them that. Noted thenar commissural band. Management – I said review as risk of rapid progression (NIDDM, first web band, etc).'

'Spot diagnosis. Why Dupuytren's? Describe deformities. What would give worse prognosis? Wanted everything to do with diathesis – Didn't let up! Surgical approaches. Consent the patients about risks/benefits/success rates.'

'Bowstringing of flexor tendon to ring finger following previous surgery . . . secondary to rupture of the pulleys.'

Hand osteoarthritis

'Old lady knitting happily as I walked in: Spot diagnosis and describe the features. Osteoarthritis with Heberden's nodes and adduction of thumb with CMC joint subluxation. What next? X-ray, three views – Had them – Describe. Eaton grade IV base of thumb changes. Treatment? Knitting happily – Is there pain or loss of function? Went through non-operative first. If needed, surgery? Trapeziectomy, but aware may need more because of STT involvement.'

Brachial plexus injury

'Asked to examine patient's right upper limb, concentrating on the neurological examination of his hand – He had profound wasting of his intrinsic, thenar and hypothenar muscles with less than grade 3 power of median and ulnar nerve-innervated muscles. Mentioned I will examine cervical spine.'

'Injury from C5 to T1. Only functioning muscle was biceps (power 3/5). Described the wasting from shoulder to hand. I asked the patient if they had a loss of sensation (sensory deficit) in the upper limb and then I started examining muscle power methodically from trapezius, periscapular muscles, rotator cuff and came to biceps. Time was up. Realized I had missed Horner's on inspection!'

'Weak, painful shoulder with numbness, neck scar from lump excision. Asked to examine shoulder movements, then asked diagnosis. Patient had full abduction, restricted IR and ER. Said likely to be a neurological lesion due to scar and numbness, but difficult as no chance to do any further examination. Then quiz on rotator cuff. I found out later that patient had C6 root problem and previous excision of neurilemoma – Didn't feel I was given much chance to examine.'

Congenital radioulnar synostosis

'Examine forearms. Talk about deformity. Diagnosis. What investigations (x-rays!). Treatment – Conservative. What operation would you do if operative? Benefits/risks of osteotomy vs fascia lata interposition. What position would you fuse?'

Bilateral cleft hand deformities

'Forty-year-old woman with congenital hand deformities. Recent surgery to left little finger, which turned out to be for Dupuytren's disease. I described the deformities and mentioned Swanson classification. They seemed happy with this but didn't want me to recite it. I performed a hand functional assessment on the patient and asked if she currently had any problems with her hand – She said not. I concluded that there is no need for intervention based on my findings. They were satisfied with this and moved me on.'
'As I was leaving they asked me about Mendelian inheritance and the possibility of her children having the same problems.'

Cubitus varus (adult)

'Describe deformity and old lateral scars. Neuro exam normal. Function problems (nil). What other problems would he have? Wanted instability, got there eventually. X-ray of recent posterior dislocation, hence instability! Clinical testing for varus/valgus instability, including posterolateral pivot shift.'

Ankylosing spondylitis

'Gentleman with thoracic kyphosis on examination, went to palpate and told not to as too sore. Virtually no spinal movements. Asked what I thought and said ankylosing spondylitis, examiner happy. Asked to examine shoulder – Reduced and painful movements that I abandoned mid way through and said patient too sore to assess. Offered monoarthritis associated with ankylosing spondylitis when asked directly what it was (no idea). Finished with discussion on management. I offered conservative physiotherapy and rhemuatology review. He wanted to chat about surgery so I offered to examine hips for flexion contracture and SI

joints before deciding on surgery of spine. Happy with THR in contracture before spinal osteotomy. Asked me about osteotomies – Saved by the bell.'

Congenital ulna dysplasia/absence with neurofibromatosis type 1

'A guy with Nf-1, something I initially didn't pick up on. Asked to examine the left forearm. No palpable ulna. No forearm rotation. X-rays showed a surgically created single bone forearm. They weren't interested in classification or anything else. Once it dawned on me that this guy had Nf-1 they asked what other skeletal manifestations I'd look for – Scoliosis and bowing/pseudarthrosis of tibia. Was asked to describe the difference between Nf dystrophic curve and non-dystrophic spinal curve.'

Miscellaneous

'Ulnar claw hand: Causes, examine, paradox.'
'Degenerate elbow: Indications for and when TER.'
'Nail–patella syndrome with bilateral radial head dislocations.'
'Fascioscapulohumeral dystrophy.'
'Winging of the shoulder.'

Lower limb short cases

Hereditary multiple exostoses with leg/foot deformities

'Clinical examination of right foot and ankle. Discussion on treatment of osteochondromas in general.'

Infected non-union of tibia

'Bilateral 3B fractures 15 years ago. Both flaps had healed well. Was still getting pain on left side. Small healed sinus. X-rays showed hypertrophic non-union. Asked to describe pathophysiology of osteomyelitis and options for treatment in this case. Said I'd refer to frame surgeon.'

Previous open pilon – treated with frame and ankle fusion

'Took me a while to get this one, multiple scars and picked up on fused ankle but given Hx of previous motorcycle accident and saw the circumferential pin scars from previous circular frame. In addition, multiple scars around ankle, missed that his fibula wasn't there on palpation, had a horrible non-surgical looking scar medial which I was asked about and the examiner confirmed it was from an open fracture. Asked what the lateral longitudinal scar could be and said fibula plating – Had missed the fact he didn't have a fibula. Managed to say he had likely had ankle fusion using fibula as graft. Not a great start to the lower limb shorts.'

Bilateral surgically corrected clubfoot

'Sixteen-year-old with short stature and bilateral feet deformities. Far from classical appearance, had multiple lesser toe deformities and scars with splayed forefoot and collapsed medial arches. Neutral hindfoot and poor musculature of calves asked what I thought it was after inspection and suggested surgical correction of clubfoot based on the lateral and medial hindfoot scars. Examiner confirmed the diagnosis and then asked me about management of the lesser toe deformities. Finished with Ponseti discussion.'

Ankle – Patient described anteromedial joint pain

'Examine ankle – Tender anteromedial joint + malleolus. Not really tender over tib post but apparently that was the diagnosis! Quick chat about staging/management of tib post tendonitis.'

Marfan's syndrome

'Seven-foot tall (ish)! Asked to examine patellofemoral joints. J sign, patellar tilt, apprehension, etc, Beighton's index.'

Hallux valgus with overriding second toe

'Fifty-year-old woman with pain over the second toe PIPJ. Brief history – Not bothered about hallux valgus. Full examination. Discussion of management options – Correction of hallux valgus first or just deal with second toe. Reviewed x-rays – These didn't look nearly as bad as the clinical picture (probably not weight-bearing views). This case was rushed but I managed to cover a lot.'

Painful hip

'Forty-year-old male manual labourer with 3 years of left hip pain, much worse over past 3/12. Brief history (but took too long). Examination – Didn't get through it all. Forgot Thomas' test. After the bell rang, I was asked the diagnosis. I said either OA or AVN.'

Polio deformity correction

'Sixty-five-year-old woman who had polio as a child, developed increasing hip/knee pain over the past few years, and who had just finished a 24-month course of corrective management. She had femoral correction over a nail, subsequently removed. She had tibial correction with a Taylor Spatial Frame. This case had almost no examination at all – Just a brief history followed by a good discussion on the principles of deformity correction. I was asked how I would counsel a patient prior to embarking on a prolonged management course (length of treatment, pin site infection, rehab, stiffness, complications, etc).'

