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The Physical Exam

An Innovative Approach in the Age of Imaging

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Preface

This book invites the clinician to take a fresh look at the "routine" physical examination. The approach taken here is intended for those who have already learned the fundamentals and are interested in adapting a more efficient approach to this time-honored discipline. While technological advances provide astonishing diagnostic information even in the most remote parts of the body, they have not diminished the value of a careful "hands-on" examination.

To modernize the physical examination, the clinician can adopt a systematic sequence and focus that may prove more productive than the methods taught in the pre-imaging era. At the same time, the regional approach developed here – rather than the serial (head-to-foot) approach – provides a keen opportunity to observe the astonishing interactions of anatomy and physiology. These interactions, furthermore, are sometimes the basis for symptoms that can be challenging diagnostically. For just such situations, an insightful physical examination can reveal relevant information that is beyond the scope of the digital image.

Presented here is a step-by-step sequence of the physical examination, mindful of the balance between being comprehensive and rapid while at the same time stressing accuracy. This approach emphasizes the signs that reveal how well an organ system is functioning and helps to sort out interrelated problems. Abnormal findings are organized descriptively and presented in regions where they are most likely to appear. With continued applications of these guidelines, the clinician will gain further confidence in the direct assessment of a patient. These skills, applied efficiently, are considered a critical supplement to imaging, not as an obsolete procedure required for medical records. Indeed, the severity of an organ failure, its interrelationship with other organs, and the identification of coexisting problems are within the special realm of the physical examination. It is hoped that this approach to the routine physical examination will help the clinician achieve this balance while, at the same time, adding to the satisfaction of practicing medicine.

Here, I am honored to acknowledge those who have reviewed portions of this manuscript and contributed to its development. These are Kristina H. Petersen, PhD (biochemistry); Daniel C. Doyle, DMD (mouth); John L. Phillips, MD, FACS (genitourinary system, abdomen); and Giang Nguyen, DPM (podiatry). Joanie G. Sheeran, PhD, meticulously reviewed the entire script for matters of language. Wendy P. Kahn, MEd, provided special guidance in writing some of the more challenging sections. There is no way to

thank individually the hundreds of residents, other healthcare professionals, and medical students with whom I have had the privilege of working over the years and who have shared in the concepts and details that serve as the substance of this book.

The majority of illustrations in this book were drawn by Paul E. Kmiotek. Figures of the shoulder and various hernias are those of Alex Bievenour. Sophie D. Phillips contributed illustrations of lesions that affect the face. In addition, the W. B. Saunders Company kindly released other images from my authored books in cardiology and vascular medicine. The staff experts at the computer center of Mahopac Library were graciously helpful throughout the preparation of this book. The Health Science Library at New York Medical College has been an invaluable resource since its beginning.

I must also express my appreciation to Michael F. Griffin, the developmental editor of Springer Medicine, for his keen attention to the details in this production. Richard Lansing, editorial director of Clinical Medicine at Springer, provided early encouragement that continued along the way.

Kent Lakes, NY, USA R.E. Phillips

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Part I

Patient Sitting, Facing Examiner

Introduction

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The physical examination continues to be a core skill of the clinician. At a time of overuse of and overreliance on technological advances and on the checklist culture of the electronic medical record, there remains an essential medical and medicolegal value in the efficient bedside patient examination. The sequence and highlights of the guidelines presented here emphasize a systematic approach that is thorough, rapid, and – to a reasonable extent – precise. They are meant for healthcare professionals who have accomplished the basic principles of the physical examination (Fig. 1.1).

Physicians in the later nineteenth and early twentieth centuries became highly skilled in diagnostic acumen on the physical examination, the only method to obtain objective pathophysiological information on their patient. The percussion of the chest and their description of cardiac murmurs became an art form; conclusions based on occult findings attained a kind of prestige among peers and learners. Yet, even that skill level could not reliably define diseases. The incorporation of laboratory blood studies, the x-ray, and the electrocardiogram so rapidly filled in the diagnostic uncertainty that they – to some extent – superseded the laying on of "educated" hands *and* ears. Now in the early twenty-first century, clinicians enjoy the additional diagnostic perspective of highly detailed imaging, serology, cytology, and laparoscopy. These advances have proven so colossal that we have come to depend upon them for decision-making, sometimes to a fault.

The CT scan, MRI, organ system function testing, and serological analyses – however spectacular and refined – cannot tell us all of the most critical information in a patient's evaluation. Fundamentally, they do not reveal how sick a patient is! Evaluating abnormalities in skin color and texture, pulsatile vascular dynamics, mobility, sensations, and language, to name a few, are still important. These observations are within the realm of the physical examination. Overlooking such signs in the initial encounter

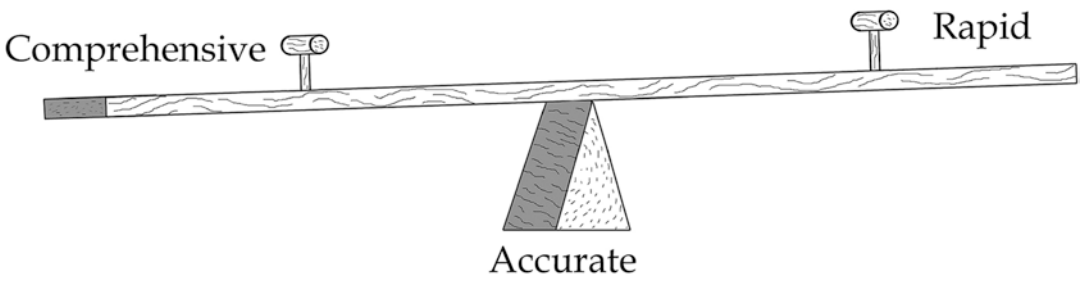

Fig. 1.1 Seesaw (Image courtesy Paul. E. Kmiotek)

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can prove a serious oversight when the examiner has become overdependent upon hi-tech diagnostic methods. Identifying anomalies on the physical examination, in fact, guides the clinician on what hi-tech tests to order or perhaps confirms that further exploration with expensive tests is not necessary.

Certainly, the immense value of modern technological achievements does not at all diminish the utility of the age-old discipline of the physical examination. For a more personal reason, being able to conduct a well-organized and speedy physical examination that is insightful and meaningful can provide a satisfying perspective to the everyday practice of medicine.

The place of the "routine" or physical examination is now in serious question [1]. Certainly there has been no proof of its value in the patient without symptoms (admittedly, a difficult value to prove). Indeed, an unexpected finding (e.g., a thyroid nodule) may only cause worry until an additional (and expensive) battery of tests has ruled it benign. It is accordingly more effective – cost and time directed – that the physical examination be determined by specific symptoms as they arise. Still, an argument is made that the periodic physical examination provides an opportunity for the clinician and the patient to review all the pertinent health issues, ongoing or preventative [2]. Certainly, our time-pressure mode of medical practice dictates that the "laying on of hands" must become more efficient. It is toward this ideal that this book is directed.

The approach taken here is anatomically regional, presented in a sequence that can be adapted easily to individual patients. Details in each region can be covered rapidly and explicitly with acceptable accuracy. On finding an abnormality in any region, the examiner then can then refer to an appended description in that category for details. Abnormalities are described in the region where they are most likely to occur; this regional approach does entail some repetition. The sequence of the examination presented here places the positions of the patient into those that are most advantageous for the clinician while minimizing the instructions to the patient.

The level of the text presupposes that the examiner has already acquired the basic skills of the physical examination. The organized sequence develops a rhythm and pace that can be adapted to virtually every encounter. It is with disciplined practice – just as an accomplished musician works on scales and fingering – that the efficiency and productivity of physical examination can be realized.

Meanwhile, the mindful patient, who is at a heightened state of awareness when being examined, is sizing up the examiner. The patient's impression of a smooth flow of the examination may, later on, have a substantial effect on the patient's confidence and cooperation in his or her own care.

Those entering the healthcare profession, having been exposed to massive amounts of preparatory information, often find themselves overwhelmed at the bedside for want of feeling comfortable about how to proceed. Having a clear idea of a logical sequence will help immensely. That said, the traditional "head-to-toe" method does not emphasize functional regions. Often neglected, for example, in the general physical examination, are the hands that can provide sensitive telltale signs of vital organ functions, systemic illnesses, and lifestyle. Since, according to this method, the feet are the last to be examined, these anatomical workhorses are routinely given less attention than, say, extraocular muscle function, yet the problems of the former in the general clinic population are far more common than the latter.

For purposes of these guidelines, cogent details of the more commonly found clinical conditions in each region are denoted. It is on recognizing an abnormality along the way that the examination must slow down to consider the significance of such a finding. These guidelines, then, provide a summary of various types of findings, be they spots, bumps, weakness, asymmetry, and a host of other categories. Some rare conditions are included, mostly those that have serious implications. Examples are certain endocrine disorders (particularly of the thyroid and adrenal glands) that evolve slowly and are often

ascribed to other conditions (such as aging) yet can have dire consequences when missed diagnostically.

Concerning the "annual" or "routine" physical examination not prompted by a symptom or special concern, an argument has been made that there is little point in using time and resources in what will almost certainly be a nonproductive effort. Emphasis instead should be on the patient history and the impact of social issues. Furthermore, there is concern about some findings that are not "medically significant" may lead to unwarranted worry and expensive follow-up testing. Others point out that the "routine" physical examination may disclose no meaningful information but at least the "laying on of hands" establishes a special bond with the patient in a setting conducive to discussing on-going medical issues. Addressing all is the position that the clinician incorporates experience and good judgment into the everyday gathering of information and decisions made on a patient's behalf.

To restate the position taken here, the clinician can gather a substantial amount of information on the health of the person examined, and this can be obtained in a very short time. With continued practice of an organized and concentrated approach, efficiency can only increase. It is when there is a significant finding (a sizable mole, possible clubbing of the finger tips, increased warmth of a joint, a minor tremor, etc.) that the clinician must slow down to evaluate the finding in more detail. In addition, it is only after extensive experience that the most effective clinician can detect subtle but meaningful differences that separate normal from abnormal (color, texture, power, etc.). On the personal side, the clinician can appreciate enough differences in every patient to make a well-organized and highly focused routine physical examination an interesting accomplishment.

The "Little" Things

We begin with a reminder of ideal conditions for the physical examination that were taught and emphasized on beginning medical training. While these conditions may seem only too self-evident, some are easily compromised in the rush from patient to patient. No apology is given for going over them once again.

Preparation for the physical examination should habitually include hand washing (or an alternative), maximum lighting, proper access, useful tools, and an appropriate sense of confidence. Also, the care given to preparing for the examination may provide the most obvious facets of the encounter by which the patient judges the clinician. These simple interventions convey an impression that the examination will be well thought out and performed in an earnest – not a perfunctory – manner. A brief description of each follows.

Hygiene

Patients expect doctors to examine them with freshly cleaned hands and well-trimmed nails. A vigorous scrub with soap and water is optimal.

An antiseptic rinse is effective only in copious amounts and then only if well-scrubbed into the whole hand. A quick dab not covering the entire hand may do little good. Furthermore, microorganisms resistant to the chemical cleanser are being recognized with ever-increasing frequency.

Gloves may be preferred; they are mandatory when "open" lesions are encountered. For purposes of the general physical examination, "non-sterile" gloves taken directly from the box are adequate. When touched on the "working" outer surface with the unwashed hand – a common observation – they merely provide an illusion of proper hygiene. The examiner must take care to touch only the cuff with a bare hand in the same way that he or she would don sterile gloves in the operating room.

Of course, every "external" item touched during the examination further compromises the microorganism barrier. It is recommended, when feasible, that the personal stethoscope or other diagnostic tools should be cleaned with an alcohol wipe in full view of the patient. Indeed, it is

well documented that the uncleaned stethoscope carries multiple drug-resistant *Staphylococcus aureus* (MRSA) from patient to patient [3].

Where a gown is required, the examiner is well-advised to wear it fully robed and tied. The patient's impression of a gown hurriedly put half-on may reflect the patient's impression of the quality of the encounter.

Lighting

The physical examination in inadequate light seriously compromises its reliability. Turning on all available lights, pulling aside curtains, and fully opening window shades seem too fundamental to need mentioning. Yet, examinations performed in the poorly lighted areas are all too commonplace on a busy clinical service.

Appropriate lighting is particularly important in regard to color of the skin and sclera. For example, incandescent light emphasizes icterus, whereas neon lighting lessens its intensity. The combination of these two sources, available in "plant grow lights," can provide the nearly "white light" that is optimal and cost-effective.

Access

Clinicians put themselves at disadvantage when bedside paraphernalia clutter easy access. In the hospital, chairs, mobile tables, and other impeding objects should be moved out of the way. Turning off the "audio" control of radio and television eliminates a powerful distraction and conveys the impression that the clinician intends to concentrate on the examination.

The typical hospital bed is generally kept low to allow the patient to step easily onto the floor; the position is not meant to facilitate the examination. The level should be raised to a height that allows the examiner to easily bend and reach the opposite sides of the body. In this elevated position, however, it is imperative that the clinician always stands directly in front of the patient to prevent his or her stepping down. It is just as important that the bed be lowered fully when the encounter has been completed.

Tools

The minimal tools that a general clinician should have on hand for virtually every examination include: a flashlight (the pocket-sized disposable ones are quite serviceable), blood pressure cuff, stethoscope, tongue blade, percussion hammer, vibratory fork, and measuring tape. A small "fanny pack," available at stores for outdoor activities, is invaluable for keeping these items close at hand.

The otoscopic and ophthalmic examinations are often too important to skip for wont of easily available equipment. At least, every general clinical service should have both instruments within reach. The circumstances may dictate that the patient examination be completed in a room where this equipment is available.

Attitude

Patients expect and in general appreciate a carefully conducted examination, especially during the initial encounter. Here the clinician's attitude is important.

When the examiner begins with an apology, adding that he or she is going to do a "quick physical if you don't mind," the words may diminish the quality and the authority of the encounter in the perception of the patient. Even in follow-up examinations of a shortened nature, it is preferred to state in an assertive way that a few areas (or systems) "should be examined again." After all, the patient is there to be examined, and the doctor is there to examine the patient. It is the succession of examiners checking an "interesting finding," as happens in the teaching hospital setting, that patients can understandably develop some objection. Of course,

any necessary aspect of the examination that may produce some degree of patient discomfort deserves both explanation beforehand and assurance of it being performed as gently as possible.

"Privacy"

The busy practitioner can easily overlook our commitment to preserving patient privacy of both information and body exposure. Be assured that the patient does not overlook it.

Clinicians must be fully cognizant of the need for a chaperone – documented by name in the chart – when examining patients of a different gender. It is good practice to have another observer present with any patient whose gender orientation is an issue.

Listening

Letting a patient describe a "chief complaint" for a minute or two (or longer) without interruption can be highly rewarding in the search for what really concerns the patient. Only then should the interviewer begin the incisive questioning. The period of listening provides the clinician with an opportunity to "reconstruct" the salient features of the history, rambling or fragmented as the patient's version may be, into a coherent and concise form.

It has been well established that physicians tend to interrupt the initial explanation by the patient within seconds, often jotting down each answer as the interview proceeds rather than waiting until a rather comprehensive concept of the presenting problem can be formulated [4]. Computer entry at the bedside seems to have exaggerated this disconcerting tendency.

In essence, the physical examination begins with the interview. The clinician has had an opportunity to notice capacity of the patient's attention, cognizance of details, memory, coherence, and speech. Questions about home, family, and occupation are generally sufficient to ascertain normal mental faculties. Only if there has emerged a suspicion of thought derangement will a need for formal mental status examination be indicated. It is on completion of the physical examination that "thank-you" would be appropriate and appreciated.

The ABCs

Before proceeding, three principles deserve consideration throughout the physical examination: symmetry, direction, and sequence.

Symmetry

First of all, people are generally symmetrical. As the examination progresses, it is critically important to compare each observation or function with its contralateral analogue. Keeping this in mind throughout the examination will be helpful in identifying abnormalities not producing symptoms. Sometimes asymmetry becomes a key factor in defining or at least leading to a diagnosis. Facial appearance, pupil and tendon reflexes, joint enlargement, strength, skeletal imbalances, and prostatic enlargement are but a few examples.

Evaluating symmetry is where accuracy is highly challenging. For optimal evaluation of symmetry in most regions, the physical orientation of patient to examiner is crucial. It is the reason for this presentation begins with the patient sitting and facing the clinician.

Direction

It is most helpful to develop a rigorous habit of examining symmetrical details in a consistent direction: right to left (or, if preferred, left to right). While not infallible, this simple practice is invaluable for performing an organized physical examination and – equally utilitarian – for remembering on which side a notable or even minor finding occurs.

Using a consistent direction does not mean, for example, examining the right eye and then the left but rather examining the right lids and then the left; observing the right palpebral fissure, then the left; checking the right pupil, then the left; and so on. What may seem cumbersome and slow is neither once sufficiently integrated into one's practice.

Sequence

These guidelines present a sequence for performing the "routine" physical examination. This region-based outline presupposes that the patient is not critically ill or disabled and can follow basic requests. It maximizes the examiner's access to the various regions while keeping time-consuming instructions to the patient for changing position to a minimum. Furthermore, this approach to the physical examination begins at the periphery rather than the center, emphasizing how organ systems are working, not what the sound like.

There are times when a practiced sequence as expounded here should be modified. For example, finding hot and sweating palms on tremulous hands at the initial phase of the encounter brings into sharp focus the possibility of hyperthyroidism. The findings may logically prompt the clinician to straightaway check for all the usual signs of hyperthyroidism: tachycardia, bounding arterial pulse, thyroid enlargement and bruit, and lid lag along with leading questions pertaining to history. Having satisfied a thorough search for corroborating clues while they are uppermost in mind, he or she could then return to continue from where the sequential format had been interrupted.

The Essentials

The "Essentials" text boxes within this book are meant to highlight those specific features that should cross the mind of the clinician when he or she moves through each region. As such, the discipline forms a mental checklist. These foci of attention are listed in box form at the beginning of each region. They will become second nature with practice.

When features on the checklist are found normal, one can quickly move on to subsequent regions outlined in the protocol. When any feature is abnormal and considered significant, a quick reference is provided that expand categories of abnormalities.

[Brackets around a subject indicate that it needs to be examined only if those subjects listed above it turn out to be abnormal.]

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First Impression

2

Ideally, the sitting patient faces the examiner directly on the edge of bed or examining table. (Of course, a patient may be too ill or injured to allow this position with tolerable comfort.) Also raising the hospital bed to eye-to-eye level provides an enormous advantage. With the bed elevated, the examiner must *always* stand directly in front of the patient to prevent his or her stepping down.

Essentials
The healthy face
Eye-to-eye contact
Sense of mood
Pitfalls

With this perspective, take 5 s to look carefully into the face of the patient in silence. This simple beginning – although not at first easy to do – enhances the encounter in a powerful and positive manner. Almost always, the patient will make eye contact. This point is important. It is a moment to assess the patient's alertness, ability to focus, and general amenability for being examined. Further, this advantage – even for these few seconds – establishes a connection between patient and examiner that is meaningful. It conveys the impression that a well-concentrated evaluation is about to begin.

Our everyday experience of "reading faces" is very different from the riveting objectivity of a medical evaluation. Indeed, some clinicians may find some discomfort in the "medical face-off," even for the several seconds suggested.

This initial full facial look tells the patient that the clinician means to perform more than a perfunctory examination. For the clinician, an objective observation offers more. Of course, it provides obvious indications of age, sex, ethnicity, and well-being (although always with a wide margin of uncertainty). Basic emotional states are said to be expressed by a facial "universal language". A smile, a frown, or a myriad of other facial expressions provides a level of nonverbal communication of feelings. We are familiar with the look of surprise, the grimace of pain, and the wincing on being touched in a tender place. The tense look of anxiety or fear needs no description. A vacant stare, the semblance of fatigue, or an unsmiling, melancholy expression tells us something. Added to the mix are personalities ranging from the immobile stoic to the fidgeting. The inability of the patient (excepting small children) to look you directly in the eyes for these seconds may reflect exceptional anxiety, a psychiatric disorder, or mind-altering drug effect.