OA varus knee

'Discussion of deformity correction, etc.'

Adolescent scoliosis

'Described the pattern of deformity, balance, role of MRI in certain situations.'

Lateral tibial plateau fracture

'Split depression injury, acute patient in back slab with only toes seen. Acute management, assessment of neurovascular status and compartment syndrome. Classification and management of plateau fractures. Largely discussion except looking at toes.'

Middle-aged woman with bilateral pes planus

'Short first metatarsal (had 'bunion surgery' 20 years ago) and cock-up deformity of big toe. Valgus heels which corrected when going up on toes, said, therefore, mobile with functioning tib post. Noted medial scars over first metatarsal (not obvious, but I looked for them). Made her walk and said, flat foot gait with poor heel strike and no toe-off on big toe (stayed extended). Felt ankle and foot bony landmarks and swelling around midfoot and identified

region of tenderness. Moved ankle and subtalar joints to show they were mobile. Passive correction of first MTP possible.'

A 16-year-old boy with rigid pes planovalgus

'Asked to go straight into examination. Again, just told them what it looked like and got him to walk. His heels didn't correct on tiptoes and clinically had no movement in the subtalar joint that was painful laterally when stressed. They wanted diagnosis – Tarsal coalition. What next? X-ray. Showed me and asked to describe – Pretty barn door bony bar between talus and calcaneum.'

A 65-year-old man with painful, deformed knee

'Told to go straight to examination (told it was long-standing and no longer pain-controlled). Stood him – Obvious flex and varus deformity and quads wasting (forgot to measure later, but they didn't seem to mind!). Walked him – Antalgic, and knee never fully straightened.'

'On couch quickly did Thomas' test to prove not in hip then confirmed fixed flex deformity and partially correctable varus. Medial and PF joint joint tenderness. PFJ crepitus on ROM, which was restricted.'

'Diagnosis: OA. What next – x-ray. Showed me: Medial OA and PF joint OA. Said what I saw (including joint space narrowing, sclerosis. Treatment? TKR. Why not uni? Answer: PF joint OA and FFD.'

Arthrogryposis

'Middle-aged woman with multiple scars. Thankfully I was informed by the examiner that this is a difficult case and that they were only interested in my approach.'

Neuromuscular foot deformity in child

'Unilateral equinus/cavovarus deformity. Describe deformity + gait. Correctability and where is the problem? Had a muscle biopsy (posterior calf scar). Put up MRIs of lower limb! – Mass in the calf. Examiner did say it was complex and they didn't have an answer for diagnosis as yet!'

Complex limb deformity

'Gross valgus knee and failed THRs. Chronic osteomyelitis tibia. Very stiff knee was all he wanted (fixed flexion 30°, fixed valgus 30°). Valgus approach to the knee, all the releases, etc. X-ray – Spot diagnosis osteopetrosis, with two failed Charnleys – Both fractured!'

Bilateral below-knee amputee

'Which stump is better than the other? One stump looked ideal length, healed scar, firm, etc, but very painful due to neuroma. Other stump had a vertical scar, previous infection, some redness, very short stump with fixed contracture. He also had psoriatic patch.'

ACL deficiency

'Gait – Look feel, move and special tests routine.'

Adolescent girl with bilateral hallux valgus and flat feet

'Gait, double/single leg stance (after prompting by examiner), spine exam, assessment of hallx valgus. Beighton scoring.'

The orals (viva voce)

In this section you will experience four orals lasting 30 minutes each: These include trauma, adult elective orthopaedics, hands and paediatrics, and applied basic science. The order is variable. During each viva, one examiner will question you for 15 minutes whilst the other one marks you. They will then change roles. You will usually have a break between each one to have a drink of water and clear your mind. The orals in general pass very quickly. Most candidates will be taken through a huge range of topics so it is impossible to know everything. Try not to mention anything that you can't talk about. Nerves can get the better of you but if you do say something stupid, quickly retract it and apologise. The examiners will understand the stress you are under and are, after all, human too! When answering, start with the basics and try to keep your answers focused and organized; this takes practice.

The examiners are generally helpful and nice but can be easily annoyed with little patience for nonsense. Overall, there is a general consensus that the exam is fair and that the examiners are not there to trick you. If you don't understand a question, say so and ask the examiner to repeat it. If you feel that you have a bad session, ignore it, clear your head and move on. This is difficult to do, but essential. Not all parts of the exam will go smoothly, but usually you will have done much better than you thought. If you don't know the answer, say you don't know. The examiner will either help you or move on. They are there to find out what you know. If you find an examiner difficult or awkward, just remember he is not the one marking that section.

Trauma oral

General comments

'They took me through 7 or 8 scenarios of fractures in 5 minutes. Bang-bang, no x-rays, just verbal discussion as if you are called by SHO A&E describing patients and x-rays.'

'One examiner disagreed with most things in this oral and asked for evidence for almost everything. The most stressful oral of all. They covered enormous ground on various fractures.'

'I almost enjoyed this viva! Everything flowed nicely and was discussed in a very informal way, as you would in a relaxed trauma meeting or clinic.'

'This is your chance to shine and score highly.'

Comminuted intertrochanteric fracture

'Options for treatment: Sliding hip screw with or without additional trochanteric plate, Cephalo-medullary nail.'

'Treatment of a failed DHS.'

Spinal fractures

'Classifications, types of spinal cord injury, initial treatment, difference between neurogenic and spinal shock, indications for surgery, associated injuries including specific intra-abdominal injuries at different levels. Asked about papers guiding treatment and papers guiding use of steroids in spinal injuries (NASCIS trials, BOA guidelines).'

Open distal femur fracture

'Emergent management of open wound, Gustilo and Anderson classification in detail. Management of fracture – Discussed temporary ex-fix and definitive fixation. Talked through principles of management of intra-articular bit and then metaphyseal/diaphyseal – Direct, open, absolute/length, alignment, rotation, bridging – He seemed to like that, before saying that I would use LISS or equivalent.'

Dislocated elbow with radial head + coronoid

'Terrible triad, emergent and definitive management, including surgical approach, rationale for reconstruction or replacement.'

Spiral proximal femoral fracture in 8-year-old

'Options – Talked about traction, time to heal (I said 6 weeks originally and was bargained down!), operative options – Why IM nail inappropriate; said I would plate if operative but would prefer non-operative.'

An 8-day-old, locked post dislocation of humerus

'Given AP x-ray only. Discuss open reduction in detail, detailed deltopectoral approach and management of reverse Hill–Sachs with lesser tuberosity advancement.'

Supracondylar elbow fracture in child

'Usual management principles with pulseless hand, etc.'

Pelvis and acetabular fractures

'Early management principles.'

Talar neck fracture

'Closed Hawkins 3 fracture. Discussed management, AVN, blood supply. Surgical approaches and fixation principles. Postop management – weight-bearing status, Hawkins' sign, likely surgery in future. Dorsomedial approach to fix.'

Small fragment/locking screws

'Recognise and what drill size to use.'

Use of a dynamic compression plate (DCP)

'Demonstrate two methods of achieving compression in detail.'

Fracture dislocation proximal humerus

'Plan, surgical approach.'

Acetabular central fracture–dislocation on x-ray

'What would you do? Showed me a CT – Shattered walls and columns beyond surgical repair. Management options: Detailed discussion about non-operative treatment.'

Displaced intracapsular fracture neck of femur in young patient

'Management options and evidence for it. What classification, why, reliability, levels of evidence, etc. Management and evidence for it. Osteoporosis – Risk factors.'

Tibial plateau split depression fracture

'Shown CT. Discussed operative treatment and bone graft. Showed me less than satisfactory postop x-ray, probably one of his own patients, so carefully criticized it.'

Cervical spine fracture dislocation

'X-ray of C6/7 fracture dislocation. Discussed management, ATLS®, etc. Investigations? Examiner wanted to see if I could manage this patient safely and appropriately prior to transfer to a definitive centre. Early involvement of anaesthetist, appropriate imaging, etc. Discussed spinal levels, spinal shock.'

Tibial non-union

'X-ray of tibia nailed elsewhere, gone on to non-union in valgus with broken nail. Critiqued x-ray and poor technique. Options, problems (e.g. nail removal), approach. Bone graft + plate vs re-nail.'

Midfoot fracture dislocation

'Described x-ray. Explained normal patterns (Lisfranc types) but realized that this injury did not involve the Lisfranc ligament. Management (soft tissues before surgery), CT, surgical approach/principles.'

Open tibial fracture

'Management of open fractures: In A&E, principles of surgical management, antibiotics, dressings, ex-fix. Awareness of recent guidelines (BOA/BOAST). I wanted to talk about compartment syndrome but ran out of time.'

'Grade IIIB open tibial fracture (photo and x-ray) extending to but not into ankle. Large skin defect with periosteal stripping and some mud on medial side of tibia. Classification? No plastics on site – Went through ATLS®, open fracture initial management in A&E, including letting plastics at next hospital know. Management? I debrided and ex-fixed and transferred to unit with plastics: Wanted details of where to put pins. Bridge ankle or not? Came back with pin tract infections! What definitive, e.g. frame or complete construct, avoid pins where pedicle of flap may be, so check plastics op note.'

Posterior shoulder dislocation

'AP x-ray of shoulder – Lightbulb sign (fell down stairs). You're in theatre; what to do – Ask for axillary view. Radiographer not keen – Went down to department myself. Confirms posterior dislocation with huge Hill–Sachs. Management: Told them I'd not let A&E reduce – Risk of leaving the head behind. Showed me x-ray of just that – What now? Short discussion on ORIF or hemi. Risk AVN.'

Cauda equina syndrome

'GP call – 36-year-old man in bed with back and bilateral leg pain, 3-week history, now worse and not passing urine. What do you tell him? Send to A&E as emergency and I'll see on arrival – Suspect cauda equina syndrome. Wanted me to go through full history and exam and investigations. Established no anal tone or sensation, showed me the MRI scan I asked for, huge disc prolapse. Wanted me to describe MR in detail, including T1 or T2. What now? Surgery. Tonight? Surgical approach.'

Comminuted intra-articular fracture of the distal humerus in a 65-year-old woman

'Investigation, treatment options, principles of treating this fracture, positioning of patient, surgical exposure (details of olecranon osteotomy). How to manage the same injury non-operatively, how long in plaster, when to mobilize, etc.'

Neer's type 2 fracture of lateral end of the clavicle

'Treatment options. Said I would fix it with clavicular hook plate. Details of positioning the patient, incision, exposure, surgical details (having done this before I was able answer the details of this). Other surgical options, mentioned Weaver–Dunn.'

Comminuted extra-articular fracture of the distal fourth tibia and fibula (football injury)

'Significant abrasion and contamination with soil but no open wounds. Clinical examination, further investigation to exclude intra-articular extension, talked about staged approach (joint spanning ex-fix to allow space to care for the soft tissue followed by definitive fixation); either conversion to hybrid ex-fix or to plate tib and fib (not suitable for IM nail).'

Polytrauma/open fracture forearm

'How would you treat this patient? Had been inadequately resuscitated and also had open femoral fracture which I picked up from history/examination and secondary survey. Standard ATLS® stuff. X-ray of massively comminuted open distal one-third forearm fracture from RTA with joint intact but smashed radius/ulna. Classification and treatment principles of a compound wound. What would you do for this (spanning ex-fix)? X-ray of spanning ex-fix with very small pins, one of which was in the fracture site. Discuss this x-ray – Essentially saying why it was suboptimal. What would you do next? Shown picture of long bridging plate now in same patient with large bone loss. What would you do next? Prognosis and timespan to healing. What could influence this?'

Spiral closed humeral shaft fracture

'Diagnosis. Describe fracture pattern. How would you treat? Comes back with fracture ulcerating out of lateral arm. What would you do next?'

Achilles tendon rupture

'Clinical photo of prone patient – diagnosis? What are the treatment options? Who would you treat operatively and non-operatively? How would you treat operatively? Draw out a percutaneous repair. What dangers are there to percutaneous repair? How would you rehabilitate?'

Jefferson fracture

'Tiny crack in C1 but large soft-tissue swelling anterior to C1. Diagnosis? What other views (obvious on Peg view). Discuss CT findings. How would you treat? If delay to spinal centre by 7 days what would you do? Why do C1 fractures not present with cord injury? How would you treat operatively?'

Acetabular and pelvic ring fracture

'Diagnosis? What further views? Describe what Judet views show? Classify pelvic fractures. What pelvic fracture configurations have a risk of arterial injury? Discussion of emergent management of pelvic fractures. What pelvis ex-fix would you use and how do you put it on? What controversies are there with an anterior frame?'

Hoffa fracture

'Coronal fracture of medial femoral condyle (Hoffa fracture) with Segond fracture in same knee. What investigation? MRI to exclude ACL injury. Management principles of intra-articular fracture.'

Mason III radial head fracture

'Assessment? Will exclude MCL injury/Essex Lopresti as may be part of fracture dislocation. Treatment options. Reconstruction vs excision vs replacement.'

Transverse patellar fracture

'How would you fix? Tension-band wiring (TBW). Principle of TBW. Draw picture. Where else TBW?'

Lisfranc fracture/dislocation

'Initial assessment, definitive management, timing of surgery, surgical approaches and methods of fixation, complications and outcomes.'

Basic science oral

General comments

'The examiners managed to take me through a huge amount in 15 minutes, scratching the surface to seemingly check I could say something about everything. We got through, among other things: Draw cartilage and proteoglycans, cartilage injury, laminar flow, prophylactic antibiotics, hip FBD and tourniquets. The second examiner went through topics that included stress–strain curves for various materials, creep/stress relaxation, nerve injury, alignment of TKR, a failed tibial base plate, metal fatigue and properties of titanium.'

'This viva seemed very straightforward – I kept on wondering what I was missing but it seemed they just wanted to cover a lot of basic stuff. No tricks. It flowed nicely from one subject into another.'

'Shown a photo of a theatre with no laminar flow. Told was a new consultant, had two TKRs on next day in there. What do I think? Spent the first 15 minutes of viva *chatting* about laminar flow, theatre design, theatre discipline, antibiotic prophylaxis, ring fencing elective beds, washing hands . . . (you can guess the rest).'