Yet, the examiner must be careful to avoid overgeneralizing, embellishing stereotypic impressions, and misinterpreting expressions observed. Facial expressions – intentional or not – are far too complex to be of diagnostic quality. How complex are they? A reference book for writers, "Word Finder" published in 1951, cites 775 descriptive adjectives under "face" [1].

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What Is the Healthy Face?

The patient looks back at the examiner – eyes meet eyes – and there is a moment of attentiveness. Symmetry of the face is evident with general oval form, perhaps a bit rounder for women and squarer for men. The cheeks and temples are full. The nose is not pinched. There is no discoloration of lips. The distribution of hair is appropriate for age and gender, allowing for wide, normal variations. Palpebral fissures are equal and there is no sclera showing above the iris. Nasolabial folds and mouth are symmetrical. There are no prominent irregularities of the skin. Breathing is not noticeable; in effect, it is effortless. There is no tremor. Most patients will return a smile – and the smile is symmetrical. All this can be observed with momentary concentration.

There are seldom enough distinctive features in the face to identify any systemic disease. Rather, the experienced observer finding clues in the face of something other than normal initiates a search for collaborating observations on the physical examination or by other diagnostic pursuits. For example, increased frequency of blinking associated with cold, moist palms and an increased heart rate along with a robust peripheral pulse as well as excessive fidgeting suggests that the patient is nervous. If, instead, the palms are warm in the presence of the other findings, the clinician should consider a hypermetabolic state, most specifically thyrotoxicosis.

Telltale signs from smell can contribute to the first impression. These include the breath from tobacco smoke or alcohol consumption. More ominous are the smell of urea with advanced renal failure and the sweet smell of ketones where diabetes is uncontrolled. A fetid breath may come from a necrotizing inflammation in the oropharynx. The long-unwashed person will probably have other manifestations of body neglect.

What is the unhealthy face? There are many revealing signs. The "Hippocratic facies," described 2000 years ago, is that of a dying patient: "the nose sharp, the eyes sunken, the temples fallen in, the ears cold and drawn in and their lobes distorted, the skin of the face hard, stretched and dry, and the colour of the face pale or dusky … and if there is no improvement within [a certain time], it must be realized that this sign portends death" [2].

Can suspicion of the unhealthy face, less extreme than in Hippocrates' case, cross the mind in a few seconds? Yes – to a degree – by the well-practiced and focused clinician. It is when there is an abnormal finding, of course, that he or she must pause to expand the focus for appropriate evaluation. An approach to these details can be found in Chap. 11. This extended evaluation of the face is done later when the patient may be more comfortably settled into the encounter.

As with all first impressions, it is important to keep in mind that these are clues, not conclusions. Nevertheless, a condition suspected early in the encounter will help guide the emphasis as the examination proceeds. Energy should not be expended to prove to oneself that a first impression was correct – quite a normal tendency – but rather to search for related information that may support or lead to other possibilities.

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Hand

3

Now begins the "hands-on" part of the physical examination. Indeed, the hand is a good place to start. For the clinician, the hands provide a productive area where evidence for local and systemic disease is often apparent. It is where the physical examination can begin with mutual comfort.

We are accustomed to touching the hands of others – strangers or not – in everyday life. They also provide sensitive clues to vital organ failure such as compromised respiratory or circulatory functions. The stethoscope tells us how the heart and the lungs *sound*; the hands tell us what these organs *do*. A studied look at the hands can detect disorders of nerves, blood, muscles, and joints as well as to some metabolic diseases. Systemic diseases often have recognizable manifestations in the hands. Moreover, the hands can tell us something about the patient's activities and habits.

The practice of routinely examining a patient from "head to toe" may miss the hands altogether. After all, the arms are lateral "offshoots," not in the mainstream of the examiner's approach. The hands that can be a rich source of information concerning general health can thus be easily overlooked.

The examiner needs only to present his or her hands, palms up, and the patients will almost invariably offer theirs, usually palms up. This response tacitly invites the doctor to examine them. If an objective, initial mutual glance registers "contact" with the patient, the offering of the hands becomes the "contract" (Fig. 3.1).

Fig. 3.1 Hands examined

In general, check the palms and dorsa for color, temperature, moisture, and texture. Furthermore, the clinician looks for tremor, spots, bumps, muscular atrophy, joint enlargement, and nails configuration and care. When the hands are normal, however, the experienced clinician can perform a satisfactory assessment well within a minute.

Essentials
Color
Temperature
Moisture
Texture
Shape
Movement
[Capillary refilling time]

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Color

The color of the normal palm varies appreciably between individuals and at different times for the same individual, depending upon both the quantity of peripheral blood flow and the quality of blood. Vasomotor control is the determining factor, affected by the emotional state, physical exertion, medications, and body and ambient temperature. Comparing a patient's hand color to one's own is useful but not fully reliable owing to individual variations in vasomotor tone.

To appreciate fully these color variations, much experience is required; even so, separating normal from abnormal hand color is always challenging. Certainly, the importance of optimal lighting here cannot be overemphasized.

Erythema

Vasodilation

Exaggerated redness noticeable in the palms commonly reflects strong vascular dilation. Fever, prolonged heat exposure, and heavy exercise typically cause erythema. Indeed, blood flow through the hands can increase many fold under such stimuli. The hypermetabolic state – as in thyrotoxicosis – produces prominent vasodilation in excessively moist hands that serve as a major heat-dissipating surface. Atropine-like drugs that reduce natural sweating cause erythema from vasodilation; in this case, the hands are remarkably dry.

There are distributions of hand erythema that have more specific patterns. Palmar erythema, for example, is a frequent finding in chronic hepatic disease, the so-called "liver palm." Here, red blotches tend to occur on the ulnar surface of the palm. Such a prominent flush can also be seen in diseases of connective tissue, particularly rheumatoid arthritis.

Irritation

Persistent florid color of the hand may come from the irritation of frequent soap and water handwashing. Typical "dishpan hands" is a form of contact irritation from solvents or detergents. Dermatitis of the hands is a more intense redness, often accompanied by vesicles, excoriations, scaling, and, when chronic, thickening of the skin.

Allergy

It is no surprise that the hands are the most common area for allergic reactions of the skin to occur. Inciting agents are cosmetics, dyes, fragrances, latex, metals (most especially nickel in jewelry), and plants such as primrose and poison ivy. Clues to a contact dermatitis are involvement of the back of the hands and deep vesicular eruptions between fingers that resemble tapioca. Sometimes the outline of jewelry or a wristwatch leaves a telltale, red impression. Poison ivy is easily identified by tiny vesicles, often appearing in a nearly straight line and often between the fingers. On repeated allergic contact, the skin of the hands becomes reddened, indurated, and scaly.

Polycythemia

Erythema may also be caused by an excessive red blood cell mass, such as in polycythemia, either primary (polycythemia vera) or secondary to hypoxia. In the last mentioned, the color takes on a deeper, purplish hue, referred to as "rubor."

Toxicity

Poisoning from carbon monoxide causes an even more intense erythema, described as "cherry red." The degree of toxicity, however, is far advanced before the typical cherry-red color becomes evident. Severe headache and/or altered mental functions in the setting of an underventilated heating device suggest the diagnosis.

Interdigital Eruption

A pruritic eruption mostly confined to the webs of the fingers is suspect for scabies (*Acarus scabiei*). Tiny vesicles or pustules and hair-like burrows in the webs of the fingers are clues. The presence of scratch marks heightens the suspicion.

Pallor

Vasoconstriction

Adrenergic stimulation in the skin – from strong emotions, from exposure to cold or to substantial

reductions in blood pressure – causes vasoconstriction with resultant pallor. Pallor from vasoconstriction is most obvious in the palms and face. Exposure of the face to cold can produce intense vasoconstriction in the unexposed hands.

Anemia

Pallor in the absence of signs of vasoconstriction indicates anemia. Substantial anemia is usually present, however, before pallor becomes a reliable sign. Natural skin pigmentation and skin thickness are influential factors. Many clinicians prefer to look at the conjunctival sac, lips, or tongue rather than the palms or nail beds. No site has proven reliably superior for detecting pallor. Indeed, the degree of vasoconstriction in the different vascular bed is highly variable among individuals.

Arterial Flow

Palmar pallor that is unilateral indicates interference of arterial flow in the arm. Causal examples are compression of the subclavian artery by a cervical rib, an embolism from the heart, or an obstruction by atheroma (as in Buerger's disease).

"Raynaud's phenomenon" causes changing discoloration in which pallor (from intense vasospasm) occurs in the first stage of the reaction. There follows within minutes a change to cyanosis (from stagnated blood) and then hyperemia (reactive vasodilation), giving a bright red appearance. The changes are generally most dramatic in the fingertips, involving one or more sites. Features of "Raynaud's disease" are episodic or permanent segmental vasoconstrictive activity along with localized destructive changes of the skin, connective tissue, and bone.

Cyanosis

The nail beds are usually the most reliable place to detect cyanosis. Deoxygenation of blood causes cyanosis, either from (1) stagnation of blood flow or from (2) impaired oxygenation of blood.

Reduced Blood Flow

The nail beds best reveal cyanosis from a reduction of blood flow. Cyanosis is a bluish discoloration in varying intensities. It may be a benign condition, or it may be a sign of a serious circulatory problem, either peripheral or central in origin.

Acrocyanosis is a variant of normal vasomotor control in which constriction of arterioles in the terminal digits is accompanied by compensatory vasodilation of capillaries and venules, causing blood to flow sluggishly. The resultant peripheral discoloration is a dusky mottling. It is the result of extraction of oxygen from hemoglobin by tissues during this relatively stagnant flow.

The prefix "acro," of Greek origin, refers to the outermost or tip, and acrocyanosis is generally found in the nail beds. When intense, the discoloration may extend farther up the finger.

Usually, all fingers are involved uniformly in benign acrocyanosis and it is generally symmetrical. Peripheral arterial pulsations at the wrists and in the palms are normal. There are no signs of ischemic complications. "Physiological" cyanosis commonly occurs on exposure to cold, particular from immersion in water. A great variability in vasomotor responsiveness between individuals must be considered.

Acrocyanosis from pathological vasomotor control could be cardiac in origin or from obstructed blood vessels. Each has several diagnostic possibilities. Intense adrenergic stimulation may be the cause ranging from highly emotional states to Raynaud's phenomenon or pheochromocytoma. Sluggish circulation with acrocyanosis is also characteristic of circulating cold agglutinins, sometimes a feature of rheumatoid arthritis.

If acrocyanosis of the hands is not symmetrical, the clinician should check for signs of obstruction in the regional vascular flow. In proximal arterial obstruction or arteriovenous fistula, the hand will be cooler than the other hand. In an obstructed venous drainage, the cyanotic hand may be warmer.

Reduced Oxygenation

The nail beds and lips are the most revealing signs of central hypoxia. It indicates a serious

compromise of ventilation, cardiac function, arterial or venous obstruction, and arteriovenous shunts, either acquired or congenital. Central hypoxia also occurs in oxygen-poor ambient air such as at high altitudes or in small spaces where combustion engines or heating devices are in operation. Correlation between observations of cyanosis and arterial oxygen by oximetry is but a rough estimate of hypoxia owing to variations in skin pigmentation and thickness as well as to any accompanying vasoconstriction.

Jaundice

Hyperbilirubinemia

Bile pigment deposited in the skin causes jaundice (or icterus). One cause is insufficient excretion of bilirubin, the breakdown product of hemoglobin. Hepatitis, cirrhosis of the liver, and obstruction of the biliary tract are examples. Bilirubin gives the skin an orange-yellow appearance. Bilirubin is further metabolized to biliverdin that – in chronic jaundice – casts a greenish hue.

Jaundice also occurs from excessively rapid breakdown of hemoglobin, overwhelming the natural clearing of hemoglobin products. Hemolytic anemia and malaria are examples.

There is a propensity for bilirubin to attach to tissue that is highly elastic, such as the sclerae. Jaundice is less obvious in the hands. Dim light as well as fluorescent lighting will mask the presence of jaundice at low levels of bilirubin.

Carotinemia

Excessive levels of carotene tend to deposit in the more callous tissue of the palms, thus sparing to some extent the sclerae. Yellow palms and nonicteric sclerae, then, reveal a person who is eating large amounts of carrots, tomatoes, or other high carotene-containing vegetables.

Hyperpigmentation

Adrenal Insufficiency

Excessive pigment deposited in the skin is an important sign of insufficient adrenal cortical function. Hyperpigmentation is often most obvious in the creases of the palms. These creases are naturally heavily pigmented, however, in dark-skinned persons.

Hyperpigmentation from primary insufficiency of the adrenal cortex induces high levels of ACTH secretion (Addison's disease). At the same time, an increase in melanin-stimulating hormone from the pituitary gland occurs. It is this concurrent hypersecretion of MSH that results in the hyperpigmentation. Parenthetically, hyperpigmentation does not occur when adrenal cortical insufficiency is caused by failure of the pituitary gland.

Hemochromatosis

Hemochromatosis is a disease of iron overload known as "bronze diabetes." Iron deposits in all organs cause a wide range of symptoms. The skin, including the hands, takes on a grayish hue. The nail plate may have a central concavity (koilonychias), called the "spoon nail."

Discoloration

Discoloring of the skin may result from contact with many agents. Most common is cigarette smoking that typically produces a yellow-brownish smudge between fingers #2 and 3 of the dominant hand. There is a long list of revealing hand discolorations that relate to direct contact with industrial dyes and metals.

Moisture

The normal palm is slightly moist. Even when moisture is not visible, it is detected by the sense of traction as the examiner runs the back of his or her hand lightly across the palm. Lack of moisture, by contrast, feels more like running the hand against a desktop. Moisture in beads indicates excessive sweating.

Wet Palms

Hands that are excessively moist have visible and tactile sweat, observed and felt principally in the

palms. Sweating is a parasympathetic function. Sudomotor fibers, however, travel along with the sympathetic chains and thus undergo collateral stimulation from adrenergic agonists, whether natural or acquired.

High Temperature

Exposure to ambient heat as well as to endogenously stimulated heat production from heavy, sustained exercise causes beads of sweat to accumulate on the palms. Excessive endogenous heat is typical of hyperthyroidism. In these examples, wet palms are associated with increased core temperature.

Adrenergic Stimulation

Excessively sweaty palms are also caused by intense adrenergic stimulation. It can occur from great anxiety, from an insulin reaction, or from the rapid onset of an event that compromises circulation and results in hypotension. The latter is the result of failure of cardiac output or from vascular collapse from substantial blood loss or profound vasodilation of the splanchnic bed. It is important to remember that – in each of these pathological conditions – reactive vasoconstriction causes the palms to be cool.

Pheochromocytoma, although rare, is another cause of intense vasoconstriction; it is likely to produce beads of sweat in cold hands. Severe hypertension is typical; it may be intermittent and/or refractory to conventional antihypertensive treatment. In addition, patients with pheochromocytoma often experience symptoms, paradoxically typical of postural hypotension.

Hyperhidrosis

A benign but persistent and annoying condition causing excessively moist hands is hyperhidrosis**.** It tends to affect the soles as well as the palms. Here, vasomotor activity is not an issue and the palms are normal in temperature. Rarely, excessive sweating is an isolated disorder that can be caused by either central or peripheral neurological disease. Hyperhidrosis may be a neuropathic complication of diabetes; here, excessive sweating of the face and hands may occur along with excessive dryness of the feet.

Dry Palms

Excessively dry hands are all too commonplace in healthcare workers, food handlers, and others who must wash their hands frequently. It can be a serious problem in the obsessive person who finds it necessary to hand wash with compulsive frequency.

Dehydration

When no traction is felt by the examining hand passing lightly over the palm, the surface is excessively dry. Excessively dry palms occur, of course, in severe dehydration.

Sicca

Excessively dry hands from an underlying autoimmunologic cause is known as sicca disease (Sjögren's syndrome). Dry eyes and mucous membranes, however, tend to dominate the constellation of symptoms. Rheumatic diseases are fairly commonly associated with sicca manifestations.

Anticholinergic Drugs

Concerning drug toxicity, those with atropine-like action are most culpable. Such anticholinergic agents are commonly contained in remedies intended to relieve gastrointestinal symptoms; as OTC preparations, they can be used to excess. By impairing heat dissipation systemically, the dryness of skin is coupled with an increase in body core temperature. Erythema from vasodilation and excessive warm and dry extremities are characteristic results from overuse of these parasympathetic-acting agents.

Temperature

Palms are a preeminent surface by which the body dissipates heat. There is a strong correlation between blood flow in the hands and the adjustments made to maintain normal body temperature. Blood supply in the hands can increase many times from basal levels during exposure to a hot environment, to exercise, to fever, or to hypermetabolic states. The adjustment appreciated, normal people in normal circumstances

have a wide range of natural temperature in their hands. This fact is easily appreciated when touching the palm of another person and feeling whether it is cooler or warmer. That other person, of course, will have an opposite sensation. Assuming that two persons have similar physical characteristics and undergo the same laws of metabolism, the wide variation in hand temperatures must represent differences in the location of heat-dissipation mechanisms.

Hot Palms

Increased Heat

There are several causes of excessive heat in the hands, a finding providing evidence for important diagnoses. Regulation of body temperature in a hot environment and on strenuous and sustained exertion depends upon vasodilation in the hands as a major radiator of heat. In the same way, the hands respond to fever. Indeed, while a mother may prefer to check her child's forehead for fever, the palms are at least as reliable indicator.

Hypermetabolic conditions should be considered on finding excessively warm hands with no obvious explanation. In the absence of fever, heavy exercise and a hot environment and when coupled with fine tremor and sweating, hyperthyroidism is highly suspect. Polycythemia vera is also associated with excessive heat in the palms. Here some degree of rubor is likely associated.

Anticholinergic Drugs

Where excessive warmth of the hands with dry palms is not readily explained, impairment of cholinergic reflexes is suspect. An atropine-like drug may be responsible (mentioned above under the section on "Moisture"). A large number of OTC drugs contain such ingredients, particularly those marketed for various gastrointestinal symptoms. A critical finding in toxic cholinergic blockade is that the reddened, hot palms are bone dry, thus contrasting from that caused by physiological and adrenergic overdrive.

Cold Palms

Peripheral Causes

Palms that are cold reflect reduced circulation in the extremity. The cause, of course, may simply be from exposure to a cold environment. Of clinical significance are cold hands caused by strong adrenergic stimulation, the result of vasoconstriction. The problem may be merely that of a highly nervous person about to perform in public, to take an important test, or to be interviewed.

Palmar vasoconstriction could also be due to an insulin reaction in which a rapidly falling blood sugar stimulates a massive secretion of epinephrine from the adrenal gland. This adrenergic response "protects" against developing hypoglycemia by stimulating the conversion of liver glycogen to glucose. In effect, the cold palms, sweating, and tachycardia typical of the insulin reaction are *not* the results of hypoglycemia per se but rather are in defense against a rapidly falling blood glucose.

A similar but gentler reaction can be experienced in the "ice cream rush" with cold skin, sweating, and palpitations. Here, the sudden elevation of blood glucose triggers a powerful release of natural insulin. The ensuing rapid fall in glucose triggers a strong, reactive adrenergic discharge. The suddenly released epinephrine is responsible for the transient symptoms.

When a cold hand is unilateral, the clinician must consider obstruction of a radial or ulnar artery as well as injury to the ulnar nerve. These subjects are covered in the section on Chap. 4.

Central Causes

Cold palms may also reflect a reduction in the central driving force. A serious compromise in cardiac output should be suspect. The cause may be anything from the sudden onset of extreme tachycardia or bradycardia to acute myocardial insufficiency. Other possibilities are valvular obstruction from an intracardiac thrombus or valvular regurgitation after rupture of a papillary muscle or chordae tendineae. A pulmonary embolism may also be responsible.

"Benign" syncope from fright or sudden anxiety reactions probably represents both central

and peripheral contributions. Fainting occurs after a period of prolonged generalized vasoconstriction from adrenergic overstimulation resulting in exhaustion of norepinephrine stores when parasympathetic stimulation dominates. The result is sudden onset of extreme bradycardia. Hyperventilation and immobile standing (as in tilt table testing) are known to induce such a sequence of syncope-inducing reactions.

Texture

It does not take a diagnostician to recognize hands that have labored decades in farming or other hardscrabble pursuits from those of the sedentary person. Certainly, exposure to the elements and repeated physical adversities over a lifetime leave their imprint of rough-textured hands.

Taut Skin

Tightened skin of the palms and fingers is one feature of *system sclerosis* (scleroderma). This evaluation is facilitated by pinching the skin on the dorsum of the hand. Abnormal tight skin will be evident. Pitted scarification may occur on the palms. Taut skin limits full extension of the stiff fingers that tend to be held in flexion (acrosclerosis). The fingers take on a spindle shape (sclerodactyly). Similar findings occur in the more restrictive diagnostic entity of Raynaud's disease.

Loose Skin

Loosened skin of the dorsum of the hands occurs naturally with aging owing to the loss of elasticity. The examiner – through extensive practice of pinching the dorsum of the hand – can learn to appreciate the normal differences occurring through the decades of life. The child, teenager, adult, and the elderly all have distinctive textures. Skin texture is further described in Chap. 6, another site favorable to pinch testing.

Dehydration exaggerates "tenting" of the skin on the dorsal hand surface by a gentle pinch. Smokers tend to have premature loosening of the skin. An extreme example of stretchable skin is the Ehlers-Danlos syndrome where a defect in collagen formation, including elastic tissue, is an eminent feature. This syndrome is also marked by an increased range of motion in major joints.

Thick Skin

Thickening of the skin in the hands is found in myxedema. Pinching or twisting the skin on the back of the hand gives the feeling of kneading dough. It appears wrinkled and does not quickly spring back but remains somewhat "tented" for several seconds. This finding may be more easily detected, however, in the dorsum of the forearm.

Movement

Some abnormal movements are particularly noticeable in the hands. These neurological disorders are covered in this section on the hand. Should an abnormal movement of the hands at rest be detected, it may be most easily brought out when the arms are outstretched.

Tremor

Rest Tremor

A rest tremor is often most noticeable in the hands. The tremor is a rhythmic and involuntary action that does not impair sensations or coordination.

Rapid oscillations in the hands resting on the lap are typical of Parkinson's disease. Tremor of the thumb may be the earliest sign and it can be unilateral. When the whole hand is involved, it takes on a "pill-rolling" movement.

The tremor of Parkinson's disease is a dysfunction of neurotransmitters (dopamine and acetylcholine) that results in rigidity of movement. The cause is usually idiopathic but can be secondary to encephalopathy or drugs (see below). This type of tremor is denoted "non-intentional"; that is, it tends to go away or at least lessen when

the hand is performing a task. Fine motor skills such as handwriting, however, can be severely compromised.

Tremors similar to those typical of Parkinson's disease may be induced by some psychotropic drugs that block dopamine. These include lithium, tricyclic antidepressants, and sedatives. Neuro-stimulation from bronchodilators can also produce a rest tremor. Exposure to industrial solvents, notably heavy metals and carbon monoxide, is known to damage the substantia nigrans and so is implicated as a possible cause of a Parkinsonian tremor [1].

For optimal evaluation of tremor, draw the patient's arms fully forward to look at the fingertips for exaggerated tremor. With the palms facing up, check for "palmar drift." Dorsiflex the wrists while the arms are extended, checking for detecting asterixis (suggesting hepatic or renal insufficiency). If any degree of incoordination is detected, this is the opportunity to check the finger-to-nose test; such an abnormality indicates a disorder of the cerebellar system. These observations are described in later chapters.

A fine, rapid hand tremor is typical of hyperthyroidism. Should there be any suspicion of tremor, it will become more prominent with the arms outstretched forward. The patient's arms can be brought to full extension simply by the examiner drawing the tips of the patient's fingers toward himself or herself. The hand tremor with the extended arms can be most easily detected when viewed from the side. Keep in mind that normal people have a slight tremor when observed from this perspective.

Asterixis

A jerky tremor (or the "liver flap") can be elicited in patients with advanced hepatic failure and uremia. The patient is instructed to dorsiflex each hand maximally with the arms fully extended. Inability to hold the fixed position is soon observed as the hands drop slightly and then resume the original position. This action creates a slow, flapping motion with somewhat irregular oscillations. The frequency of oscillations in asterixis is generally two or three times each second.

Athetosis/Chorea

A continuously writhing movement, usually most obvious in the hands, is called athetosis, as is common in cerebral palsy. Choreiform movements, in contrast, are irregular muscular jerks that are involuntary and unpredictable. They may be evident in the hands and brought out by intermittent maintenance of gripping power, the "milkmaid grasp." There is also an heredity form of chorea in addition to causes from acquired encephalopathy and drug toxicity.

Spasm

Carpopedal spasm is characterized by adduction of the wrists and flexion of the fingers. Such involuntary contractions of distal muscles may be intermittent or persistent. Severe hypocalcemia and respiratory alkalosis are the most common etiologies.

Contractures

Proliferation of connective tissue in the palmar fascia shortens the cords that extend into the fingers (Dupuytren's contracture). The shortening causes permanent flexion of deformities of the fingers, typically starting in the fourth and fifth fingers. The palmar creases become distorted (Fig. 3.2).

Scarring of the central palmar fascia can be present in heroin users. The finding suggests that the insensitive palm was being used as an ashtray.

Contractures of the fingers are caused by injury or disease of lower motor neurons. These deformities are described later in this chapter.

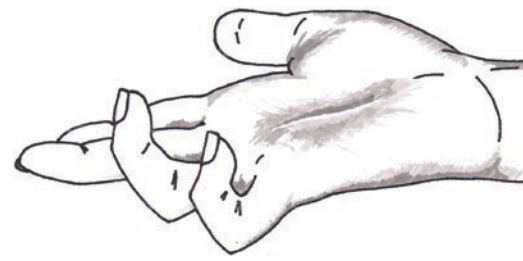

Fig. 3.2 Dupuytren's contracture

Spots

Various spots that appear on the hands deserve serious attention, however trifling they may appear. This section includes tiny, flat lesions that have a predilection for the palmar surface.

Rash

Palmar Rash

The palms are remarkably resistant to developing a rash from illnesses that characteristically cause rashes elsewhere. There are, however, important exceptions. A rash consisting of red blotches on the palms that does not itch is suggestive of secondary syphilis. The lesions may be flat (macular) or raised (papular), scaly, reddish brown, or copper colored. If raised lesions on the palms appear more like small pustules that are tender and pruritic, gonorrhea is the more likely cause.

Erythema multiforme typically appears as "target lesions." These are plaques with a central dark red, around which there is a pale zone of edema, and that is surrounded by a ring of erythema. The condition is notable for its symmetry. While they can occur anywhere, the hands are commonly involved. The target lesions or plaques are usually a reaction to a wide variety of drugs, most notably penicillin and sulfonamides.

Other vesicular or pustular lesions on the hands could be a feature of Reiter syndrome. Associated with this syndrome are conjunctivitis, mucocutaneous lesions, and reactive arthritis of major joints.

Petechiae

Petechiae are minute, dark red clumps of extravasated platelets that are deposited in the skin. They are not much bigger than a pinpoint. Petechiae can be macular or papular. The difference is of critical importance when it comes to cause and management. This finding is described in further detail in Chap. 6, where they are usually more prominent.

Purpura

Cutaneous hemorrhages in the dorsum of the hand are common with aging. These benign, spontaneous lesions occur when the structural integrity of both the skin and the terminal blood vessels is compromised. They are referred to as "senile purpura." Of course, they must be distinguished from the more serious problems of coagulation defects. This subject is expanded in Chap. 6.

Microemboli

Microemboli in the skin are red spots of various densities and shapes that arise from vegetations on cardiac valves. In endocarditis, erythematous or hemorrhagic spots on the palms that are neither painful nor tender are called "Janeway lesions." Similar palmar lesions could be emboli from disintegrating cholesterol plaques on proximal arteries; these contain fat globules or large clusters of platelets.

Raised pink to purple lesions in the hands that are painful and tender are microabscesses harboring bacteria or fungi. The central part of the nodule may be blanched or pustular. These are embolized fragments from infected vegetations on a heart valve that are known as "Osler's nodes." While such lesions can appear in the palm, they are more likely to be found on the fingertip pad or over a knuckle. They occur along with splinter hemorrhages in the nail (see below). Similar nodules may appear in some infections (gonorrhea, typhoid fever), in hemolytic anemia, or as a vascular complication of lupus erythematosus.

Splinter Hemorrhages

Splinter hemorrhages are slender, linear deposits that are red to brownish that are found beneath the fingernail. The vast majority are benign, the result of trivial trauma. More worrisome, however, is the possibility of splinter hemorrhages being microemboli from infected cardiac valves. They can also occur in systemic diseases such as rheumatoid arthritis, scleroderma, and lupus erythematosus (Fig. 3.3).

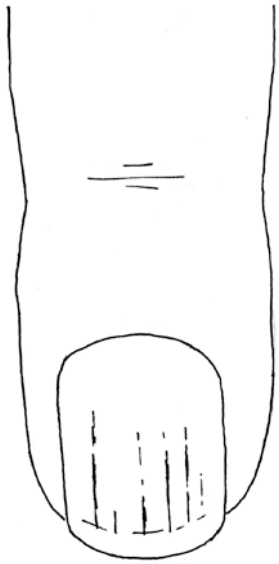

Fig. 3.3 Splinter hemorrhage

Telangiectasias

Telangiectasias are tufts of dilated capillaries that form a small, subcutaneous bright red spot. On the hands, the predilection is for the fingertips, but they can appear on other parts of the digits as well as on the palms. They are nontender, and they blanch on direct pressure, differentiating them from petechiae. Telangiectasias are commonly a sign of vasculitis and as such may complicate Raynaud's disease and lupus erythematosus.

Telangiectasias are also common in systemic sclerosis, particularly in the pads of the fingers. In this condition, they comprise one of the five components of the CREST syndrome (calcium deposits, Raynaud's disease, esophageal constriction, scleroderma, and telangiectasias).

When innumerable telangiectasias are also found on the lips and mucous membranes, hereditary hemorrhagic telangiectasia is suspect. Also known as Osler-Weber-Rendu syndrome, the lesions are distributed throughout the gastrointestinal tract and predisposed to bleeding.

Bumps

Callouses

Callouses represent overgrowths of the skin caused by habitual friction. The pattern of callouses reveals the job-specific occupation or hobby. They tend to disappear after several weeks without such stimulation. A look for callouses on hands is warranted in an insurance examination when the insured is receiving compensation for a work-related disability.

Nodules

Nodules on the hands have several possible explanations. Xanthomas are stony hard, cholesterol-containing nodules that form on tendons, especially on the extensor tendons of the fingers. A predisposing feature is marked hyperlipidemia (especially when triglycerides are predominant). That they are firmly attached to tendons differentiates them from nodules of rheumatoid origin.

In tophaceous gout, nodules on the hands have a tendency to form on joints or on the tips of fingers. The tophi, which are usually not tender, can usually be identified by their surface irregularity and their yellowish cast. Because the depositions are quite superficial, the urate crystals may give a white appearance to portions of the nodule. They can occur as a solitary lesion in one finger or as multiple bumps on several fingers with no particular pattern. Sometimes a gouty tophus is exquisitely tender when inflammatory changes of acute gout are superimposed around a tophus (Fig. 3.4).

Nodules in calcium pyrophosphate deposition disease (formerly pseudogout) mimic tophaceous gout in both appearance and distribution. The differentiation comes only from finding calcium pyrophosphate crystals in affected joint aspirate rather than urate crystals.

Nodules or scaly patches over the knuckles are common in various collagen vascular diseases (Gottron's papules). When together with erythema around the fingernail and telangiecta-

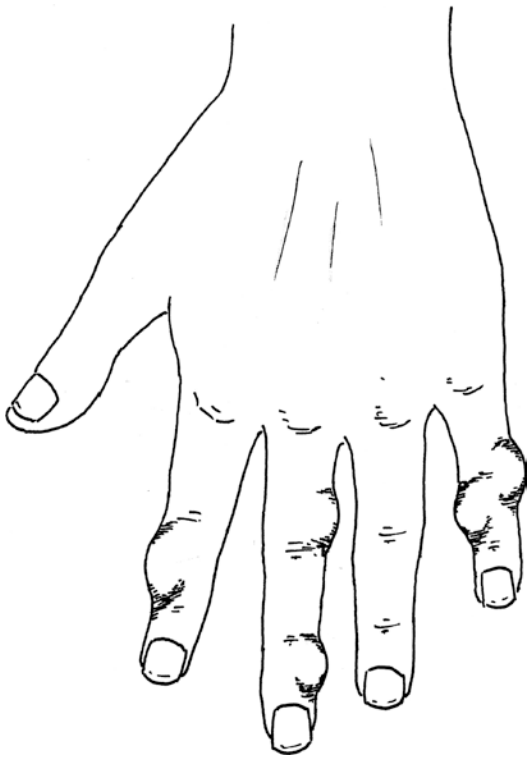

Fig. 3.4 Tophaceous gout

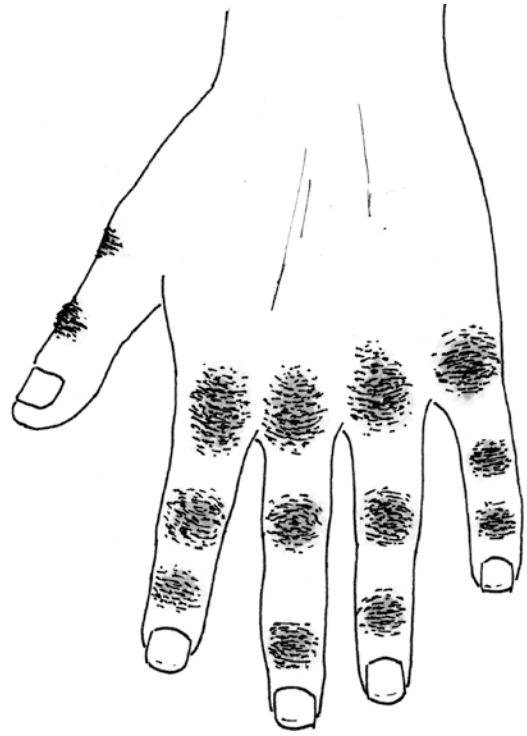

Fig. 3.5 Gottron's papule

sias, the underlying condition is usually dermatomyositis. Characteristic of dermatomyositis is a purplish discoloration over the back of the hand and joints of the fingers. These findings should prompt the clinician to examine the face for additional signs; these include periorbital edema and swelling of the eyelids (Fig. 3.5).

Nodular deposits of calcifications in the hands may be the only physical clues of hyperparathyroidism. This finding should also alert clinician to the possibility of Raynaud's disease, particularly the CREST syndrome (calcinosis, Raynaud's phenomenon, esophageal hypomotility, scleroderma, and telangiectasias).

Bullae

Blisters on the dorsa of the hands may represent *porphyria*, an abnormality in the synthesis of heme. The most common form is porphyria cutanea tarda that affects only the skin. The bullae which are beneath the epidermis are tense. When denuded, they form crusted erosions and may eventually induce scarring.

Palmoplantar pustulosis presents as crops of sterile bullae on an erythematous base. They are inflammatory lesions that are easily fissured. Smoking has a preeminent role in most cases, perhaps by the effect of nicotine that interferes with sweating.

Size

Enlarged Hands

Edema/Lymphedema

Hands that are puffy with pitting edema may be caused by venous obstruction or an infiltrated intravenous infusion. Lymphatic obstruction anywhere from the axilla distally produces a firmer swelling of the hand that is more resistant to pitting.

Deep vein thrombosis of the upper extremity should always be considered in unilateral and otherwise unexplained puffiness of the hand. While far less common than venous thrombosis in the legs, it occurs after trauma to the arm. It may be caused by any obstruction of venous return in the shoulder as well as extensive overhead work. One of a host of thrombogenic disorders may be underlying these etiologies.

Thickened

Thickening of fingers occurs in acromegaly. The skin becomes coarse, hardened, and thick. Excessively sweating palms are also typical. There are, of course, many other signs – particular in the head – that characterize this syndrome.

Finger Length

Elongated fingers (arachnodactyly or "spider digits") are characteristic of Marfan's syndrome. The abnormally long fingers can be demonstrated by making a loose fist with the thumb tucked inside. The revealing sign is the thumb protruding beyond the edge of the palm. Structural weakness of supporting tissue tends to occur in the arteries and the lens (Fig. 3.6).

Shortened fingers, especially the fourth and fifth metacarpal, are typical of Turner's syndrome, a condition most notable for amenorrhea and the "webbed" neck. In Raynaud's disease, acquired reduction in the size of the distal metaphalanges occurs by ischemic destruction of bone.

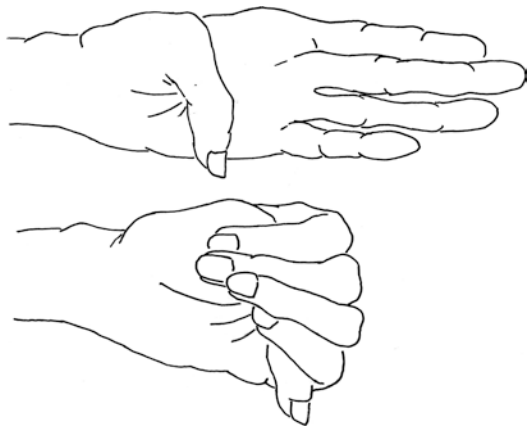

Fig. 3.6 Arachnodactyly

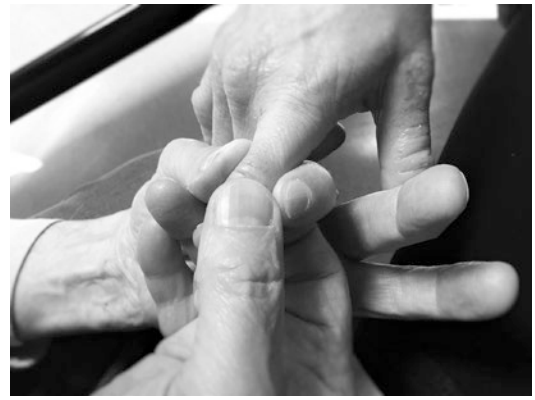

Fig. 3.7 Ballottement of finger joint

Joints

There is a great diversity of diseases of the joints. Characteristic features of each condition are often most distinctive in the hands. Enlargement of a joint in the fingers may represent bone growth, thickened synovia, and excessive fluid in the synovial space or any combination of these.

A distended synovia is identified by ballottement. To test for synovial fluid in an interphalangeal joint, apply firm compression on the lateral aspects with the fingers of one hand while compressing the joint – dorsum to volar aspects – with fingers of the other hand. The clinician must gain enough experience with ballottement of normal finger joints to know when there is excessive synovial fluid (Fig. 3.7).

Enlargement

Osteoarthritis

Characteristics of osteoarthrosis are small bony swellings on the interphalangeal joints and on the base of the thumb (Heberden's nodes). There is a predilection for the nodules to appear in the distal interphalangeal joints. During times of active inflammation in the area, they can be tender. Since the typical osteoarthritic hand is so often found in the elderly, it is colloquially known as "grandma's hand" (Fig. 3.8).