'There was a lot of "What's the evidence for that?" during each bit. I was never guided, he just kept quiet, so I'd move on to next bit about reducing infection as I thought of it, trying to stick to BOA guidelines for arthroplasty.'

Anatomy and approaches

'Shoulder anatomy: draw glenoid and ligaments, function, stabilisers, joint reaction forces (with patient holding magnum of champagne – Figured I was doing OK!).'

'Meniscus: draw, structure, function, contents, hoop stresses.'

'Deltopectoral approach: step-by-step relevant anatomy. How to make it extensile.'

'Approaches to hip: had to list them and then when I got to anterior had to describe in detail.'

'Anterior approaches to knee: step-by-step. All, including anterolateral, where do you use them what are advantages and disadvantages, etc?'

'Surgical approaches to proximal one-third of radius: Complications?'

'Deltopectoral approach.'

'Anterior Henry's approach to the forearm to expose the midshaft and proximal shaft radius fracture. What plate would I use for forearm (DCP)?'

'Draw a physeal plate. Mention one or two conditions affecting each zone. Which zone is involved in physeal injury?'

'Axial mid-thigh transection: Label muscles/nerves/vessels.'

'Axial mid-tibial transection: Label muscles/nerves/vessels.'

'How and where would you do a fasciotomy for compartment syndrome?'

'Which approach for open reduction of DDH? Smith–Petersen: Describe the approach, including incision, internervous plane, structures at risk, etc.'

'Cartilage: Structure, lubrication, changes with age and OA.'

'Posterolateral approach to the ankle.'

'Structure and function of skeletal muscle including "sliding filament theory"'.

'Identify nerves on an unlabelled brachial plexus picture.'

'Compartment syndrome of the anatomical leg and fasciotomies (BAPRAS/BOAST guideline).'

Materials and implants

Uncemented THR stem

'Talk about titanium, hydroxyapatite, osteoinduction, conduction, porosity.'

Loose TKR

'Discussion of wear, mechanics of loosening and biology of osteolysis.'

'Discussion about cement – What is it, materials, properties, etc. Wedges for reconstruction of tibial defect; how wedges work; why wedges and not cement for build up, etc.'

Trauma implants

'Draw a diagram of a screw, then an interfragmentary screw. How it works step by step? How would you put it in?'

'Picture of cortical and cancellous screws: Explain. Cannulated screws. Flutes, self-tapping, reverse cutting.'

'Describe how a dynamic compression plate (DCP) works with drawing. Principles of different modes of plate fixation (bridging, compression, etc). Given two types of self-tapping screws (one normal, one reverse cutting as well as self-tapping) – Describe these implants. Why would you use a reverse self-cutting screw? Given dynamic condylar screw (DCS) and dynamic hip screw (DHS). What are these implants? Describe the differences. How would you apply them (exactly, with order of screw placement and why)? What are the principles and design specifications of a compression plate?'

'Shown radiograph of DCS in a proximal femur and asked where it would fail if there was a non-union then shown radiograph of plate broken and discussion around metal fatigue failure.'

'Intra-medullary nails – working length with different fracture patterns, reaming, locking, bending and torsional rigidity.'

Materials

'Explain how you would choose your ideal knee arthroplasty implant and why. Factors that affect function, wear, poly thickness.'

'Ceramics – What they are, their structure and the proprieties they have.'

'Titanium – given various implants (tibial base plate, femoral head, femoral stem, plate, nail) and asked what were the good and bad properties of each.'

Arthroplasty

'Given fractured femoral component of Oxford partial knee arthroplasty and worn tibial component. Why? In depth basic science about stress risers and fatigue of metal (anterior well cemented, posterior not), interdigitation of cement, etc. Why would it occur? During insertion of component.'

Cement

'Constituents of bone cement.'

'Different viscosities of bone cement and their properties/uses.'

'Visco-elastic properties of bone cement.'

'Picture of broken cement mantle – Reasons for this, mantle thickness, asked what would you inform your juniors – Too little or too much cement? I replied optimum (both examiners laughed).'

Tribology

'What is lubrication? What lubrication is in a native joint? How does this work? What lubrication is in an artificial joint? How does this work? Tell me about weeping lubrication.'

'Failed long-stem TKR: What failure pattern and SN curve.' (**S**tress σ; **N**umber of cycles *N*; an SN curve for a material defines alternating stress values vs the number of cycles required to cause failure at a given stress ratio.)

'Charnley THR: Poly cup: What is poly, manufacture advances, wear; what is stem made from, stress strain of stem.

'Poly wear in TKR: Discuss all factors.'

'Corrosion – types, why it occurs, methods of prevention.'

Statistics

'Design trial to compare bone substitutes. Randomised controlled trials: How to calculate power, effect size, type I and II errors, significance, *t*-tests.'

'How would you set up a trial to compare two different implants?'

'What is sensitivity? If you are designing a test, what would you want it to be – Sensitive or specific? Draw the table (disease/test table).'

'What is a Forrest plot and what do the different parts represent?'

Radiology

'MRI: How does it work including how it converts signal into an image ? What is the difference in T1 and T2?'

'DEXA: What is it, how does it work, interpret the results, Rx?'

'X-ray: What is it, how do you make an x-ray?'

Biomechanics

Hip

'Draw free body diagram of hip showing joint reaction force. Draw how a walking stick affects it.'

Knee arthroplasty

'What angle do you put in a TKR, why? Draw mechanical axis, etc (wanted great detail on difference between mechanical axis and anatomical axis). Why is trochanter lateral to hip joint?'

'Stress–strain curve: Label all the points, axis and nomenclature and describe what the areas underneath signify. What is hardness?'

'Stress–strain curve of materials with three different moduli of elasticity. Pick three items from a display in front of me that match those stress–strain curves. Describe their material properties (ceramic/bone/ligament).'

'SN curve: What is this, what does it mean and label the axes. Relate this to both THR and TKR.'

'Explain viscoelasticity. Discuss creep/hysteresis/stress relaxation.'

Prosthetics

'Pictures of Olympic runner's BK prosthesis, young patient's BK prosthesis with ankle articulation, old patient's BK non-articulating prosthesis: Talk about these three prostheses (design and components, etc) and why they are different. What would you choose for a particular patient group?'

Elbow

'Draw free body diagram of elbow. Asked about joint reaction force.'

Foot and ankle

'Free-body diagram of foot and ankle.'

'Picture of foot force plate analysis: what is this picture?'

'Picture of Usain–Bolts shoes: difference between running gait and walking?'

Miscellaneous topics

Osteoporosis

'Define osteoporosis. How do you measure it? How does DEXA work? Given a DEXA scan to report. How do you treat? What is your hospital's osteoporosis policy? What are the NICE guidelines for osteoporosis management?'

Bacteriology

'Shown picture of an agar plate: Explain. Which bacteria?'

'Does your hospital have an infection control policy? What is it? Shown a slide of plated agar with inoculation and antibiotic sensitivities test – What is this, which is sensitive? How do bacteria become resistant (wanted all the modes!)? How do viruses aid bacterial resistance? Draw a bacteria. Describe the mechanism of action of antibiotics in relation to this picture – Bell went!'

Bone healing

'Primary vs secondary. Fractures. Relative/absolute stability.'

'Slide of diaphyseal fracture with callus formation – Asked about primary and secondary bone healing, types of callus, cutting cones.'