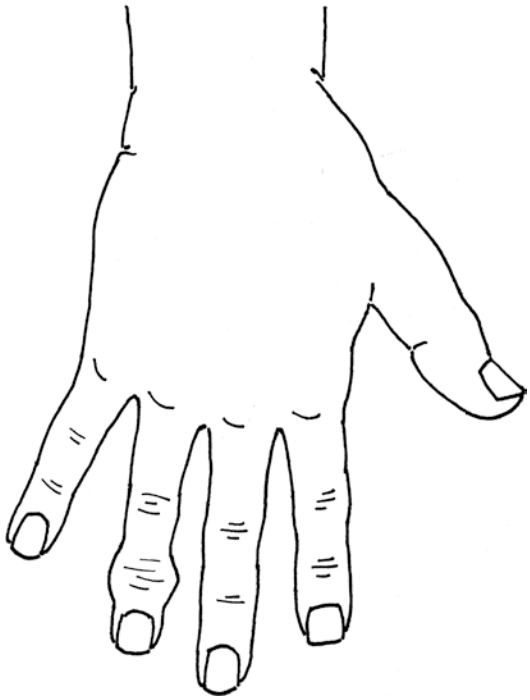

Fig. 3.8 Heberden's nodule

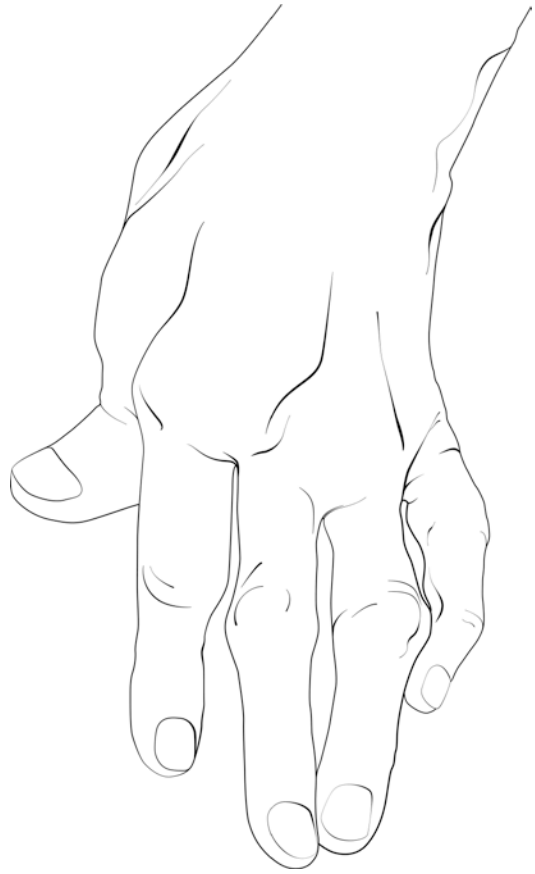

Fig. 3.9 Rheumatoid hand

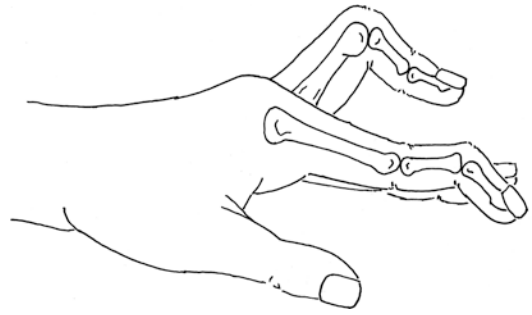

Fig. 3.10 Boutonniere + swan neck deformities

Rheumatoid Arthritis

The hand in rheumatoid arthritis has highly characteristic features including – importantly – symmetrical changes of affected joints. Typical is the deviation of the fingers toward the ulnar side owing to inflammation-induced shortening of the tendons. Thickening of the synovia has a predilection for the proximal interphalangeal joint in contradistinction from the distal interphalangeal joint in osteoarthritis. Affected joints are often tender with increased temperature; they are certainly painful even on slight movement. Coupled with excessive fluid in the joint, the fingers develop a spindle appearance. Spindle-shaped joints are also found in systemic sclerosis and Raynaud's disease (Fig. 3.9).

Some deformities of the fingers are quite distinctive in rheumatoid arthritis, and they go by picturesque names:

Boutonniere's Finger The joint between the proximal and middle phalanx in bent toward the palm, leaving the distal phalanx, is extended.

Swan Neck Finger Both the proximal and the distal phalanx are flexed while the middle phalanx remains straight (Fig. 3.10).

Fingertips

The pad of the finger is a sensitive indicator of the health of the microvascular system supplying it. That appearance in turn reflects the dynamics of the whole circulatory and respiratory system.

Vasomotor Tone

Episodic vasoconstriction in the fingertip is typically a Raynaud's phenomenon. It is often precipitated by direct contact with cold objects or by strong emotional provocation. The prolonged marked pallor of the digit is followed by cyanosis (from venostasis) and subsequently by erythema (over-reactive vasodilation). Thus, the reaction goes through a white-to-red-to-blue sequence, usually over several minutes. It may be a normal variant of vascular activity or it can result from earlier vascular injury such as frostbite.

Chronic ischemia of the fingertip is typical of *Raynaud's disease*. Here thinning of the pad occurs from compromised arterial flow. With thinning, the pad becomes smoother and, eventually, it loses the 'fingerprint' markings. Ischemic ulcers that are painful occur as the disease progresses (Fig. 3.11a, b).

Enlargement

"Clubbing" of the terminal digits consists of three features: (1) an increase in the soft tissue around the fingertips, giving them the shape resembling a drum stick and (2) a rounding of the nail that gives it an exaggerated convex curve when seen in profile. (3) At the nail bed where it extends into the cuticle, the nail is level rather than dipping slightly when observed from the side. When the nails of corresponding fingers are placed opposite each other, the normal diamond-shaped space formed at the cuticle virtually disappears. Usually, all fingertips are affected in clubbing (Fig. 3.12).

Actually a more subtle but earlier sign of clubbing is a spongy texture of the underlying tissue. It is evaluated with pressure exerted on the nail. A spongy nail bed, from underlying edema and microvascular changes, is common in amyloidosis but is also found in sarcoidosis and hepatic cirrhosis.

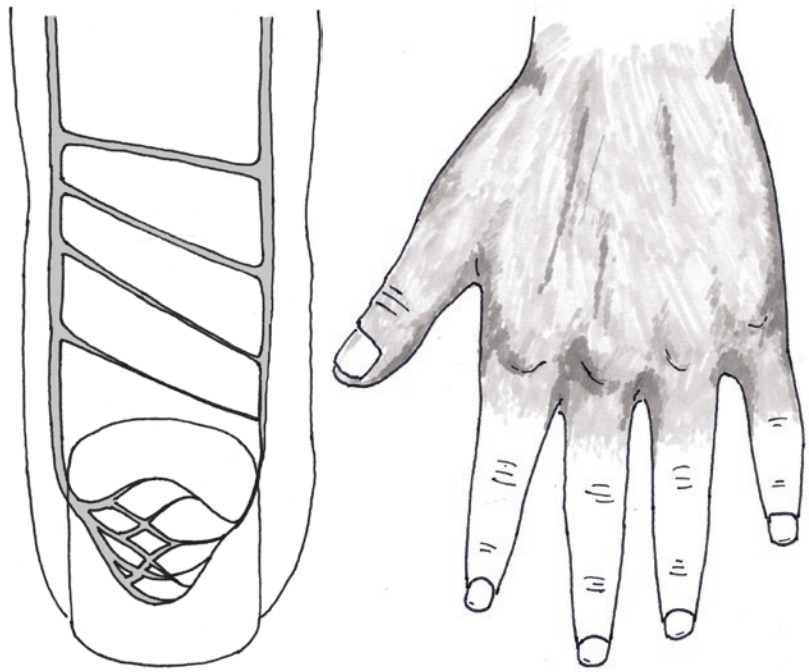

Fig. 3.11 Raynaud's disease: arteriospasm

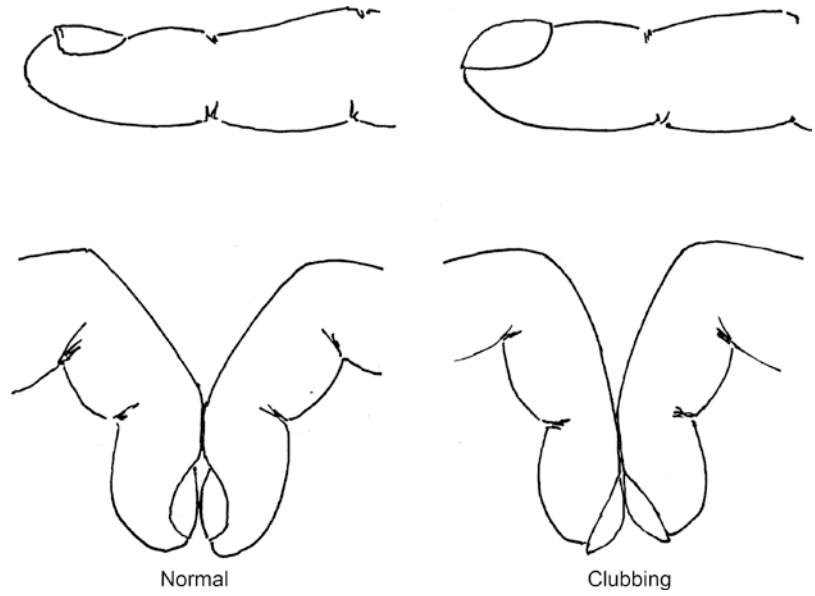

Fig. 3.12 Clubbing of fingers

Clubbing is most closely associated with prolonged hypoxia from chronic obstructive lung disease or from a congenital cardiopulmonary disorder. When clubbing is found along with cyanosis, congenital heart disease should be considered at any age. Here a "right-to-left" shunt is more likely than pulmonary disease to be causal. Other diseases associated with clubbing are cancer of the lung, inflammatory bowel disease, and hepatic cirrhosis.

Infection

Tinea manus is a fungal infection manifest by whitish, peeling, and macerated skin in the web between fingers. It is the hand equivalent of *tinea pedis* ("athlete's foot") which is far more common and almost always accompanies *tinea manus*.

Fingernails

The perceptive clinician can find an enormous number of telltale signs of local or systemic diseases on examination of the fingernails. While the full spectrum is left for the dermatologist, there are a few common and/or important entities that should be recognized.

Appearance

Grooming of the fingernails reflects to some measure personal hygiene and is a marker of one's general self-care (or caregiver's care). Clean and neatly trimmed nails most likely indicate a person who is at least somewhat fastidious, likely carrying over to other personal health matters. Highly decorated nails suggest a cosmetically oriented attitude that may have implications related to prioritizing general health habits. The obvious nail bitter is likely a worrier, an observation cautioning the clinician to be exceptionally careful on discussing all matters pertaining to his or her health.

Neglected nail trimming with soiled subungual spaces suggests a lack of general upkeep in heath maintenance. Exceptions to this conclusion are granted to farmers, auto mechanics, and other workers who must get their hands dirty.

The concave nail plate is generally linked to long-standing iron deficiency (koilonychias, in Greek meaning "hollow nail"). Connective tissue disorders such as rheumatoid arthritis and systemic lupus erythematosus may also cause loss of normal convexity of the nail plate and even lead to indented "spooning" of the nail (Fig. 3.13).

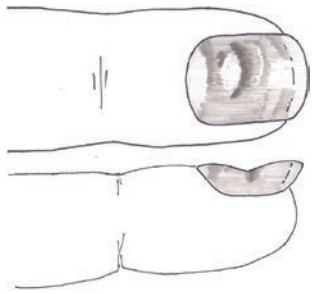

Fig. 3.13 Concave nail

Circulation

Capillary refilling time is a fast and easy way of accessing arterial circulation. On blanching the nail bed with slight pressure for a few seconds, the return to the original pink on release normally occurs within 3 s [2]. Delayed refilling indicates cardiac insufficiency or obstructive peripheral arterial disease. A somewhat longer refilling time does occur naturally in the elderly.

An arterial pulsation observed in the nail bed is caused by aortic regurgitation. Slight compression on the nail plate exaggerates the visibility of this phenomenon.

Growth

Major acute illness can affect nail growth. A few weeks after the illness, a furrow or ridge across the nail plate appears at the base (Beau's line). With nail growth, the side-to-side line migrates outward. It takes about 6 months for the line to reach the edge of the nail. The nail behind the ridge may have a rough surface (Fig. 3.14).

Measuring new growth according to "Beau's line" bestows on the clinician the perspicacity of Sherlock Holmes, revealing not only that a serious illness had occurred but when it began. On the other hand (no pun intended), one has to be careful because Beau's lines are also reported in psoriatic arthritis, human immunodeficiency virus (HIV-1) infection, crush injury, fractured arm, and bone marrow transplant. They can appear after exposure to very high altitude or to deep sea diving, presumably from acute hypoxia.

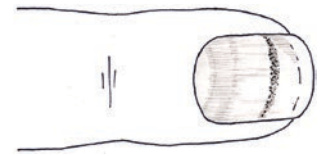

Fig. 3.14 Beau's lines

Malformation of the nail plate is generally due to fungal infection (onychomycosis). The culpable agent is a species of *Trichophyton*. Nails become thickened, brittle, discolored (white or yellow), and separated from the nail bed. Trauma, often years before, is the most common initiating event.

Texture

The normal nail bed is slightly compressible. It is greatly increased in severe edema. Amyloidosis, sarcoidosis, and hepatic cirrhosis also cause the nail bed to become spongy.

Infection

Paronychia

A paronychia is a red, swollen, and tender reaction around the base or sides of the nail. Paronychiae are most common among people employed in "wet" work (e.g., food services, fishermen, healthcare workers) and in children who habitually suck their thumbs. The infectious agent is a fungus, usually a species of Candida.

A single paronychia is likely a localized infection of little concern beyond local care. An abscess or extensive regional cellulitis, however, is a potential complication.

Multiple paronychiae, occurring in several fingernails, are a more serious matter. Underlying causes may be an immune-compromised state or an occult, systemic fungal infection (or both).

Abscess

A painful abscess localized to the tip or to the base of the fingernail is most likely an infection by *Staphylococcus aureus*, known as a "whitlow."

Herpes simplex, however, is another possible cause. Searching for an occult, underlying foreign body (glass shard or wood sliver) with an x-ray is entirely appropriate when an infection of a fingertip persists.

Fragility

Nails that are roughened, opaque, and brittle can reflect infection from ringworm. The condition is most common in hands that are immersed in water for prolonged times.

Microembolism

Splinter hemorrhages, mentioned earlier under SPOTS, are small emboli that lodge into the terminal arterial circulation, i.e., the nail bed. As a sign of septic emboli from a cardiac valve, the finding has implications of critical importance. It is noted, however, that splinter hemorrhages can also be caused by direct trauma. They can also be incidental findings associated with hepatic cirrhosis, psoriasis, and trichinosis.

Pitting

Pitting of the fingernail plate is a distinctive feature of psoriasis. Pitting is particularly common in psoriatic arthritis when the joints of the fingers as well as large joints may be affected. The nails can be thickened with a red-yellowish (salmon-colored) discoloration of the margins along with separation from the nail bed (onycholysis). Advanced cases are further complicated by crusted and pustular fissuring beneath the distal margin. Eczema of the palms may be present. Among these confounding signs are the typical pink and scaling patches of psoriasis found elsewhere, most commonly on the elbows and the knees.

Discoloration

White nails can be seen in persons with hepatic cirrhosis. Clubbing of the fingertips, palmar erythema, thickened and brittle nails, and a spongy nail bed make up some of the remote sequelae of this liver condition.

Brown nails are common in pregnancy and in malnutrition. Thyroid disease and habitual use of nail varnish are additional causes.

In heavily pigmented persons, a dark stripe coursing from nail base to free end is common and generally benign. Alternatively, a trauma-induced hematoma beneath the nail may appear as a band. A brown or black stripe that does not go away, which slowly enlarges, warns of a subungual melanoma.

A persistent nail deformity with discoloration, with or without ulceration, may represent squamous cell carcinoma. This possibility should be considered in an indolent fingertip lesion that does not fit the pattern of the previously described diagnoses.

Neuromuscular Function

Even a glance of the hands by the experienced clinician can reveal much about muscular and neurologic diseases. This approach embodies a principle of neurology by beginning to examine distal targets. If an isolated function is normal there, more proximal function is presumably normal. If an abnormality is detected, the clinician progresses more proximally until normal function is ascertained.

Neurological lesions that affect the upper limbs are readily detected in the hand. Motor or sensory defects may be central in origin or may be incurred from injury to a peripheral nerve. With the natural balance of flexor and extensor muscles perturbed, deformities of hand and fingers will occur.

Motor Function

Peripheral Motor Nerve Lesions

Here, the signs of injury in each of the three peripheral nerves are described individually. Because the peripheral nerves to the hands carry

sensory and motor impulses, both modalities must be considered in the evaluation.

Ulnar Nerve

Weakness from injury to the ulnar nerve reduces strength of flexion and leads to obvious wasting of the small muscles of the hand. It may be most obvious on the hypothenar area, the palmar surface between the fourth and fifth fingers. Paresthesias are present in these fingers but only on the outside half of the fourth finger.

Permanent injury of the ulnar nerve results in hyperextension of the proximal finger with flexion of the distal finger. The combination creates the "claw-hammer" deformity. Before vaccines were available, poliomyelitis was the preeminent cause.

The ulnar nerve injury originates usually in the elbow area and is caused by direct trauma, entrapment within the cubital tunnel, or from prolonged compression, including a "compartment syndrome." Fracture or an ill-fitted cast at the elbow can obstruct the brachial artery, causing ischemic damage to the ulnar nerve.

Strong and persistent pressure on the palm can injure the terminal ulnar nerve, causing motor impairment. Such injury may come from long-sustained grip on an oar or a hand-driven tool.

In addition, ischemic denervation of the muscles of the forearm causes permanent flexion of the wrists and extension of the fingers (Volkmann's contracture).

Medial Nerve

The most common cause of injury to the medial nerve is entrapment at the wrist as it passes through the fibrous sheath; the sheath also contains several flexor tendons. Termed the "carpal tunnel" syndrome, atrophic muscle complications are most noticeable by weakness in the flexors of the thumb. The defect is associated with atrophy of the thenar prominence (the muscle on the palmar surface at the base of the thumb).

More prominent than weakness in the carpal tunnel syndrome, however, are paresthesias, described under section "Sensory Function." Tests for entrapment of medial nerves within the carpal tunnel are covered in examination of the "wrist."

Radial Nerve

The radial nerve innervates the extensor muscles of the fingers and wrists. Injury to the radial nerve may occur proximally, in the axilla from crutches or at the elbow from compression against the humerus for a prolonged period. Abnormally deep sleep in a single position from alcohol or oversedation is the more common cause. The extensor muscles of the wrists and fingers are weakened, creating various forms of flexor deformities.

Express Evaluation of Hand Strength

For a quick evaluation, the strength of all three peripheral nerves to the hand can be performed using the thumb alone. Always apply opposing pressure in each maneuver. Of course, each test is compared with that on the contralateral side.

1. Flexion of the distal thumb (medial nerve)
2. Extension of the distal thumb (radial nerve)
3. Adduction of the base of the thumb (ulnar nerve)

Shoulder-Hand Syndrome

Reflex dystrophy with weakness, painful hands, and muscle atrophy is seen in the shoulder-hand syndrome. A common if imprecise term is the "frozen shoulder." All peripheral nerves to the hand may be affected from injury to the neck or shoulder. Associated abnormalities occur as well in autonomic and vasomotor control with changes in color, temperature, and moisture. Initially, there is increased warmth and moisture. Later on, as muscle atrophy progresses, cooling and dryness of the hand occurs.

Central Motor Lesions

In contradistinction to a lesion of the peripheral nervous system, a lesion in the central nervous system (including the spinal cord) does not gen-

erally cause atrophy of muscle. It is likely to be associated with some degree of spasticity. Some muscle bulk will be lost owing to disuse.

Motor Cortex

Weakness in the hand from a central motor lesion involves the cerebral cortex or any portion of the pyramidal tract. A localized lesion in the motor cortex may involve the hand alone or extend to those areas in close proximity: the wrist and neck.

Corticospinal Tract

Because the pyramidal tract is a network of fibers descending from the entire motor cortex, a lesion anywhere along is course and extending to the spinal cord is likely to cause weakness and spasticity in more than just the hand. Such a central motor lesion is described in Chap. 9.

Weakness from such central lesions is associated with preserved muscular bulk in contradistinction to atrophy from peripheral nerve lesions.

One test of upper motor neuron disease to perform on the hand is reflex flexion of the thumb. This occurs on snapping the edge of a nail on another finger on the same hand (Hoffmann's sign). This sign of a pyramidal tract lesion is equivalent to the plantar withdrawal (Babinski) reflex on stroking the sole (Fig. 3.15).

Fig. 3.15 Upper motor neuron disease test (Hoffman's sign)

When signs of pyramidal tract injury are associated with sensory losses within the same distribution, the clinician should think of a cortical lesion that subtends both the motor cortex and the sensory cortex, that is, crossing the sagittal sulcus. A mass lesion rather than an interruption of a vascular territory is suspect.

Sensory Function

As in the motor examination, moving from distal sites to proximal is recommended for testing sensory functions. Admittedly, testing of the sensory system – depending on a patient's subjective response – is considered the least reliable form of neurological assessment. A key to successful evaluation is insuring that the patient is clear about the perception that he or she is about to feel.

Peripheral Nerves

Increased Sensitivity

Hyperesthesia comprises a spectrum of ill-defined conditions in which the skin is excessively sensitive. Itching from dermatologic disease, liver failure, and psychological etiology is often associated with hyperesthesia. To add to the complexity of interpretation, light touch can be perceived as pain in the hyper-esthetic state.

Decreased Sensitivity

Reduced sensitivity from dysfunction of peripheral nerves is often preceded by paresthesias. These are usually referred to as a "pins-and-needles" sensation.

Injury to a nerve in the peripheral nervous system causes atrophy of muscle as well as loss of sensation. Therefore, when paresthesias or reduced sensations are associated with muscle atrophy in the same territory, peripheral nerve disease should be considered. The causes of such injury take in numerous of possibilities.

The specific nerve damaged can be readily identified with a relatively high degree of accuracy. Territories of the individual sensory nerves are

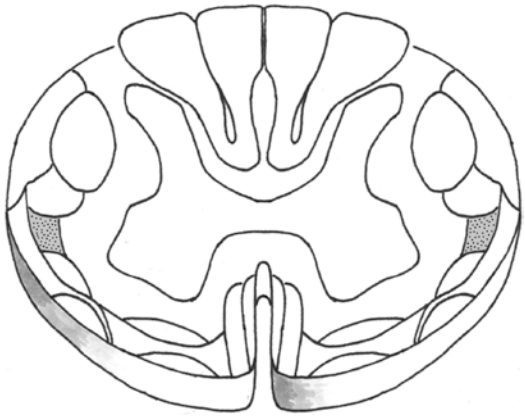

Fig. 3.16 Sensory tract: pain and temperature

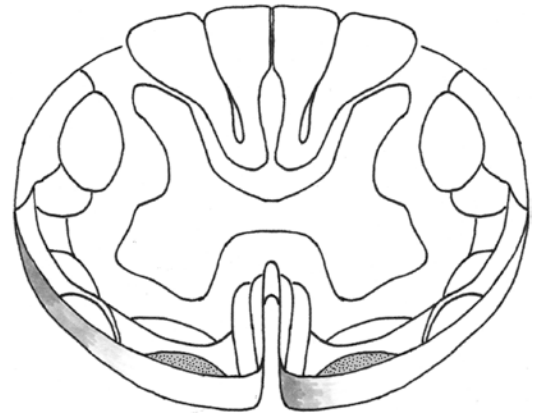

Fig. 3.17 Sensory tract: light ouch

described below. For most cases, function of three major sensory tracts can be rapidly determined, placing the examining tool on the fingertip. The patient is asked to look away during this testing.

Pain and Temperature

An ascending pathway in the spinal cord carries sensations for both pain and temperature. This is the lateral spinothalamic tract. It is activated by sharp stimuli. A good tool for assessing pain is the sharp end of a wooden tongue blade after having been twisted apart. It can be applied gently to the fingertip without danger of breaking the skin or causing pain. Indeed, reporting the sensation as "sharp" rather than "pain" is preferred for medical records (Fig. 3.16).

Light Touch

Sensations for light touch travel in the ventral spinothalamic tract. Testing can be done by sweeping a facial tissue across a fingertip (Fig. 3.17).

Proprioception

Impulses for proprioception and vibrations are carried in the posterior columns of the spinal cord. An activated tuning fork held firmly against the bony structure of the fingertip serves to test these functions. For this purpose, a fork with a frequency of 128 cps is generally optimal. Vibrations created by a thump of moderate intensity on the fork are sufficient (Fig. 3.18).

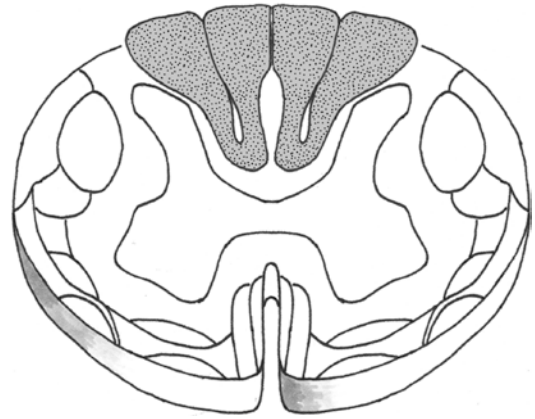

Fig. 3.18 Sensory tract: proprioception

The individual sensory nerves accumulate on the lateral aspect as the proprioceptive tract of the posterior column proceeds toward the head. Hence, sensations from the hand and arm are carried in the lateral column (the fasciculus cuneate) and the leg in the medial column (the fasciculus gracile).

Sensory Territory

Ulnar Nerve

Ulnar nerve pathology causes sensory loss over the ulnar side of the palm, on the fifth finger, and over the ulnar surface of the fourth finger (Fig. 3.19).

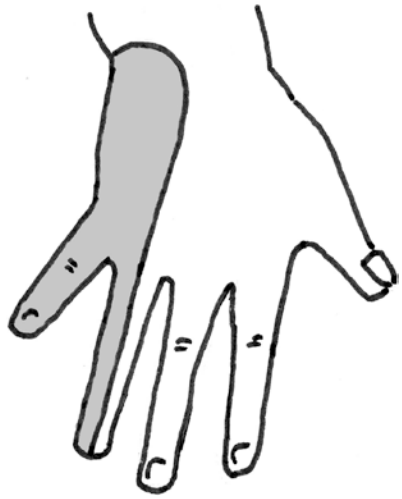

Fig. 3.19 Sensation: ulnar nerve

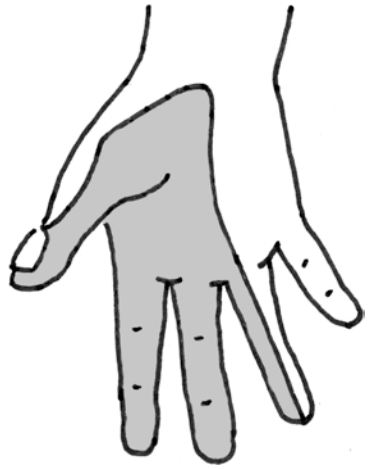

Fig. 3.20 Sensation: medial nerve

Medial Nerve

Paresthesia from injury to the medial nerve involves the remaining surface of the palm, the radial surface of the fourth finger, and the remaining fingers, including the thumb (Fig. 3.20).

When the medial nerve is entrapped within the tendon sheath as it passes through the ventral part of the wrist, paresthesias in this distribution occur. Denoted the "carpal tunnel syndrome," two tests are meant to reproduce the sensation of tingling or other paresthesias. These tests, however, are not wholly reliable for diagnostic purposes:

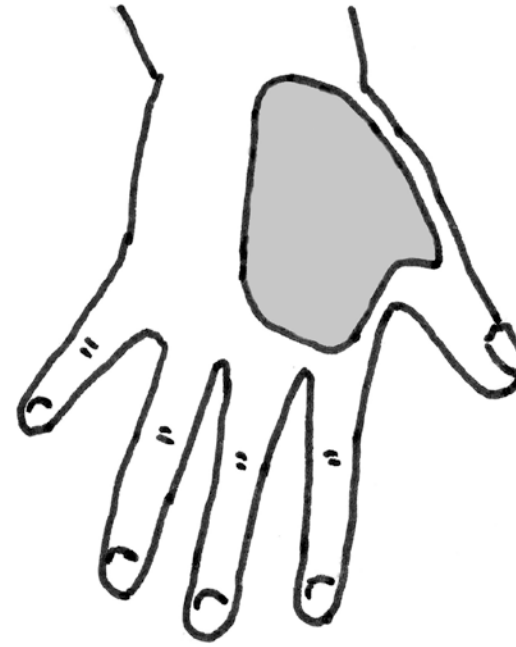

Fig. 3.21 Sensation: radial nerve

1. Abrupt flexion of the wrists (Phalen test)
2. Tapping the mid-wrist on the volar surface with a reflex hammer (Tinel sign)

Radial Nerve

Sensory defects from damage to the radial nerve occur over the dorsum of the hand on the radial side. Remember that the fingers on that side receive sensory innervation from the medial and ulnar nerves (Fig. 3.21).

Central Nervous System

The etiology of sensory deficits in the central nervous system may be deduced by characteristic features. Lesions in the sensory cortex, often predominant in the hands, tend to impair the ability to appreciate the shape, texture, and position of familiar objects. Lesions in the thalamus can impair any form of sensation on the contralateral side of the body. While the character of the dysesthesia is difficult for patients to describe, it may have a burning or stabbing quality. Vascular, neoplastic, or degenerative causes may be responsible.

Sensory deficits in the long tracts from the brain or the spinal cord may or may not have concomitant motor defects, and they are not associated with atrophy of muscles. Disruption of sensations from a central cause produces symptoms on the opposite side of the body.

Coordination

Coordination demands highly integrated functions of several systems. For the hand – where incoordination is readily observed – two of these systems (motor and sensory) have already been evaluated.

Intact cerebellar function is confirmed by a smoothly performed finger-to-nose test. Typical of cerebellar dysfunction is an ever-increasing swaying of the hand as it approaches the target.

If the finger-to-nose test is indeterminate, alternating tapping of each hand can quickly reveal compromised coordination. If these movements are slow and clumsy, further evaluations for rhythm, posture, and sense of the body in space are indicated. These include testing for balance, nystagmus, and gait.

Drug toxicity causes cerebellar ataxia, usually with some degree of confusion. Alcohol, of course, is the primary offender but any sedative or hypnotic agent may be responsible. Bromide in a widely used remedy for gastric distress can lead to severe incoordination when these antiacids are taken in high doses. Transient cerebellar ataxia may also be a complication of an infection, especially viral diseases. Demyelinating diseases causing cerebellar dysfunction include multiple sclerosis.

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Wrist

4

Essentials
Range of Motion
Weakness
Stiffness
Bumps
Arterial pulses

Although the wrist and hand function as an integrated unit, they are considered here in separate sections for convenience in description.

Range of Movement

The wrist is flexed and extended (dorsiflexed) to the fullest comfortable range. The movement may be either active (performed by the patient) or passive (performed by the examiner). Weakness or stiffness is readily evident.

Weakness

Strength here as elsewhere is tested by individualizing power against resistance, first on one side and then on the contralateral side so that a point-by-point comparison can be made. Weakness that is confined to the wrist and hand – that is, not involving the arm – can be assumed to be a peripheral neuropathy.

Flexion Weakness

The presence of wrist weakness for flexion implicates injury of the medial nerve. The most common cause is entrapment of the nerve within the carpal tunnel. Symptoms associated with this syndrome have been described in the Chap. 3.

Extension Weakness

Compromised extension of the wrist is the inability to fully dorsiflex the hand. With the arms outstretched palms down, the hand hangs flaccid. Wrist drop signals injury of the radial nerve, the largest branch of the brachial plexus that originates at the C5–8 spinal cord level.

Causes of wrist drop are numerous, compression injury being most common. It can occur at the axilla from ill-fitted crutches or from sleeping with the arm draped over the back of a chair ("Saturday night palsy"). An injury to the radial nerve near the elbow may be from a fracture of the humerus or a penetrating wound. Compression injury at the wrist from overtight bracelets or handcuffs is more likely to produce "finger drop." Most notorious of neuropathic causes is lead poisoning although alcohol abuse, vitamin B_{12} deficiency, and diabetes are possible etiologies. Infections (such as diphtheria, typhoid fever, leprosy) can also produce wrist drop.

Paresthesias frequently accompany wrist drop. These usually occur in the back of the hand between the thumb and index finger, the distribution of the radial nerve.

R.E. Phillips, *The Physical Exam*, https://doi.org/10.1007/978-3-319-63847-8_4

Stiffness

Resistance of the wrist in flexion and/or extension (with or without pain) has many possible causes. In a nutshell, consider the following:

- Prolonged immobility, as from a full arm cast over several weeks.
- An old injury to one or more of the eight small bones in the wrist. Most susceptible is a fracture of the navicular bone (the one just proximal to the thumb). This "ice fall" injury may cause an aseptic necrosis of the bone that is not evident in an x-ray taken in the days immediately after the injury.
- Arthritis from either rheumatoid disease or osteoarthritis. Both are likely to be more pronounced in the hands.
- Urate or calcium deposition within the synovial space of the wrist can impede movement. The possibilities should be considered in patients with either gout or calcium pyrophosphate deposition disease (formerly called "pseudogout").

Repeated heavy working of the wrist may eventually result in the "intersection syndrome" [1]. It is so-called an overused tenosynovitis, because long muscle tendons that extend the thumb cross over those that extend the adjacent fingers. Occupational tasks and sports – weight lifting, canoeing, raking – that require a strong, repetitive griping are usually responsible.

Bumps

In the wrist, a ganglion cyst occurs either in the front or back of the wrist. Typically, a ganglion cyst is firm, non-tender, somewhat mobile, and, because it contains a jellylike fluid, somewhat ballottable. It may be large enough to impair movement. Evidence of its cystic nature is gained by transillumination. It becomes firmer from fibrotic changes over the years.

The origin of a ganglion cyst is probably a bulge of tough tendon sheaths or synovial membrane that surrounds the small bones of the wrist. They protrude outward, perhaps after a sharp and powerful impact. Of historical interest, a home remedy for a ganglion cyst was to strike it with a heavy blunt object, the family bible being the most convenient object in most households to fit this description.

Arterial Pulses

Feel both radial pulses simultaneously, comparing intensity of the pulsatile force. This evaluation is one of the few times that both hands are used together for palpation. The ulnar artery serves as an alternate should the radial artery be difficult to palpate.

Both a rapid rise and a fall in the pulsatile dynamics suggest adequate peripheral circulation and elasticity of arteries. If both radial pulses are full and brisk, there is probably no reason to measure blood pressure in both arms. Should one pulse be found much stronger than the other, measuring blood pressure in both arms is imperative.

An abnormally slow rise suggests an arterial outflow obstruction such as aortic valve stenosis or brachial atherosclerosis. An exaggerated fall in pulsatile dynamics after peak is indicative of aortic insufficiency. Perceiving these subtleties, admittedly, comes only with considerable practice of palpating normal and abnormal peripheral pulses.

Doctors of the past era attempted to diagnose valvular heart disease by the nuances of the peripheral pulse. It is enough for the modern clinician to appreciate the dynamics of the normal, hyperactive, and hypoactive pulse.

Hyperactive Arterial Pulse

The bounding pulse may reflect increased metabolism, as is typical of hyperthyroidism. Fever and exposure to heat are other causes and relate to generalize peripheral vasodilation.

In the realm of rare causes of the exaggerated radial or ulnar pulse is coarctation of the aorta. The condition is sometimes first discovered in an

adult who has likely long suffered from its oversight. Of course, palpation of pulsatile flow in the legs is indicated to check for a paradoxically marked reduction, and then this possibility can be considered. Patients with coarctation of the aorta have serious limitations of lower limb activities. The patient's life story will readily reveal how he or she has adapted, perhaps unwittingly, to the problem. The adrenergic-driven hyperdynamic pulse from anxiety and, rarely, from pheochromocytoma can be recognized by other peripheral signs such as cool and moist skin and elevation of blood pressure.

Hypoactive Arterial Pulse

Decreased dynamics of pulsatile force (the "thready pulse") is a sign of hypotension. The cause may be threefold: critically reduced cardiac output for any reason, the reduction of peripheral blood flow owing to blood loss, or the divergence of blood to the viscera, as seen in "vascular' shock. Tachycardia is, of course, a normal response to these critical events. Cold, blanched hands and face along with overt sweating (producing a "clammy skin") are from intense stimulation of the adrenergic system in response to a critical stage of hypotension.

A rigid artery from arteriosclerosis may feel hypodynamic yet require increased pressure in order to obstruct flow. When blood pressure is measured over the artery, the reading will be elevated. This higher recorded blood pressure is known as "pseudohypertension."

Should one radial pulsation be absent or substantially reduced, the clinician can further evaluate peripheral blood flow with the "Allen test." He or she uses the finger tips to exert strong pressure simultaneously over both the radial and ulnar arteries on one wrist. The patient then clinches the fist firmly five times in rapid succession and then opens the hand fully. On release of the radial artery pressure, the palm rapidly recovers its normal color within 10 s. The test is then repeated, this time releasing pressure over the ulnar artery while maintaining

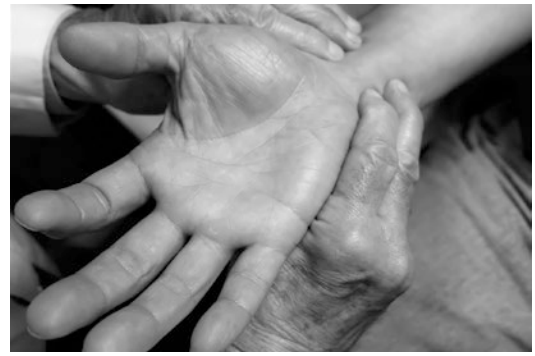

Fig. 4.1 Allen test

obstructing pressure on the radial artery. Blockage of either of these arteries is revealed by markedly delayed refilling while the other artery is pinched shut (Fig. 4.1).

The Allen test may be particularly useful *before* performing an arterial puncture for a blood gas study. A congenitally absent or poorly developed peripheral radial or ulnar artery occasionally occurs; injury to the other from a needle stick could have a dire result.

If the distal pulsation is determined as normal, there is no need to palpate the brachial artery. Being familiar with the location of the strongest pulsation of the brachial artery, however, is helpful when using a blood pressure cuff.

Sinus Rhythm

The perceptive clinician can detect the presence of normal sinus rhythm from the peripheral pulsation. The following sequence will confirm its presence. Arterial waveforms were recorded by Doppler ultrasound over the radial artery of a 24-year-old subject.

1. The resting cardiac rate is 70, the interval between beats extrapolated to 1 min DI. The subject takes a rapid, deep inspiration and maintains it for the duration of the tracing.
2. The heart rate suddenly increases to 97/min. The expanded lung volume induces an increase in blood volume in the pulmonary circulation, resulting in an abrupt reduction in

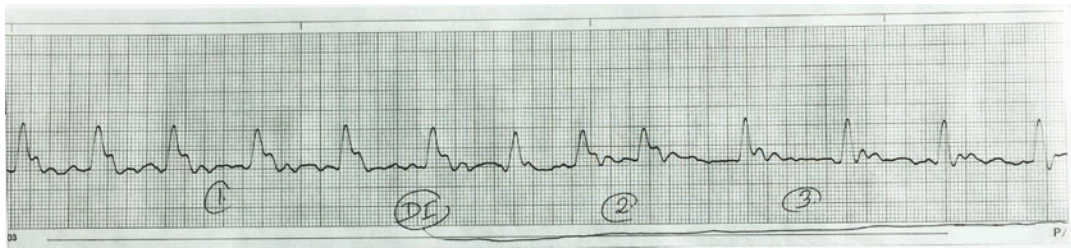

Fig. 4.2 Normal sinus rhythm. Electrocardiographic tracings

right atrial volume and pressure. Note that the amplitude of the waveforms is attenuated in these more rapid beats (Fig. 4.2).

3. The rate slows to 59/min. This response is caused by an increase in vagal tone from the expanded chest. An equilibrium of intra-atrial blood flow has been reestablished. The original amplitude of the waveform has returned.

If the breath were held for longer than half a minute, the heart rate will gradually accelerate. The acceleration is the response to accumulating carbon dioxide. As this response is delayed in onset but totally predictable, having the patient breath-hold beyond the time frame shown here serves little purpose.