'Describe the motor end plate.'

Botox

'What is botox and what is its mechanism of action and clinical applications?'

Nerve injury

'Classifications, Wallerian degeneration, re-growth, Tinel's, repair, graft.'

'Principles of tendon transfer including when you would consider this rather than nerve graft.'

Clotting cascade

'Draw the cascade, explain points where different anticoagulants take effect and coagulopathies occur.'

'Describe the treatment of major haemorrhage.'

Common postoperative electrolyte imbalances

'Hyponatraemia: likely causes postop, investigation and treatment.'

Hand and paediatric oral

General comments

'The paeds examiner repeatedly said things like, 'the mother is very concerned about the appearance – She wants something done'. This is to see if you stray from your non-operative decision. Do not fall into this trap!'

'I was dreading this section but actually was asked about very straightforward and common paediatric cases which I knew well. Doing attachments in paediatrics and hand surgery gave me confidence.'

What candidates were asked (paeds)

Supracondylar humerus fracture

'Diagnosis. How would you manage from first meeting the child to definitive stabilisation (everything). What nerves are damaged and how to test in an uncooperative child? Scenario with no pulse, perfused hand. Scenario with no pulse, no perfusion. Anterior approach to distal humerus/elbow. Late complications. Ulnar nerve lesion – What do you do?'

Perthes' disease

'Describe the x-ray, differential diagnosis, treatment aims. Containment of femoral head – Operative and non-operative.'

Osteomyelitis distal femur

'Describe x-ray, differential diagnosis (I went for Ewing's but was told infection), why metaphyseal, management, what organisms, what antibiotics, when surgical management?'

Unilateral pes cavus

'Photograph – Discuss examination, investigations. Managed to say MRI before examining the back – Picture of hairy patch and x-ray of spina bifida occulta!'

Hip dysplasia

'Late presenting, hip arthrogram – Describe the features on the arthrogram in some detail.'

'Picture of a Pavlik harness. Discussion on screening manoeuvres, USS, Mx after failed conservative measures. I fell into the trap of doing a closed reduction after 4 weeks of an irreducible hip – Wrong!'

Curly toe

'Picture of 3-year-old with curly little toes. Discussed management (or not!) with managing parental concerns.'

Gallows traction

'Picture: What is it, what is it used for, from what age you should not use it and why? Malunited femur: Acceptable limits in children – Why it is acceptable?'

Ollier's disease

'Clinical photograph and x-ray, discussed about Ollier's, Maffucci's and treatment of enchondroma.'

'Diagnosis? What is the genetic transmission? Principles of autosomal dominance/recession and draw out the typical penetration. Counsel parents on the risk of transmission based on various scenarios. What other genetic orthopaedic AD diseases did I know?'

Tibial bowing

'X-ray of posteromedial bowing. Mentioned the three types but only discussed this one. Parental concerns. I was asked about associations and eventually realized that the examiner wanted to talk about leg length discrepancy. I happily discussed this when I realized.'

Slipped capital femoral epiphysis

'Photo of 13-year-old boy: Obese, immature features. Spot diagnosis.'

'Classification: Said Loder, what is the significance? (Unstable 50% AVN.) *Very* gentle manipulation may be attempted to improve position as otherwise technically can be difficult to achieve optimal fixation.'

'What other investigations? Hormone profile. The examiner showed me a lateral radiograph of the proximal femur and asked me to point out where I'd put my screw and why. The examiner expected me to know that screw placement is quite anterior on the neck (not like a DHS), and the direction would be towards the centre of the head, avoiding breaching the back of the neck (where the retinacular vessels can be endangered). In severe cases of the slip, this might be impossible; hence, the indication for open reduction (there is still some controversy about this).'

Septic arthritis of the hip

'Baby's pelvic x-ray. I jumped to DDH conclusion, but quickly backtracked and redeemed myself, but not before being asked to describe lines in pelvis and acetabular index, etc! Widening of medial joint space on one side – Told me baby was in ITU, so septic arthritis. Treatment? *Emergency*. Surgical approach in detail, splint afterwards – Anterior dislocation if capsule left open.'

Talipes equinovarus

'Do not say clubfoot as some examiners/parents will be offended.'

'Diagnosis? Describe all deformities. What orthopaedic disease associations are there? Ponseti treatment from start to finish with order of correction and why. Success rates. Counselling for parents. Pirani score details for monitoring progress.'

Genu valgum

'Three-year-old: Discussion of pathological/normal and management.'

Congenital limb abnormalities

'Do not call it a radial club hand or ulnar club hand or worst of all a lobster-claw.'

'Classification of upper limb congenital abnormalities (aplasias): Transverse/longitudinal, pre/postaxial/central, etc.'

Osteogenesis imperfecta

'Aetiology, classification, treatment of fractures and treatment of deformity.'

Cerebral palsy

'Classification: types, GMFCS.'

'Orthopaedic manifestatios and treatment including single-event multi-level surgery.'

What candidates were asked (hands)

Gamekeeper's thumb

'Acute: Management, approach, repair; chronic management – Free tendon grafts.'

Perilunate dislocation

'Management, classification. Didn't want Mayfield: Wanted CID, CIND, CIA, etc. Surgical approach for open reduction.'

Camptodactyly

'Picture: Principles of management.'

Wounds, infection and incisions

'A sketch of the hand with a flexor aspect wound: Principles of exploration, extension, tendon repair.'

'Photo of hand, swollen and lots of little ragged wounds (dog bite): Assessment, size of skin wounds belies underlying damage,

check tendons, collection. Needs theatre. Which organisms and antibiotics – How long? If large skin flap – How to manage in theatre? Principles of postoperative splintage and why. If was human, better or worse, which organisms?'

'Picture of swollen finger. I asked about Hx – May have been gardening 2 days ago, very vague. I expressed concerns of flexor sheath infection and proceeded to mention six cardinal signs of Kanavel (there are only four!). I explained surgical approach. Examiner kept asking 'are you sure?' which does nothing for your confidence.'

'Zone 2 flexor tendon injury. Asked about structure that could be damaged and clinical examination. This was a farmyard injury, talked about management of laceration (wound toilet, toxoid, tetanus immunoglobulins and antibiotics) and management of the flexor tendon repair. How would I extend the incision proximally and distally?'

Scaphoid fracture

'Delayed presentation, non-union of proximal pole scaphoid – Options, prognosis.'

'Was given a scaphoid bone and asked how to find out if this is left or right. Asked to point out the anatomical landmarks and the articulating surfaces.'

'Scaphoid non-union: x-ray of an advanced SNAC wrist with established radiocarpal arthritis. Asked about the pattern of progression of SNAC, treatment options in middle-aged labourer – Conservative (analgesic, steroid injection) and surgical (neurectomy, limited or complete fusion). PIN and AIN neurectomy; where would I find the PIN and AIN? One- or two-incision technique?'

Dupuytren's disease

'Draw spiral band/neurovascular bundle; mark incisions.'

Mallet injury

'Was asked about the surgical management.'

Intercondylar proximal phalanx fracture

'Was asked about surgical options including Suzuki frame.'

Wrist effusion

'Approach to management.'

Carpal tunnel syndrome

'Diagnosis, clinical exam, findings, showed EMG nerve conduction study results without any normal values and asked what is wrong in it, operative treatment, results, complications, percentages, etc.'