This tracing demonstrates the exquisite sensitivity of the sinus node to pressure-volume changes in the right atrium. Typically on taking a deep breath, the heart rate speeds up for a few beats and then slows to below the original rate. The sequential changes of heart rate in response to a single held deep breath is a reliable indicator that the rate is controlled by the sinoatrial node. Ectopic rhythms – atrial, A-V junctional, and ventricular – do not respond in this highly predictable manner. These arrhythmias, whether in bradycardia or tachycardia, may exhibit clocklike regularity or be totally irregular; both are independent of the dynamics of breathing. Premature ectopic beats confound the evaluation, but the principle of sinus node reactivity to changes in intra-atrial pressure holds.

The changes in heart rate with a deep inhalation can usually be appreciated easily by palpation of the radial pulse. With aging, vagal tone tends to diminish. Consequently, the induced slowing of heart rate is less prominent in the older patient.

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The One-Minute Physical Examination

5

Clearly absurd, the title introduces this section to make a point. The initial part of the physical examination already outlined can – when carefully and rapidly performed in an organized sequence – provide reasonably reliable information about the general condition of the patient. It is akin to judging the health of a tree by examining a leaf. If all details of the leaf are normal, one can assume that its nurturing twig, branch, limb, trunk, and roots are functioning, even if other limbs are missing or if there is an owl's nest in a decayed cavity somewhere up there.

To review at this early point in the physical examination, the clinician has already gained a general impression of the patient's ability to make facial contact along with an assessment of facial characteristics, the ease of breathing, and the peripheral effectiveness of major organ systems. In effect, there has been a systematic gathering of information about the mental state as well as nutrition, circulation, respiration, and neurologic functions. Indications of endocrine, joint, and connective tissue disorders may have already been discovered as well as signs of infection. Abnormal findings can guide the clinician's focus as he or she proceeds into the remainder of the physical examination.

The sequential development of the one-minute physical examination is outlined with the estimated time when no abnormalities are present.

1. Face. First impression for eye contact, affect, symmetry, nutrition, and effort of breathing (5 s).
2. Palms up. Observe for resting tremor, color, skin lesions, deformities, joints, and muscle bulk (10 s).
3. Stroke palm. Feel for moisture, temperature, and texture (5 s).
4. Palms down. Observe nails and skin lesions (5 s).
5. Arms forward and palms up. Observe for extended tremor and palmar drift (10 s).
6. Wrist. Flexibility, skin lesions, and pulse (10 s).
7. Deep inspiration. Cardiac rhythm (15 s).

Should no significant abnormalities be found, it is a minute well invested. The clinician can be reasonably assured that no major systemic condition with impending consequence has been overlooked. If, instead, an abnormality is discovered, a decision must be made as to its immediate significance. Is the finding critical or noncritical? Does it demand immediate or

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elective attention, if any? The well-practiced clinician can quickly sense the urgency and gravity of such important and oftentimes difficult decisions. Certainly, skill and speed in this early part of the physical examination come with practice, much like the concert musician working on scales and arpeggios.

Admittedly, sorting out the difference between serious or not serious in medical practice is not easy, and it often has little to do with the intensity of a symptom. It is arguably the most pressing decision in the clinician's day. Put in another way, using the words of Sherlock Holmes in *The Reigate Puzzle*, "It is of the highest importance in the art of detection to be able to recognize, out of a number of facts, which are incidental and which vital. Otherwise your energy and attention must be dissipated instead of being concentrated." This is where experience comes in. Not every clinician is a Sherlock Holmes, but remember that the author of detective stories starts at the final revelation and works backward to construct the sequential mystery, not a luxury of the clinician at the bedside. It is perhaps not surprising that these words of a fictional detective were written by a physician.

The one-minute physical examination is not meant to replace the comprehensive physical examination. Instead, it is an approach for beginning a complex process by paying attention at first to the delicate, final benefactor of all the organ functions: the human "leaf."

Forearm

6

Essentials

Texture
Spots
Rash

Texture

Palpation for overall skin texture is probably best performed routinely on the forearm on the dorsal side. Testing involves gently pinching the skin and suddenly releasing it. Easily appreciated are the normal differences in elasticity and thickness between an infant, a teenager, someone middle aged, and an elderly person. Even so, the skill to detect discrepancies requires practice (Fig. 6.1).

Fig. 6.1 Pinched skin, normal

Thin Skin

Aging

With advanced aging, skin loses its elasticity. When held between pinching fingers, it feels excessively thin. Smoking and chronic hypoxia (chronic pulmonary insufficiency or living at a very high altitude) reduce skin elasticity and thickness prematurely.

Dehydration

A sign of substantial dehydration is the slow return of the puckered-up skin. It has, however, a rather low degree of sensitivity. Corroborating findings – dryness of palms, tongue and mucous membranes – are, of course, ancillary sites for confirming an impression of dehydration. The optimal site for observing evidence of dehydration is the membrane between the lower lip and the gum.

Systemic Sclerosis (Scleroderma)

In systemic sclerosis, the surface of the skin is not only thinned; it is taut, characteristically resisting

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pinching. This important physical sign should prompt a scrupulous search for related signs and symptoms, already mentioned in Chap. 3.

Thick Skin

Edema

Edema of the arms may be a manifestation of anasarca, certainly evident throughout the body. The upper body distribution is also more common in conditions causing hypoalbuminemia, such as the nephrotic syndrome. Unilateral arm edema is most likely from an infiltrated intravenous infusion or from obstructed lymphatic drainage. Transient edema of the forearm can occur overnight if the arm is left dangling over the edge of the bed or chair, as sometimes happens with intoxicated or over-sedated people.

Lymphedema

Lymphedema of an extremity resists pitting and when long-standing the skin becomes markedly thickened from fibrosis. Obstructed lymphatic drainage in the axilla and the absence of lymph nodes and channels (whether congenital or from extensive breast surgery) are the more common causes.

Obesity

Obesity can be confused with edema. Excessive adipose tissue, however, does not have the pitting characteristic of edema. Furthermore, excessive adiposity tends to taper at the wrists and beyond.

Myxedema

The skin in myxedema has a unique texture. It can be compressed to some degree, as if working with dough, but is resistant to pitting. Furthermore, the texture of the skin feels rough and excessively dry. Pinched skin is slow to return to its former layering. The clinician who examines a patient with myxedema should take the opportunity to become comfortably familiar with the unique texture of the skin. Of course, myxedema is associated with a host of other abnormalities involving mentation, voice, hair, temperature, and heart rate.

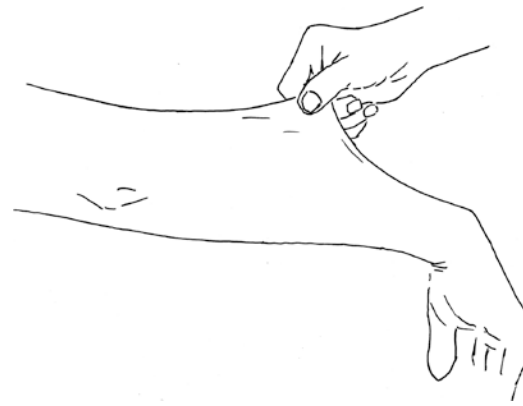

Fig. 6.2 Ehlers-Danlos syndrome

Scleredema

Not to be confused with scleroderma, this rare condition presents as an indurated, almost wood-like, non-pitting thickness. In addition to the arm, it tends to appear in the nape of the neck and the back. This thickening of the skin is generally associated with poorly controlled diabetes in which retinopathy and neuropathy frequently coexist.

Loose Skin

The Ehlers-Danlos syndrome is a rare genetic disease characterized by extreme stretchiness of the skin, far more than from aging or dehydration. The excessively loose skin is readily demonstrated on pulling up the skin on the forearm. The fragile skin may be easily bruised. Hypermobility of joints is an often associated phenomenon (Fig. 6.2).

Spots

Petechiae

Diffuse petechiae are most obvious on the volar surface of the forearm. The clinician must then sort out by palpation those petechiae that are flat from those that are raised. This differentiation is critically important diagnostically. The subject of petechiae and its larger expression, purpura, is

expanded here at the forearm because it is the region in which these conditions may be most obvious.

Flat Petechiae

Petechiae that cannot be felt as the fingertip brushes over them result from either capillary or platelet tissues. A few of these macular petechiae may be present from entirely benign causes such as mild and unnoticed trauma, even after a tight blood pressure cuff has been applied. They are more commonly seen in conditions that compromise the structural integrity of capillaries, including aging and nutritional deficiencies, by noninflammatory conditions.

Macular petechiae may also reflect a severely reduced number of platelets or ineffective function of platelets. Such defects may be inherited, or it may be an acquired reaction to some drugs.

Raised Petechiae

Petechiae that are raised to the touch imply an inflammatory process that compromises the integrity of capillaries. Causes of this expression of vasculitis range from one of many infectious diseases to autoimmune conditions. Rocky Mountain spotted fever is a prototype of the former (and is not confined to the mountains) as is meningococcemia and toxic shock syndrome. Immunologic diseases (congenital or acquired) that target the microcirculation cause palpable petechiae.

Systemic vasculitis (Henoch-Schonlein purpura) can result in cutaneous petechiae – locally or extensively – as well as bleeding into the large joints, the intestinal tract, and the kidneys. Emboli from meningococcal, gonococcal, or other infected loci may also produce palpable petechiae.

Purpura

Visible bleeding into the skin denotes purpura (or ecchymosis). Size is a factor when differentiating purpura from petechiae: hemorrhagic lesions larger than 3 mm are designated purpura. Inflammation-induced petechiae may coalesce, forming purpuric areas.

Extravasation of blood into the forearm is certainly familiar where venipunctures and intravenous infusions are frequent. Benign "senile ecchymoses" are commonly found in the very elderly in whom elasticity of the skin is greatly depleted and blood vessels highly fragile. Minor trauma, including pinching or scratching, is enough to cause patches of purpura, particularly in the forearms.

Other causes of weakened collagen tissue must be considered for evaluating purpuric lesions. These include scurvy, an affliction of monumental importance in the Age of Discovery when ocean-going sailors were at sea and on diets lacking vitamin C for months. Scurvy is still seen in undernourished populations. A common cause of purpura today is excessive glucocorticoid stimulation, either endogenous (Cushing's syndrome) or from prescribed steroid therapy. Purpura occurs in the skin in nearly half the cases of systemic amyloidosis [1].

Just as it applies to petechiae, the important point is made that purpuric spots caused by inflammatory conditions tend to be papular. Purpura from noninflammatory causes is macular.

Certainly, coagulopathy from any cause may also be responsible for purpuric lesions. Unexplained purpura, of course, deserves a workup for platelet population and function as well. When they present as hemorrhagic bullae, called "purpura fulminans," disseminated intravascular coagulation must be considered.

The age of a purpuric spot or an ecchymosis can be roughly estimated. Initially, in subcutaneous bleeding, they are deep purple. Over the subsequent 3 weeks, the area will slowly develop a yellowish hue and finally a light brown stain from deposited iron before disappearing altogether.

Neurodermatitis

Excoriations that are self-induced are particularly common on the forearm because this loca-

tion is an easy access area for scratching. Lesions are generally punctate. Many are yellowed from crusted serum or dark-red from clotted blood. They may have a linear distribution, often with scratch marks nearby. Healed excoriations tend to leave white streaks. Habitual scratching may be from the attempted relief of a pruritus-causing underlying condition. It may also stem from a manifestation of nervousness, akin to nail biting. Unexplained pruritus and incessant scratching are common problems in the elderly population.

Reference

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Elbow

7

The elbow is a hinge for flexion and extension while its pulley mechanism allows for supination and pronation of the forearm. Palpation and range of motion of the elbow can be quickly performed in the comprehensive physical examination.

Essentials
Range of Motion
Flexibility

Carrying Angle

Normally the carrying angle – the maximum extension determined by the coracoid process – is slightly greater in women. It is uniquely exaggerated in Turner's syndrome.

Flexion/Extension

Holding the arm at the wrist, slowly flex and extend the forearm to its full range of motion, paying attention to fluidity of movement. Palpate the elbow with the other hand during this maneuver. Any discomfort in the elbow or limitation of movement must be investigated. Also, feeling a jerky, alternating resistance and relaxation throughout the movement (called the "cog-wheel rigidity") is a reliable sign of Parkinson's disease.

Passive range of motion of the elbow can be justifiably skipped in relatively young persons with no local symptoms and taking no drugs. This ten-second maneuver is recommended, however, on patients well into middle age and in younger patients who are taking potentially spasticity-inducing drugs.

Pain/Tenderness

Passive movement of the elbow against resistance helps to distinguish pathology in muscles and their tendons from that of joints and periarticular tissue. When extension and flexion of the elbow hurt worse if performed against moderate resistance, pain in local muscles or tendons rather than arthritis is most likely. Many of the problems of the elbow are related to overuse activities that can be reproduced in testing.

Lateral Epicondylitis

Commonly known as "tennis elbow," the large majority of people with lateral epicondylitis are not tennis players. Tenderness is typically present over the lateral epicondyle of the humerus where the extensor tendon originates (Fig. 7.1).

In addition to location, lateral epicondylitis can be identified by increased pain on resistance to dorsiflexion of the wrist with the elbow in

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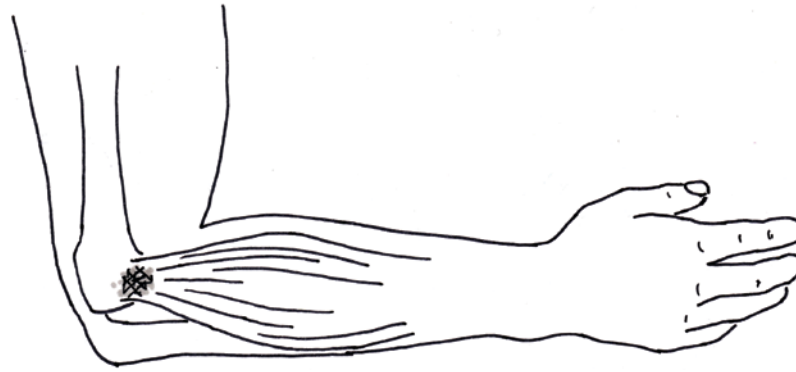

Fig. 7.1 Lateral epicondylitis of the elbow

Fig. 7.2 Medial epicondylitis of the elbow

extension. Pain can also be exaggerated when making a strong grip or on supinating the forearm against resistance.

Low-level but persistent use of the elbow against resistance is usually an underlying feature. Demanding occupations frequently repeated strong forearm movements, such as in tuning a piano and using a hand-driven pump.

Medial Epicondylitis

"Golfer's elbow" is pain and tenderness from medial epicondylitis. Diagnostic testing is characterized by increasing pain when resistance is applied to the flexed wrist with the elbow extended. The problem, by the way, is rarely present in people who play golf (Fig. 7.2).

Olecranon Bursitis

The olecranon area is highly susceptible to direct or overuse trauma. Swelling from olecranon bursitis is easily visible. Passive flexion and extension of the elbow are not impaired and do not exaggerate any associated pain. Tenderness, however, may be present at the tip of the olecranon process, with or without palpable thickening of the bursa (Fig. 7.3).

An effusion in the olecranon bursa following trauma could be associated with microfractures. Alternatively, the bursa may contain crystals of urate or calcium phosphates that could be incidental to the inflammation or are actually causal. Rheumatoid arthritis is frequently complicated by olecranon bursitis; its onset is typically associated with acute inflammatory joint symptoms in other

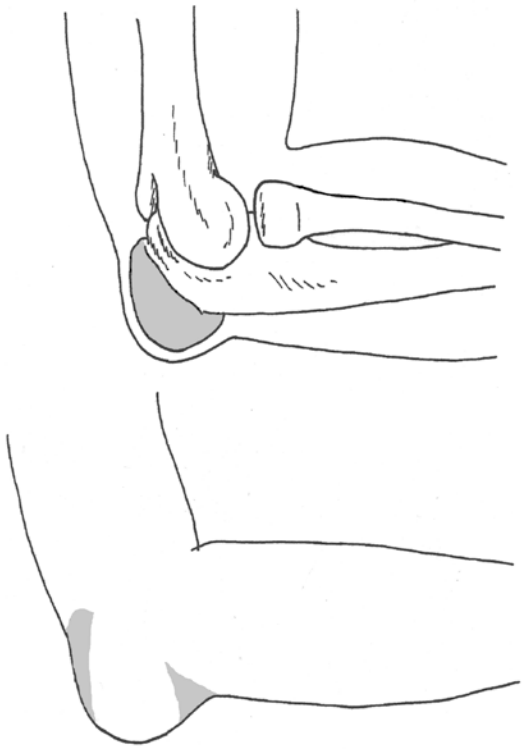

Fig. 7.3 Olecranon bursitis

areas. Rheumatoid nodules are particularly prone to develop over the long bones on either side of the elbow (see below under section "Bumps").

Olecranon bursitis is generally benign, but occasionally it is septic. Tenderness, erythema, and increased temperature will favor the latter suspicion. Infection is far more likely if a break in the overlying skin preceded the swelling.

Osteoarthritis

Generally, pathology in the soft, periarticular tissues of the elbow does not tend to increase pain and tenderness on movement against resistance. Osteoarthritis can affect only the elbow, but it is decidedly uncommon as a single site. When present, signs of osteoarthritis are nearly always present in more typical joint, namely, the hips and knees.

Referred Pain

Pain in the angina pectoris can sometimes be referred to the elbow. The left elbow is usually involved, but it could be in both elbows. At times, pain here can be more intense than the chest pain. Rarely, it is the *only* symptom of an acute coronary artery event. It comes and goes in response to the same activities as typical angina. Such pain, however severe, is not associated with tenderness, and its absence would be a strong clue to a referred pain origin.

Rash

Psoriasis

Psoriasis has a predilection for the elbow and the knee; these are usually the first involved areas. It can occur initially, however, almost anywhere: commonly the scalp, palms, nails, or gluteal folds.

The cause of psoriasis is overproduction of skin cells when the normal feedback system for cell reproduction functions inadequately. Typically, lesions consist of the heaped-up skin. Reddened papules arising from inflamed skin tend to merge, causing patches that become overlain with discarded flakes of silver-colored scales. This highly recognizable pattern of psoriatic skin occurs most often over the olecranon area where it varies in size from tiny (and often barely noticeable) to extensive.

Psoriasis may develop where the normal skin has been traumatized (Koebner phenomenon). Teasing a flake off a psoriatic lesion exposes a spot of raw, bloody surface (known as Auspitz's sign). Slight rubbing over a patch of psoriasis causes sloughing of the dermis (Nikolsky's sign).

Bumps

Rheumatoid Nodules

Nodules palpated in the olecranon area are highly distinctive features of rheumatoid arthritis. They favor bony prominences. Nodules often extend distally onto the ulnar bone. More than likely, they will occur in both elbows. Rheumatoid nodules are fairly mobile under the skin although some are firmly attached to underlying tendons. Increased warmth and tenderness over the nodules favor a rheumatoid etiology.

Tophi

Concretions of monosodium urate crystals in the elbow are common in tophaceous gout. The urate deposits are hard, yellowish, and non-tender; they are generally irregular in shape. Sometimes tophi are gigantic in size. Tophaceous gout, by the way, is not the painful inflammatory picture of acute gouty arthritis although these conditions can occur together at the same site.

Calcium pyrophosphate deposition disease (or pseudogout) can also leave concretions in the elbow that are impossible to discern from those caused by gout and from rheumatoid nodules [1].

Epitrochlear Lymphadenopathy

Lymph nodes near the elbow are rare, yet they have particular significance. They are most likely located on the ulnar aspect about 3 cm above the olecranon process. A palpable lymph node here alerts the examiner to the possibility of infection distally. A red streak extending from a finger along the forearm to the elbow is lymphangitis, what old-timers call “blood poisoning.”

If there is no point of infectious entry in the forearm and hand, the most likely causes of epitrochlear lymphadenopathy are infectious mononucleosis or HIV. Much less common etiologically are sarcoidosis, lymphoma, and chronic lymphocytic leukemia.

Reference

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Vital Signs

8

This chapter expands on blood pressure and body temperature as they vary from normal limits. Cardiac rate and respiratory rate are, of course, vital signs, but for continuity they are covered in Chaps. 17 and 18.

Essentials
Blood pressure
Temperature

Blood Pressure

At this point in the examination of the arm, it is expedient to measure blood pressure. By now, the patient – having had some exposure to the examiner's questions, gaze, and physical hand contact – is likely to have overcome the anxiety that produces "white-coat hypertension." Furthermore, the development of the examination thus far has brought us to the appropriate region.

Trained paraprofessionals, in a growing trend of clinical services, routinely take vital signs. Furthermore, they are being determined by automated instruments with ever-increasing frequency. It is strongly recommended, for accuracy and patient reassurance, that the primary clinician measures and records these parameters (or at least blood pressure) personally upon hospital admission as well as during a new patient examination. When the focus is management of hypertension, blood pressure should be taken by the clinician at least once every visit. Blood pressure varies substantially throughout the day, while its recording varies among those who measure it. Furthermore, the person who must make decisions based upon a finding is likely to be more attentive to the quality of that finding.

The "ideal" resting blood pressure is considered 120/80 mm Hg. Everyday stimuli cause such variations, however, that this value is seldom recorded. Children tend to have lower resting blood pressures than adults. A reading of 90/60 mm Hg in a young and healthy woman is fairly common. Symptoms of postural hypotension, incidentally, occur more often in these normal, low-pressure individuals.

Blood pressures taken with the patient sitting or supine are not significantly different. Wrapping the cuff around loose-fitted clothing has been shown to probably not induce a significant artifact, although direct skin contact is recommended. Palpate the brachial artery in order to place the diaphragm of the stethoscope on the most robust pulsation. Taking the weight of the arm by cradling it allows the clinician to hold it firmly against the skin with the supporting hand. This method is especially useful when determining blood pressures with changes in patient position, described below in the examination for "postural hypotension" (Fig. 8.1).

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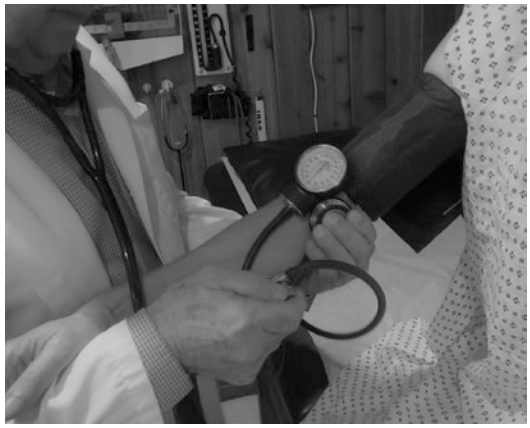

Fig. 8.1 Blood pressure cuff, holding position

Taking blood pressure in only one arm is reasonable provided the palpable amplitude of the radial or brachial arterial pulsations are equal and when there are no symptoms that suggest regional arterial insufficiency (such as pain in one arm after sustained, forceful exertion of the arms). In these exceptions, measuring blood pressures in both arms is indicated.

Bilateral measurements are also appropriately taken in the initial evaluation of a patient with an elevated pressure. A consistent difference of 20 mm Hg or more between arm pressures is considered abnormal [1]. The usual cause, if not the operator's inconsistency, is stenosis of the subclavian artery on the lower pressure side.

There is an enormous variation in blood pressure in a normal person, almost from moment to moment, depending upon the degree of mental challenge, body position, ambient and body temperatures, and recent exertion. A difference may confound the results going from one arm to the other. For example, the patient may begin to fret about the need to take a blood pressure on the other arm, causing some adrenergic stimulation. Such physiological variations must be kept in mind in making decisions in the diagnosis and treatment of blood pressure disorders.

Blood pressure taken immediately on standing is useful in patients describing symptoms that suggest orthostatic hypotension. Periodic posture-changing measurements are recommended for those on antihypertensive medications, all of which in some way blunt the mechanisms that normally regulate circulation, including adjusting to changes in position. A procedure to check for orthostatic hypotension is described under section "Reduced Blood Pressure."

Elevated Blood Pressure

Blood pressure of 140/90 mm/Hg has been well established as the upper limit of normal for the calm patient at rest. (The clinician must keep in mind that the blood pressure is predictably a bit higher in the office setting than at home.) The goal for treating hypertension is to maintain the resting blood pressure below this level and preferably nearer to a systolic pressure of 120 mm/Hg. The benefit of this lower range has been demonstrated in patients with increased risk from cardiovascular disease [2]. Concern in the aging population, however, is the greater sensitivity to side effects of antihypertension medications, most especially by inducing symptoms from postural hypotension [3]. The frail elderlies are particularly susceptible to such untoward effects of these drugs. Hypertension in the diabetic patient is ideally treated to a range of 120/80 mm/Hg range or lower, owing to the propensity for incurring microvascular injury [4]. Attaining this lower level in patients with chronic renal disease is also desirable.

Strong emotional stimuli, including nightmares, cause both systolic and diastolic blood pressures to rise precipitously to extreme levels. The same can be said for isometric exercise**,** that is, strenuous effort against strong resistance such as sustained by heavy weight lifting. Both systolic and diastolic pressures are suddenly elevated, sometimes to extreme levels by powerful emotional reactions and on performing feats of strength. There is a rapid return to resting levels when the stimulus subsides (Fig. 8.2).

Isotonic exercise, on the other hand, induces a different response. With repeated effort well below maximum (such as running, biking, rowing), only the systolic blood pressure rises, and it does so gradually. The diastolic pressure remains the same or falls somewhat with continued

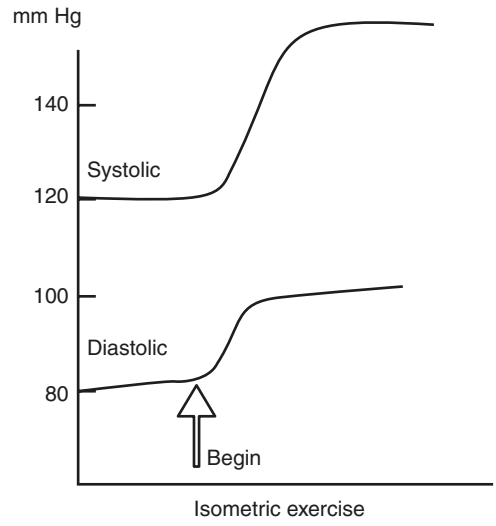

Fig. 8.2 Isometric changes in heart rate

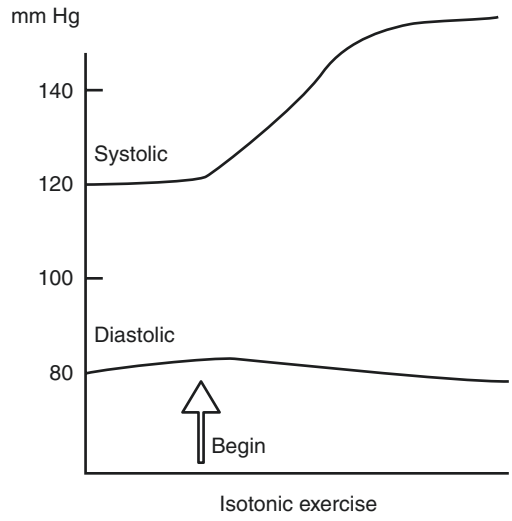

Fig. 8.3 Isotonic changes in heart rate

effort as core heat builds up. It takes about 3 min of isotonic exercise at a uniform degree of exertion to reach a steady state. Then the blood pressure persists at that level as long as that same level of effort is carried out [5]. Those trained for that particular exercise will come to a steady state a half minute or so earlier (Fig. 8.3).

Within 3 min of stopping isotonic exercise, the blood pressure returns to resting levels. This predictable response and time to baseline should be kept in mind when a blood pressure is found elevated in someone who has recently walked briskly from a parking lot or climbed a flight of stairs. The clinician should not attribute an elevated blood pressure to previous physical stimulation when measured more than 5 min after cessation.

Primary Hypertension

Sustained elevation of blood pressure is assumed to be primary (or "essential," to use an older term) when no specific cause can be determined. The two determinants of primary hypertension are (1) circulating volume overload and (2) pressor-driven vasoconstriction. These possibilities are sorted out by measuring plasma renin activity (PRA), a hormone that regulates adrenergic tone. The practitioner outside of academic circles has, at yet, no practical means to distinguish the two determinants by PRA levels; differentiation may best be inferred by the response to the chosen therapeutic regimen.

Secondary Hypertension

Secondary hypertension, on the other hand, has a discoverable explanation for its pathogenesis. Admittedly uncommon, these conditions are often overlooked by the unsuspecting clinician. The cause may eventually be discovered on evaluation of "resistant hypertension" or as incidental findings by various imaging techniques. For the patient, the implications of a delayed diagnosis often mean long periods of recurring symptoms and accumulating medical bills. In addition, the choice of drugs routinely used for initiation of therapy of hypertension can cause alarming symptoms when given to patients with certain forms of secondary hypertension.

In a nutshell, the causes of secondary hypertension as well as the clues to recognize it are outlined.

Coarctation of the aorta creates a great mismatch of pulsatile flow comparing upper and lower extremities. The cause is severe narrowing of the proximal aorta, just distal to the left subclavian artery. Robust, even hyperdynamic, radial pulses with absent or markedly diminished pulsations in the legs are defining. Blood pressure is high in the arms and almost unobtainable

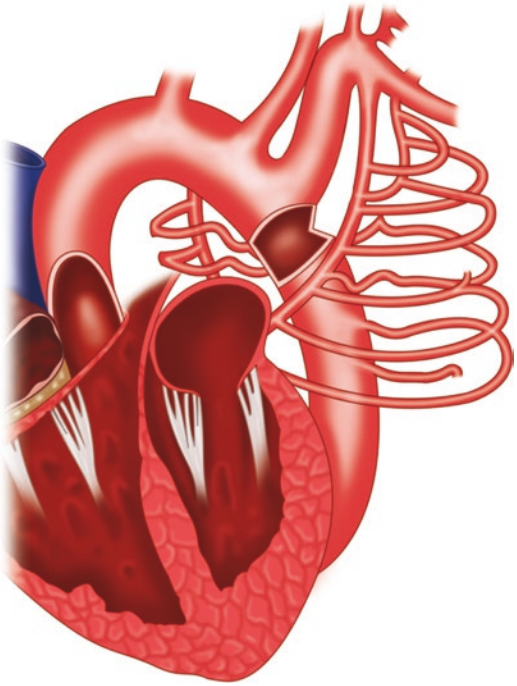

Fig. 8.4 Coarctation of aorta (Modified from Phillips [12])

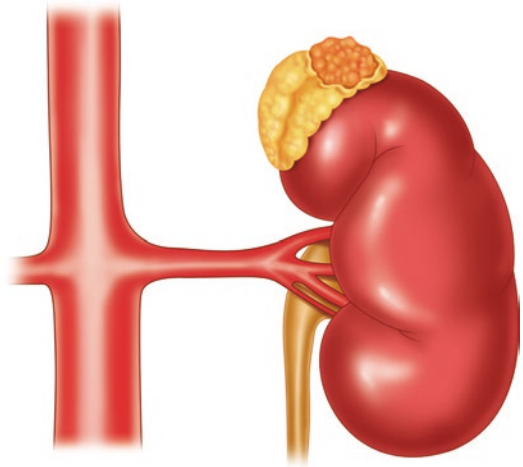

Fig. 8.5 Aldosterone-producing tumor (Modified from Phillips [12])

in the legs. The patient will have a lifelong history of poor exertional tolerance for ambulatory functions (Fig. 8.4).

The early diagnosis of *hyperaldosteronism* is basically made by an abnormality of serum electrolytes, hypokalemia being the hallmark feature. Serum sodium may be elevated or at the upper limit of normal. With these abnormalities, the preeminent symptom is weakness. Clearly, prescribing a "potassium-wasting" diuretic to treat hypertension in a patient with an aldosterone-producing tumor can prove a serious misadventure (Fig. 8.5).

Cushing's syndrome, the result of a sustained increase of endogenous cortisol production or from chronic use of corticosteroid drugs, links hypertension and diabetes as well as an electrolyte disorder, namely, hypokalemia and hypernatremia (similar to but not generally as severe as in a functional aldosteronoma). Other features are likely to be present on physical examination: hirsutism, striae (especially of the abdomen), and excessive fatty tissue of the cheeks, nape of the neck, and temporal area. The overall image is the "round face" of Cushing's syndrome as opposed to that of obesity (described in Chap. 11).

Because of its rarity and the generally transient and wide variety of symptoms experienced in *pheochromocytoma*, the diagnosis is all too often missed, even on evaluation by several clinicians over several years. The most common features of pheochromocytoma are intermittent hypertension that can sometimes spike into extreme levels, symptoms of intense adrenergic stimulation (sweating, trembling, palpitations), and, paradoxically, symptoms of postural hypotension. The sympathetic overload causes the pupils to widen (Fig. 8.6).

Although pheochromocytoma is a rare cause of hypertension, it is common in patients with extreme elevations of blood pressure. No age is free of this possibility. A beta-adrenergic blocking drug inadvertently given to a patient with pheochromocytoma can elicit an adrenergic crisis if the patient had not already been treated with an alpha-adrenergic blocker. The astute clinician who

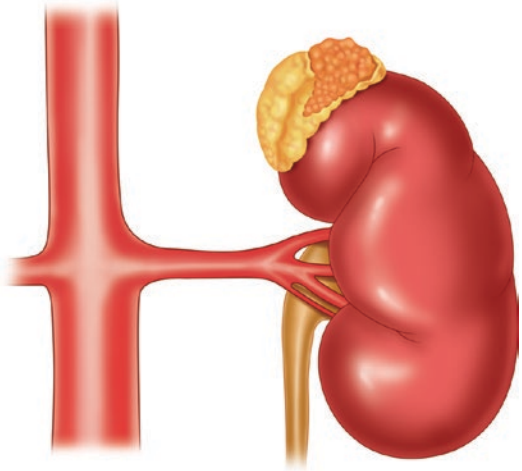

Fig. 8.6 Pheochromocytoma (Modified from Phillips [12])

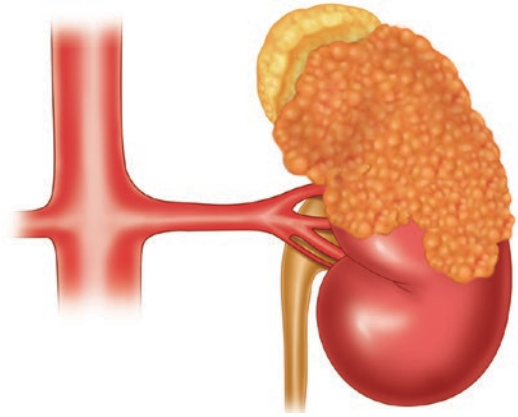

Fig. 8.7 Wilm's tumor (Modified from Phillips [12])

recognizes this condition can spare the patient a series of disabling and potentially dangerous symptoms.

Severe hypertension by *drug induction* generally involves cocaine. With non-measureable concentration of the drug in any format, overdosing is common. The clinical presentation can simulate in every way that of pheochromocytoma. Blood pressures may surge upward to extreme degrees. When a high-performing athlete without known risk factors sustains a stroke, the possibility of a cocaine-induced reaction should cross the mind. See Chap. 14 for description of a physical sign that strongly suggests chronic cocaine abuse.

Phencyclidine (PCP), also known as "angel dust," has a powerful excitatory effect on the sympathetic nervous system and on neuropsychiatric functions. In addition to raising blood pressure to alarming levels, it can induce hyperthermia, hypersensitivity to light and sound, and insensitivity to pain. What often call attention to the problem are marked and unpredictable swings in behavior.

The clinician on encountering a patient with extreme elevation of blood pressure (e.g., above 200 mm Hg systolic) should consider both a pheochromocytoma and illicit drug use, such as cocaine or phencyclidine.

Much milder in degree is the blood pressure elevation from vasoconstrictor drugs taken orally. Examples are over-the-counter drugs such as pseudoephedrine – meant to shrink swollen membranes in upper respiratory tract infections. Similarly acting products such as phenylephrine but administered nasally as topical vasoconstrictors are probably taken in too small a quantity to have any measurable effect on blood pressure.

Renal parenchymal disease of various kinds is characteristically associated with hypertension. It is associated with proteinuria and azotemia. The physical sign of "uremic frost" is present only in the far advanced stage of renal insufficiency, not a common finding in an age of dialysis. Virtually, the only such renal diagnosis detectable on physical examination is polycystic kidney disease in which both kidneys may be grossly enlarged and likely palpable. An exception is the Wilm's tumor of infants (noted in Chap. 20) (Fig. 8.7).

Renal vascular disease causing hypertension is usually from atherosclerotic stenosis, found almost always in elderly patients. Only in the advanced arterial obstruction is renal function compromised. An occasionally clue to renal artery stenosis is a bruit heard over the abdomen. Such a bruit, however, does not always mean that it is caused by stenosis of a renal artery (Fig. 8.8).

A second form of renal vascular stenosis is fibromuscular dysplasia from replacement of the arterial media with fibrous tissue. The lesion may be a single constriction of a series

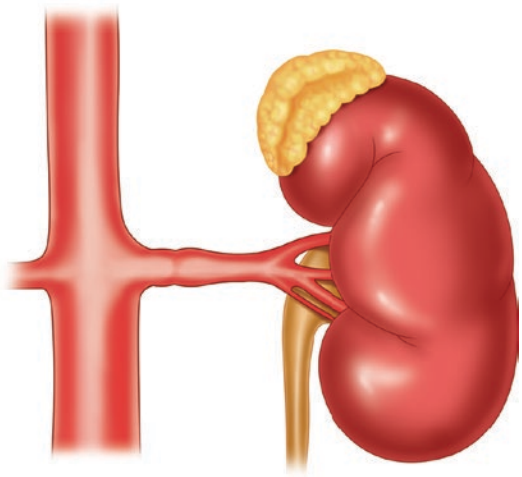

Fig. 8.8 Renal vascular stenosis: atheromatous (Modified from Phillips [12])

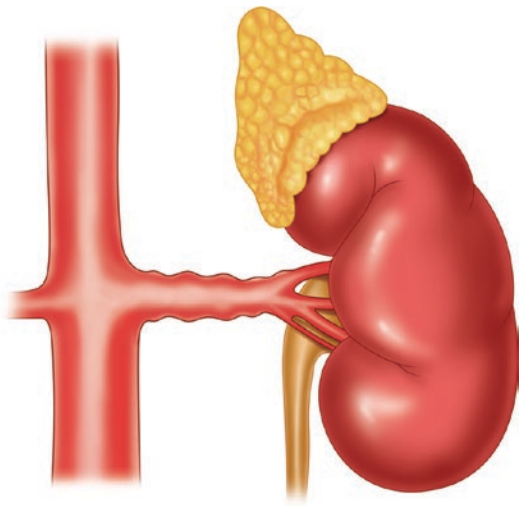

Fig. 8.9 Renal vascular stenosis: fibromuscular dysplasia (Modified from Phillips [12])

of constrictions likened to a "string of beads." The disease is most prevalent in relatively young adults but can occur at any age. It is often bilateral (Fig. 8.9).

Reduced Blood Pressure

The experienced clinician can discern hypotensive states by the blunted pulsatile quality of the peripheral artery. Although a systolic blood pressure below 90 mm/Hg is considered critical, a meaningful definition of hypotension depends more on the effectiveness of circulation to support all the organ systems than upon an arbitrary number. Adequate blood flow in the coronary arteries depends upon blood pressure of at least 70 mm/Hg.

In this complex subject, the clinician must identify which of the determinants of the circulation is the underlying cause of hypotension:

1. *Central pump failure*. Myocardial infarction is the preeminent cause. Episodes of sweating and diffuse vasoconstriction are adrenergic responses to hypotension from cardiac dysfunction of sudden onset.
2. *Vasodilation*. An excessively hot ambience fully activates the heat-dissipation mechanism through sweating and dilation of cutaneous vascular beds. Fever also promotes marked vasomotor dilatation. One form of anaphylaxis involves marked vasodilation of the visceral bed, resulting in areflexic vascular collapse elsewhere.
3. *Blood volume reduction*. Hemorrhage, whether external or internal, induces reflex tachycardia and vasoconstriction. Degrees of anemia from any cause that is severe enough to cause hypotension may be fairly well tolerated when the anemia is chronic. Remarkable, for example, are the severe degrees of anemia sustained in field workers with malaria.