Scapholunate dissociation

'AP x-ray, then, when asked for lateral view showed me completely different patient x-ray with volar lunate dislocation. Discussion about carpal instability patterns, then how would you treat volar dislocation of lunate?'

Fingertip injury

'Picture of child's crushed finger. Describe the injury and management plan?'

Squamous cell carcinoma

'Picture of hand with missing index finger. History of old man with smelly hand for 2 years. Finger fell off last week. I initially went down the line of infection and osteomyelitis before realizing this was a malignancy. I mentioned epithelioid sarcoma (incorrectly jumping to the small print stuff!). I did not get that this was SCC.'

Thumb arthritis

'CMC joint OA: Describe, classification and treatment.'

'STT arthritis: Diagnosis from x-ray (very mild CMC OA as well). What patient groups and how presents? Treatment regimen from conservative to arthrodesis. Condition comes back with clinical/radiological CMC pain and solid fusion mass – What next?'

Ulna claw hand

'Spot diagnosis from clinical picture. High/low lesion and why? Tests to prove high/low lesion, anatomy of cubital tunnel and Guyon's canal. Where can get compressed at elbow? Release alone vs transposition discussion.'

Flexor tendon injury

'Picture of flexor tendon injury (intraoperative). Closed rupture of FDP: Mechanism, classification, treatment, and rehab. regimen. Differences if soft tissue alone or bony.'

'Which tendon will you suture? Current thinking is to suture both tendons. Core and circumferential sutures. How to retrieve FDP from palm (which I wasn't quite sure about!).'

Adult elective orthopaedics oral

General comments

'This was the only time in the entire exam I was asked about a classification system!'

'This was the only time in the orals where I was asked about evidence from papers – I did not know any for that specific topic and said sorry I cannot recall. The examiner smiled and moved on.'

'There were no surprises here. I was asked about tumours and arthroplasty and managed to steer the discussion round to areas where I was comfortable quoting evidence to support my answers.'

What candidates were asked

Rotator cuff arthropathy

'Options, including reverse arthroplasty and design rationale.'

L4 radiculopathy

'Signs expected: Motor, sensory, nerve tension, etc? Disc prolapse likely cause. Given MRI, showed far lateral L4/5 prolapse. Management options: Nerve root injection, decompression.'

Two-week-old infected tibial plate

'How to investigate, manage? Plastics involvement – Reconstructive ladder.'

Non-union femoral neck and shaft

'A bit complicated – Talked about valgus osteotomy proximal femur, possible strategies to manage shaft non-union at same time.'

Hallux valgus with degenerate joint and second MTP problems

'Talked about management of each. Indications for Keller's procedure.'

Severe varus ankle OA with subtalar OA

'Talked about principles, then said would do tibiotalocalcaneal fusion – Discussed approach and fixation with IM nail – Shown picture of just that.'

X-ray of varus knee with bowed tibia and Paget's

'Describe, what is Paget's, operative problems, bisphosphonates, deformity correction.'

Cavus feet

'Principles of assessment and management.'

Hip AVN

'Classification, causes, management.'

Malignant bone disease

'Metastases: Discussion of x-ray, Mirels' score, management.'

'Pathological hip fracture: First presentation of malignancy. Management and investigations. Fortunately I discovered it was a renal cell metastasis before discussing nailing! Then discussed staging, embolisation, etc.'

'Multiple myeloma: Presented as fracture through cystic lesion of greater trochanter. History – Minimal trauma in 50-year-old man. Investigations, staging, discussion with local tumour centre. Asked to describe sagittal MRI through pelvis showing widespread disease. Concerns regarding THR with pelvic disease? Options?'

'Describe this x-ray: Diagnosis (giant cell tumour) and differential? What are the features of benign/malignant lesions on x-rays? What history do you want to ask? What further investigations? Would you do the biopsy? Treatment of a GCT. Recurrence rates.'

'C5 metastasis from breast carcinoma. Assessment, how to differentiate complete from incomplete spinal cord injury. Which nerve root will be affected?'

Osteonecrosis of the shoulder

'Diagnosis, surgical management: TSR vs hemi.'

Fractured Charnley stem

'Well-fixed cup/distal stem: how to remove? Cement in cement revision of cup.'

Sclerotic lesion in proximal tibia of an 8-year-old

'Differential diagnosis: Ewing's sarcoma/osteosarcoma. Principles of performing biopsy, what to tell the parents. Simple path slides.'

Severe valgus osteoarthritis knee

'What must you do in planning?'

Recurrent hip dislocation

'Key factors in decision making, management and surgical planning.'

Rotator cuff tear and cuff arthropathy

'Why it happens, pathology and clinical course. This leads to cuff tear arthropathy x-ray – What will you do? Why? What are the results of various methods?'

'Cuff arthropathy: In an over 70-year-old. Describe x-ray. Had MRI: Describe (note absent cuff) glenoid looked a little eroded. Treatment: Reverse vs limited goals hemi depending on glenoid.'

Hallux valgus

'Why it happens, pathology of the deformity, why pronation, what would you do? X-rays: Angles, management plan.'

'Clinical photo and x-ray. Diagnosis? Take a history and examination findings that are relevant. What treatment would you perform (mild so conservative). Patient insisted on operative treatment – What would you do? (Said no and get second opinion – Accepted!) What operation would you do for mild/moderate HV and why? Describe a Scarf and Akin osteotomy.'

'Hallux valgus in an elderly patient: Assessment, treatment options. What surgery?'

Very osteophytic OA knee x-ray

'Pathology of OA, why osteophytes, reasons for loose bodies, what would you do? Take through TKR step by step and detailed questions about medial release.'

Thromboprophylaxis

'Began with a picture of the clotting cascade. Discussed various drugs and their actions on the cascade (struggled!). Mechanical prophylaxis. Awareness of recent NICE guidelines.'

Non-union of first MTP joint fusion

'Six months following attempted plate fusion. Discussed reasons for non-union (host and other factors). I mentioned need to exclude infection so this led to further discussion regarding investigations for infected implants (bloods, imaging, bone scan timing, etc). Further management – Re-fuse with bone graft (discussed different graft options) or excision arthroplasty (discussed pros and cons).'

Spondylolisthesis

'Shown the posterior half of a vertebra. What is it and why does it look like that? It took me a while to realize that it was a specimen removed during surgery for a pars defect! Discussion about types of spondylolisthesis. Asked if I knew the classification – Mentioned Wiltse – This was the only time in the entire exam I was asked about a classification system!'

Malunited (varus) femoral and tibial fracture with severe varus OA of knee

'Rule out infection as they were open injuries at the time. Approach to management. Which TKR? Describe technique (correcting alignment, etc).'

Post-traumatic pan talar arthritis

'Needs surgery – Which operation? Surgical approaches around ankle (in detail).'

Elbow arthritis

'Aetiology of OA and its treatment'

'Manifestations of RA at the elbow – Nodules, nerve compression, joint disclocation and degeration, etc.'

'Total elbow arthroplasty: types, pros and cons, complications.'

Synovial chondromatosis (SC) with multiple cartilaginous loose bodies

'Intraoperative picture. What is SC? Common location, clinical presentation, differential diagnosis for locking of knee, treatment.'

Shoulder dislocations

'Traumatic vs atraumatic. Difference between laxity and instability. What to do with multi-irectional instability? Surgical options.'

Severe OA of knees

'Bone loss and gross joint destruction – Offered differential diagnosis: Neuropathic joint, rapidly progressive OA, juvenile arthritis, AVN (diagnosis was haemophiliac knee). Risk factors in haemophiliacs (factor levels, bleeding and, of course, HIV and AIDS).'

Sessile osteochondroma of humerus

'Clinical examination, diaphyseal achalasia, incidence of malignant transformation, clues to malignant transformation (increase in size, pain, etc). Indications for excision.'

Hip dysplasia with OA

'X-ray of a 55-year-old woman with subluxed and superiorly migrated femoral head. Diagnosis, history, examination and treatment options. Crowe's classification of DDH. Details of preoperative and intraoperative considerations in DDH. Mentioned CT as a part of preoperative planning, narrow femoral canal, safe limit for lengthening, allograft and orange segment autograft.'

Back pain

'What's the most common diagnosis of back pain? Tell me about mechanical back pain. Describe all the red flags. What investigations should you do for red flag back pain? Sagittal MRI of L4 sequestrated disc. Describe this MRI. Diagnosis. Discuss presentation and management of cauda equina. If not cauda equina but acute root pain, how would you treat (wanted conservative *but* also discussion about the role of acute epidural/root blocks).'

Idiopathic hip AVN in 40-year-old

'Given history typical of AVN. Differential diagnosis of hip pain in the young adult. X-ray of very subtle AVN. How can you classify? (Ficat.) What would you do next? (MRI to confirm.) What treatment would you offer? (Core decompression.) What other options are there in early disease? What postoperative rehab. would you recommend? What's the prognosis?'

Failed THR x-ray

'Loose THR with dislocation on radiograph and acetabular discontinuity. Options for surgical treatment. Discussed the AAOS classification.'

'Metal on metal hip replacement with a mal-positioned cup – Investigation and treatment of painful MoM THR. (Remember just because it is a MoM THR does not mean that the problem the patient has is ARMD, this patient had a malpositioned cup causing psoas impingement.)'

'Management of infected THR: investigation, 1 vs 2 stage revision THR.'

Arthroscopic picture of medial meniscus tear

'What can you see? Diagnosis? Treatment in 25-year-old? Prognosis of partial meniscectomy? X-ray then given of medial only OA in a 45-year-old. Diagnosis? Treatment? Started talking about rationale and details of an HTO, then the bell went.'

Knee medial compartment OA in 35-year-old

'Options? I said HTO and went into indications, prerequisites, calculation of angle of correction (where I faltered). Same x-ray in 60-year-old. I said TKR. Asked about medial release, Whiteside's line.'

Paget's disease

'What is Paget's? Histology, non-orthopaedic complications and surgical problems of doing THR.'

Chronic osteomyelitis proximal humerus in a child

'Organism? Antibiotics? Surgical treatment? What surgical approach?'

TB spine

'Path specimen picture, management, immunocompromised patients.'

Sclerotic lesion in the metaphysis of proximal femur

'How do you manage? History, examination looking for common sites of origin (prompted – Decreased air entry on left lower lobe), investigation, local and distant staging. Bone scan was shown (proximal femur and vertebral mets). Treatment options were discussed.'

Tib. post-tendon dysfunction

'Aetiology, classification, management of each stage.'

Foot drop post TKR

'Risk factors for development – E.g. correction of severe valgus and FFD. Immediate management: release bandages and flexion of the knee. Long-term management: AFO, physio, NCS if no improvement after 1 month.'

Summary

'This was the most stressful time in my life but there is light at the end of the tunnel and once you finish you will feel that you have achieved something special. There are no shortcuts to passing but if you put in the time and effort, you will succeed.'

Acknowledgements

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Chapter 36

Examination failure

Andrew Port and Mike Reed

By the law of averages some candidates will inevitably fail.

Introduction

The one fact that links all trainees in an orthopaedic training programme is that we are all good at passing exams! It is self-selection of the highest order and none who present for this level of exam, 12 years or so after leaving school, would have achieved this level if this were not so.

The FRCS (Tr & Orth) is not a competitive examination in that a pass percentage rate is not set. It is a competency-based exam and so the pass rate will vary from examination to examination depending on the standard achieved. In 2014 the overall section 1 pass rate was 69% and the overall section 2 pass rate was 53%.

It is clear, however, that the pass rate varies depending upon the training status of the examinee. The exam has existed in its present form for several years now and useful statistics are emerging.

- Of the trainees that sat section 1 the first time in 2016, 95% passed. Re-sitting, 60% passed
- Of the non-trainees that sat section 1 the first time in 2014, 54% passed. Re-sitting 46% passed
- Of the trainees that sat section 2 the first time in 2016, 88% passed. Re-sitting, 61% passed
- Of non-trainees who sat section 2 the first time in 2016, 14% passed. Re-sitting, 25% passed

Clearly pass rates differ for those within and outside formal training programmes. The reason for this is multi-actorial, but will relate to a trainee having the advantage of a varied rotation with exposure to multiple facets of orthopaedic specialties and trainers, and the fact that their working life is very much focused on self-learning and critiquing whereas non-training posts tend to be service-based and insular.

Results do vary depending upon the actual training programme, particularly for the part 2 examination – With first time success ranging from 85% to 100% since 2009. The reasons for the variation between training programmes are also complex. Some training schemes have more focus on the exam than others, and many have their own internal barometer for performance and don't allow trainees to sit the formal exam until various internal criteria have been satisfied, e.g. sufficient scores in the UK in Training Examination (UKITE) and a local clinical exam. These regions have a very high pass rate. Other regions rely on the subjective (but often highly skilled) opinions of that trainee's recent trainers. Of course, some training schemes may actually be better than others.

At the time of writing it remains to be seen whether national selection will lead to a more even spread of results. Although the entry bar is now set in national recruitment, some programmes are still more competitive and, thus, may attract stronger trainees.

The detail of results of all training schemes are now published on JCIE website[a].

Regulations

Since January 2012 the JCIE have implemented new regulations, which restrict the number of attempts at the examination. The following apply and are strictly enforced:

Section 1	Candidates will have a 2-year period from their first attempt with a maximum of four attempts with no re-entry
Section 2	Candidates will have a maximum of four attempts and up to one further exceptional attempt (on the basis of providing positive educational evidence) with no re-entry

Although at first sight these rules seem harsh, remember that the exam remains an exit competency-based exam and it is unlikely that a candidate from a UK training programme will ultimately fail when entered at the appropriate time, unless there are significant issues with competency.

Remember the examiners have a duty to protect the public.

When to sit

Eligibility to sit the exam is dependent on the following main standard:

For trainees in the UK applicants must have a successful ARCP Outcome 1 at ST6 onwards, and for trainees in the Republic of Ireland the applicant must have a RITA C for Year 4 onwards.

[a] ww.jcie.org.uk

A similar statement of equivalence in training and standards is made for non-trainees by their principle referee.

The final decision for trainee's entry, however, lies with the Training Programme Director and sometimes the Specialist Training Committee (STC), and there will be variation in how the eligibility will be interpreted. There are no clear guidelines as to what level of knowledge is actually of the level and standard of a newly appointed consultant. The breadth of knowledge is clearly defined in the curriculum[b], however, and the examination has been designed to test this range in full.

The programme director or principle referee is also obliged to study the applicant's portfolio including logbook evidence to ensure parity with national comparisons at the end of ST6

The exam

In our 'candidates' accounts' section (Chapter 35) we offer many dialogues when the candidate knew they had performed badly and had blown it. This is to alert candidates to be on their guard for various pitfalls and hopefully avoid them.

That said, during the examination do not under any circumstances assume that you have failed and give up – You have failed only when you have that piece of paper in your hand confirming this to be the case.

There will always be a small number of candidates who perform so poorly in the clinical that they instinctively know that they have failed. Go to the orals and give them your best shot, if only for the experience, as it will be much easier next time knowing what to expect. There are too many stories of candidates who did not turn up for the orals after the clinicals because they were convinced they had failed only to find out later that they had in fact passed well.

A common mistake is to dwell too much on a perceived poor performance in one section of the examination, allowing this to affect the performance of the remaining examination.

> 'The trauma oral went very badly and I let it affect me in the other orals, particularly the paeds/hands oral as it followed straight on afterwards. In fact, I passed the trauma oral very well (and also the other two orals), but failed the paeds/hands oral – The trauma oral completely unnerved me.'

In part 2 of the examination between the 4 orals and the intermediate and short clinicals there are a combination of 96 marking opportunities. Efforts are made to maximize your exposure to as many different examiners as possible and you often don't come across the same examiner more than once and almost never more than twice. Each examiner marks according to the following scheme:

4 Absolute fail
5 Marginal fail
6 Marginal pass
7 Good pass
8 Very good, excellent performance

There are no veto marks or questions, implying failure that will result in an inability to pass, but one must achieve an overall pass mark. (On average 96 × 6.)

There is no examiner discussion or meeting to discuss borderline candidates, the decision based entirely on the pass or fail mark. So it is possible to pass or fail by one mark although this is unusual as candidates are usually uniformly good or consistently poor.

For the duration of the examination, the chairman of examiners instructs that each examiner is not to discuss an individual candidate's performance with another unrelated examiner; in other words, one should not be hearing that a particular candidate was poor. Genuine grievances during the course of the examination can be taken up with various secretarial staff present or the chairman of examiners.

Examiners are now scrutinized much more than in the past. All examiners now undergo training in examination technique. There is a method of internal validation and examiners will occasionally be monitored by a third party who takes no part in the exam. Their scoring is compared to the average along with whether the candidates they pass or fail were in general agreement with the other examiners' assessments. If an examiner is failing too many candidates, especially in an erratic manner, this will be picked up.

The results

One of the most annoying parts of the examination in the past was hanging around afterwards for the results. If candidates were unable to wait they could phone a special telephone number later in the evening for their results. This has all changed, and results are now sent to the college for verification before being sent out in the post to candidates with the necessary paper work to reapply for those that have failed.

The information below provided by the JCIE describes the timelines:

Section 1: Results will be available within four weeks of the date of the examination. Candidates receive an email and a formal letter from the Board Chairman which will include a breakdown of performance with confirmation that they have either achieved the required standard in section 1 to proceed to section 2, or that they have not

Section 2: Results will be available within two weeks of the date of the examination. Candidates receive an email and formal letter from the Board Chairman with confirmation of the result

Candidates who are unsuccessful receive formal confirmation including a final performance report from the Board Chairman. The candidates programme director or principal referee is also sent a copy. No other feedback is provided.

Take the trouble to inform your programme director or principle referee if you pass – They won't find out any other way.

There is an appeals process, but it should be noted that an appeal cannot be made on matters relating to academic

[b] www.iscp.ac.uk/surgical/syllabus.aspx

assessment and may only be made regarding the conduct of the examination. Appeals must be received by the within 20 working days of the last date of the examination in question.

The post-mortem

The official confidential white paper summary of performance takes a few weeks to arrive. This is sent directly to the candidate, a further copy is sent to the candidate's head of training and the chairman of examiners. This in theory should offer feedback to candidates on their overall performance and deficiencies – Essentially why you failed. Sometimes with the official report there are no surprises, but for some candidates it can be a shock to find out where exactly they came unstuck and usually this is not where they had originally predicted.

When you receive the analysis discuss this with your head of training. Analyse where you went wrong and, more importantly, show that you have thought about where you went wrong and offer suggestions on how you are going to rectify things and pass next time. You should be positive and explain why you think you failed and what you are going to do about it. Your head of training or principle referee will in all probability offer some advice, but take the initiative yourself. If you are a trainee your Certificate of Completion of Training (CCT) date may need to be changed if it falls before your opportunity to sit the exam again.

Your head of training may recommend that on your performance you should prepare for a longer period of time and miss the next diet of examinations. This is a very difficult thing to stomach, but, on balance, you should probably go along with this unless there are exceptional circumstances. Many programmes will offer a trainee support service and, in the authors' experience, this can be particularly helpful for those that fail more than once.

Use the re-sit pass rates in the introduction, and your marks, to determine when to re-sit.

One reason for failure is simply lack of preparation and knowledge. In-programme mock assessments should be used as a guide as you are compared to your peers, and preparation with your peers (either between yourselves or in small organized teaching/viva sessions) can be invaluable. Entering the exam with lack of knowledge is foolish, expensive and, with the new limited attempt rules, potentially disastrous.

Another reason for failure is exam technique. It can be several years since your last exam and the price of failure in this exit exam can be huge. For candidates who fail, careers can stall, substantive jobs are lost, family life can be severely affected, not to mention the impact on finances.

The section 1 exam is best addressed by studying, and practicing exam technique using the UKITE exams and other multiple choice questions available. Discussion groups with colleagues are also useful.

Treat the section 2 exam as a discussion between two colleagues. Try to demonstrate 'higher order thinking' – Essentially taking all the factors into account for that particular patient rather than reciting a textbook. This should be how you work in clinical practice anyway. Knowledge is tested but the ability to communicate and get across a professionalism and confidence is also important. Experts in interpersonal communication agree that:

- 7% relates to words
- 38% relates to voice tonality
- 55% depends on physiology or 'state'

The next time

You will need to re-focus and re-motivate yourself for the next attempt. There are several excellent exam preparation courses all over the country, and these should be regarded as essential. Several candidates at their second attempt claimed they had put in a minimal amount of work and it all went very smoothly. It is certainly an advantage the second time around knowing what to expect. However, in some respects this is a double-edged sword, as no doubt nervousness and exam fatigue will play a much more significant role. One small crumb of comfort when sitting the examination for the second time is that, as the material is already vaguely familiar, it is a lot easier to re-learn than to learn.

Another potential advantage is that, when you do pass it, you will have learnt your orthopaedics to a much better standard than the first time round and this will undoubtedly be of benefit in your long-term clinical practice.

Repeated failure

The third time sitting either the section 1 or section 2 examinations is much harder than either the first or second attempt. If this occurs, it is a time for holding your own counsel, not saying too much but quietly thinking about your next step. Something is not happening with the exam. Is it just bad luck, deficiencies in the training programme, your own deficiencies or a combination of everything together?

The situation will need to be discussed with your head of training or principal referee and an action plan sorted out, whether it be a period of intensive coaching, a deferred sitting or a temporary move to another training programme.

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