In addition to hypotension from a reduced red blood cell mass, orthostatic symptoms can be caused by a contracted plasma space. This occurs from severe sodium depletion in adrenal cortical insufficiency. Hyperpigmentation accompanies this condition when the cause is primary failure of the adrenal cortex in which the pituitary gland releases high levels of both ACTH and melanin-stimulating hormone. Hyperpigmentation does not occur when the cause of adrenal cortical insufficiency is caused by failure of the pituitary gland. Hypovolemic hypotension can also be the result of sudden withdrawal of long-term therapy with a cortisol-like drug.

Drug Effects

Drug-induced hypotensive reactions are likely far more commonplace than generally reported by patients. It should be kept in mind that a drug used to control hypertension blunts one or more of the three determinants of circulation: cardiac output (heart rate plus stroke volume), peripheral vascular resistance, and blood volume. Many hypertensive patients take multiple drugs that blunt two or three of these determinants. It is small wonder that they frequently experience hypotensive episodes of varying intensities.

Postural Effects

Virtually everyone has experienced the "graying out" on first standing after sitting or lying in strong sunlight for a long time. Asking about this familiar symptom, incidentally, is helpful when evaluating the "dizzy" patient and attempting to sort out light-headedness from vertigo.

Orthostatic symptoms (i.e., "light-headedness" on first standing) can usually be clearly identified at the bedside. Certainly, measuring blood pressures during recumbency then immediately on standing can be extremely revealing concerning cardiovascular dynamics. Persons who complain of postural light-headedness, those who are on medications for hypertension, and others with significant anemia under consideration for blood transfusion are worthy candidates for this simple testing. Concerning the latter condition, the demonstrated tolerance for supine-to-standing testing may be a better guide than an arbitrary level of hemoglobin as an indicator for blood transfusion.

Testing for orthostatic hypotension by tradition has been a supine blood pressure measurement followed by measurement 3 min after standing, allowing for circulatory adjustments of positional changes. The protocol, however, misses those physiological adaptations (or lack of adaptations) that occurred immediately on standing and may be responsible for symptoms.

The following description provides a simple bedside method for evaluation, not requiring a tilt table or other special equipment. With the patient supine for at least several minutes under optimal resting conditions, measure blood pressure in the usual way at least twice to insure that the pressure is stable. Insert the stethoscope earpieces, and place the stethoscope head over the brachial artery in readiness to repeat the measurement while cradling the arm. Then inflate the cuff to just above the determined systolic level. It is helpful to have an assistant determine heart rate either by precordial listening or by palpating the brachial or radial pulse on the opposite arm. These simultaneous measurements require that the assistant reach across the bed during the supine test to be on the side that the patient stands on to determine the heart rate change immediately on standing.

So prepared, have the patient stand as rapidly as feasible. The instant the patient is on his or her feet, lower the cuff pressure while listening for the systolic sounds. The finding will represent the new instantaneous hydrostatic pressure. Repeating the measure in 1 min or longer with the patient still standing further evaluates the dynamics of recovery.

The sudden change in position that causes the blood to shift to the legs imposes a reduction in the central blood volume by as much as 1 L. The immediate acceleration of heart rate for several beats demonstrates the exquisite sensitivity of the sinus node to changes in right atrial pressures and volumes. Cinching of the gravity-dependent peripheral vascular system is much slower, and sufficient vasoconstriction can be assumed when the heart rate returns to nearly that determined when the patient was supine.

Normally, both systolic and diastolic pressures fall slightly on sudden standing, only to return within a minute to the same or to slightly high levels than those recorded when supine. Involved in these highly synchronized actions are all three determinants of the circulation: (1) cardiac output, (2) peripheral vascular resistance, and (3) circulating vascular volume.

Excessive lowering of blood pressure on first standing suggests an aberration of one or more of these determinants. Antihypertension medications, of course, are common contributors. If light-headedness or its equivalent is experienced, blood flow to the brain has been transiently compromised. Loss of color vision is perhaps the most sensitive result of a critically reduced cerebral blood flow.

If the heart rate does not accelerate initially on standing, sinus node disease or excessive

pharmacological suppression of the sinus pacemaker may be suspected. The result is failure to increase cardiac output at a time of acute need. Another explanation is covered next under neuropathies of the autonomic nervous system.

Autonomic Effects

The delicate and exacting synchronization between the sympathetic and the parasympathetic nervous systems, of course, determines the success of maintaining regional blood pressures in rapidly changing body positions. A schematic of this interrelationship is depicted (Fig. 8.10).

Neuropathies involving the autonomic system may be an underlying and occult cause of orthostatic symptoms. The symptoms may be from disturbances of either peripheral vascular and/or to heart rate responses to changes in position. Certainly, neuropathies are responsible for a wide variety of symptoms related to the gastrointestinal tract, genitourinary system, and sudomotor activity. They may limit exertional tolerance by blunting the normal rate response of the sinus node to physical changes.

Cardiovascular neuropathies are most common in long-standing diabetes of both type 1 and

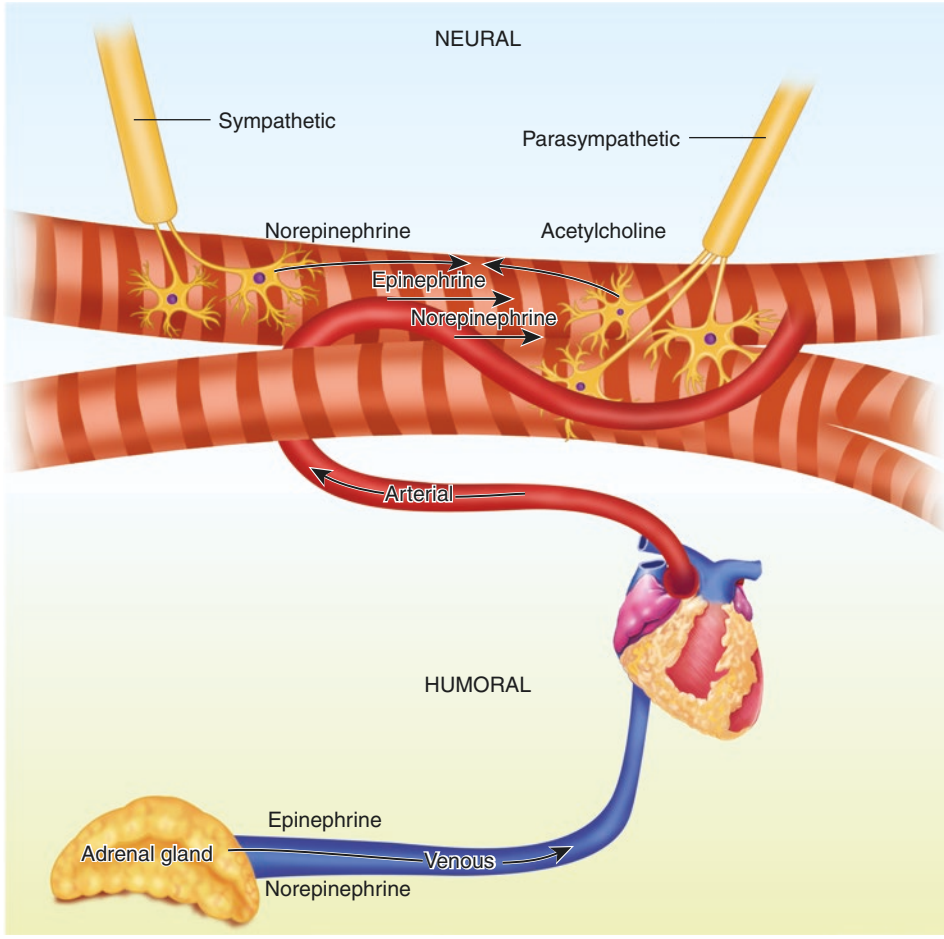

Fig. 8.10 Sympathetic and parasympathetic nervous system schematic (Modified from Phillips [13])

type 2 [6]. Orthostatic hypotension is also common in Parkinson's disease from sympathetic denervation [7]. Patients with this complication were found to have lower supine plasma norepinephrine levels than those without postural hypotension. Chronic abuse of alcohol must also be considered in neuropathic blunting of blood pressure responses to changes in body position.

Failure of the sinus node to accelerate in the orthostatic testing just described would help to identify such a propensity for an autonomic neuropathy. Alternatively, the clinician can check for the normal vagotonic responses using deep inhalation or Valsalva's maneuver. The normal response to the former was presented in Chap. 4. As an example for applying this association, finding a blunted response of the sinus node from these stimuli may help to explain the cause of neuropathic gastroparesis of a diabetic patient. This subject is explored further in Chap. 18.

A rare but debilitating form of postural orthostatic hypotension occurs mostly in older children, particularly in girls, and young women [8]. Known as the "postural orthostatic tachycardia syndrome" (or POTS), it is an expression of a disorder of autonomic vascular regulation. The tachycardia is secondary to delayed vasoconstriction in the gravity-dependent vessels. Symptoms, alas, may be misidentified as a panic attack, a paroxysmal ectopic tachycardia, or chronic fatigue syndrome.

Pulse Pressure

Systolic pressure represents the onset of increasing intra-arterial pressure from myocardial contraction. Diastolic pressure is its offset. The pulse pressure is the difference between the two. It can vary greatly in response to activity and exposures.

Increased Pulse Pressure

Peripheral vasodilation results in lowering of diastolic pressure, consequently increasing pulse pressure. The finding is expected in the febrile patient and in those exposed to a hot environment. The diastolic pressure can approach zero when the body is subjected to extreme heat for hours.

Measuring a pulse pressure greater than 80 mm/Hg is highly suspect for at least a moderate degree of regurgitation of the aortic valve [9]. A large arterial-venous fistula may also be responsible.

Decreased Pulse Pressure

Of most concern with reduced pulse pressure is the restriction of cardiac filling by a pericardial disease. The cause may be thickening of the pericardial membrane which – from an historical perspective – was most likely to be attributed to tuberculosis. Also, fluid accumulating in the pericardium can be sufficient to produce myocardial tamponade. The typical peripheral sign of either condition – pericardial membrane constriction or excessive pericardial fluid – is distention of cervical veins that do not collapse on deep inspiration. At the same time, the pulse pressure is further narrowed by a reduction in systolic pressure of more than 10 mm/Hg while the quality of the arterial pulsation may be dampened. These signs pertain to a more advanced degree of pericardial disorders but may be present in hypovolemic shock and after a pulmonary embolus.

Temperature

It may be convenient for the examiner to take an oral temperature while determining blood pressure. One ancillary benefit is that the determination will inhibit the patient from talking, an activity known to elevate blood pressure.

Oral measurement is not the most accurate place for taking core body temperature but is a more reliable site than the ear canal and axillary locations [10]. Rectal temperature is typically

about 1 °C higher than by the sublingual method. It increases slightly from morning to evening by about 1 °C. Even so, body temperature taken orally is normally regulated to a narrow limit between 34 and 35 °C (93.2–95.0 °F.). Indeed, the thermoregulating mechanisms aggressively protect the core temperature against a wide range of exposures to the shell.

The age-old practice of a mother feeling her child's forehead for fever has a sound anatomical basis. The arterial supply to the surface of the head is almost totally from the external carotid artery. The only artery arising directly from the internal carotid artery to the exterior of the head is the supraorbital artery. Consequently, it reflects core temperature. The supraorbital artery is a small branch of the long ophthalmic artery that originates in the internal carotid; it emerges at the medial aspect of the eyebrow.

Increased Body Temperature

Hyperthermia

Hyperthermia results from the failure of the heat dissipation mechanisms. A hot ambient temperature will eventually cause hyperthermia if sustained and beyond the capacity of circulatory adjustments (peripheral vasodilation and tachycardia) and perspiration. Ambient humidity is a critical factor since the principle mechanism for dissipating heat is by the cooling effect of evaporating sweat. Hyperthermia from disruption of central nervous system control in the hypothalamus by a stroke, injury, or tumor occurs but is rare.

Internal temperature rises markedly during extreme and sustained exercise even while surface temperature may fall due to blood shifting away from the skin. To illustrate the point, core temperatures above 40 °C (104 °F) have been documented immediately after a marathon run [11], while oral and skin temperatures can be below normal owing to intense peripheral vasoconstriction. High ambient humidity reduces evaporation of sweat and consequently accelerates the development of "heat exhaustion."

Perhaps the more common causes of hyperthermia are drugs that act on the central regulating system. These include the narcoleptics (such as haloperidol), releasers of serotonin (including the tricyclic antidepressants and monoamine oxidase inhibitors), and the psycho-stimulators (such as cocaine) that induce profound vasoconstriction. Among their many effects on the autonomic system, atropine and atropine-like drugs block normal perspiration. At high doses, loss of this critical important cooling mechanism results in dry skin and retaining the heat of metabolism leading to hyperthermia.

Fever

Fever is a rise of body temperature induced from endogenous pyogenic sources, namely, the cytokines that include the interleukins, tissue necrosis factor, and interferon.

The countless causes of fever can be sorted into five major classes:

1. Infection (acute, subacute, and chronic)
2. Immunologic (rheumatologic, usually with regional involvement and often constitutional symptoms)
3. Neoplasm (lymphomas, carcinomas)
4. Metabolic (hyperthyroidism, pheochromocytoma)
5. Vasoconstrictor drugs (cocaine, phencyclidine)

The development of fever can be inhibited by the salicylates, nonsteroid antiinflammatory drugs, and acetaminophen. Fever tends to be less than expected or may be absent altogether from infections in the very elderly and in persons with immunodeficiency conditions.

Decreased Body Temperature

Environmental Hypothermia

Hypothermia occurs from prolonged overexposure to cold temperatures. Heat loss beyond heat conservation is markedly accelerated when the body is immersed in water or covered with wet clothing. It may be little appreciated that hypothermia can occur in a warm climate from pro-

longed exposure to rain. Each drop running along the length of the body drips off a little warmer than when it landed. Slowly, body heat is drained. In summer, the condition of a hiker lost overnight in the rain is generally ascribed to "exhaustion" but is more likely hypothermia.

Because of the metabolic heat released in metabolism (about 60% of food energy), the body immersed in water for long periods will eventually become hyperthermic if the water temperature is above 94°. It will cool down rapidly in water below that temperature. The rate of change depends, of course, on many factors, the most important of which is the degree difference between water and core temperatures. Defense of body temperature in any environment can be severely compromised by extensive skin conditions such as psoriasis, dermatitis, and third-degree thermal burns.

Regulatory Hypothermia

Failing regulatory mechanisms that conserve heat will cause hypothermia. Head trauma and vascular events can disrupt thermal function of the hypothalamus. Alcohol probably has a complex action on depressing heat conservation, partly throughout diffuse peripheral vasodilation and partly from central nervous system effects. Neglect or inability to protect oneself against prolonged cold exposure further puts the alcoholic at risk. The reduced metabolism of myxedema typically results in a below normal body temperature. "Always feeling cold" is a typical symptom of the patient with hypothyroidism.

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Shoulder

9

The remarkable shoulder girdle is not only sturdy and pliant; it is, in fact, the most mobile of all joints. This combination is possible because the upper arm is snuggly fit by ball-in-socket joint into the heavily muscled scapula; the scapula rides freely above the rib cage that is attached by its acromial process to the clavicle. Where the clavicle is attached to the sternum is the only bone-to-bone attachment of the arm to the axial skeleton. This arrangement allows the amazing mobility of the shoulder while at the same time sacrificing some stability. It explains why a fractured clavicle is often the result of falling on the shoulder. For a functional unit so complex, there is small wonder that a great number of disorders of diverse nature can affect the shoulder girdle (Fig. 9.1).

Essentials
Symmetry
Range of motion
Drop arm test
[Motor]
[Sensory]

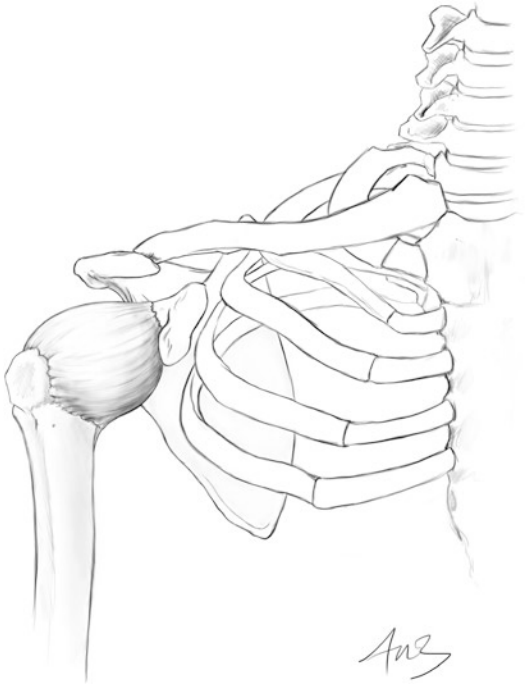

Fig. 9.1 Shoulder: normal

Pain in the shoulder region is one of the most common complaints in the primary care practice setting. Many of the causes are located within the shoulder: trauma, arthritis, capsulitis, polymyalgia rheumatica, and vasculitis. In addition, occupational and athletic pursuits often involve overuse. The clinician must keep in mind that pain in this area may be referred. Such indirect causes of pain around the shoulder are myocardial ischemia/infarction and dissection of the proximal aorta. Also included are diseases of the lung: pneumonia, pulmonary embolism, apical cancer, and mediastinitis.

The essentials for examining the upper arm and shoulder are quickly performed.

R.E. Phillips, *The Physical Exam*, https://doi.org/10.1007/978-3-319-63847-8_9

Interpretations of abnormal findings – asymmetry, pain, restricted motion, and weakness – from these simple movements are presented in outline below. A proper evaluation requires full exposure of the shoulder girdle for visualization.

Range of Motion

Evaluation of symptoms of the upper arm/shoulder requires testing the joint through its full range of motion. This can be done actively by having the patient abduct the arm and raise it to vertical and then reach back to touch the ipsilateral scapula. Similar motions can be provided passively up to the point of causing restriction and/or pain.

To begin examining the shoulder, look for symmetry, in particular, a bulge or depression over the lateral infraclavicular area.

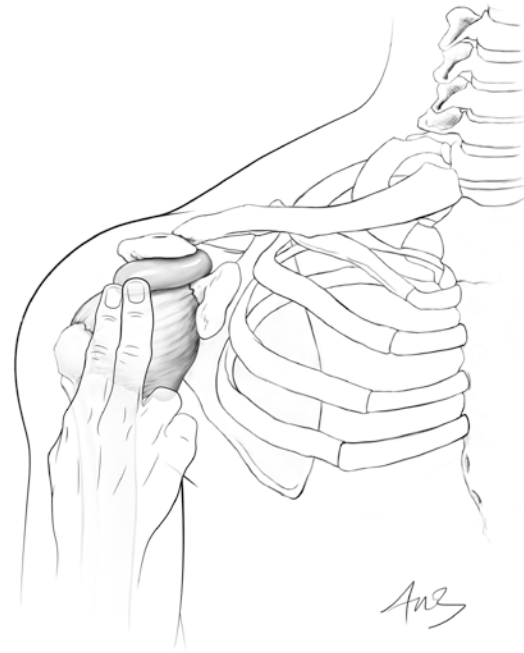

Fig. 9.2 Bursitis: subarachnoid-clavicular notch

Bulge

Bursitis

An abnormal prominence over the subacromial-clavicular notch may be caused by inflammation of the bursa just superior to the humoral head. Localized pain on rotation of the upper arm is characteristic. Tenderness at the site of pain confirms the diagnosis (Fig. 9.2).

Separation

Swelling and bruising over the clavicular-sternal joint results from a traumatic separation of the two bones. The common term "separated shoulder" is somewhat misleading. While pain may occur on movement, full range of motion is maintained. The usual causal injury is from a lateral blow to the body.

Depression

Dislocated Shoulder

Anterior dislocation of the humeral head can produce a depression over the glenoid fossa. The rounded head of the humerus is driven from the shallow glenoid socket in a forward and downward direction. The normally rounded lateral aspect of the shoulder appears flattened or squared off. Noted are restricted and painful abduction and external rotation of the upper arm (Fig. 9.3).

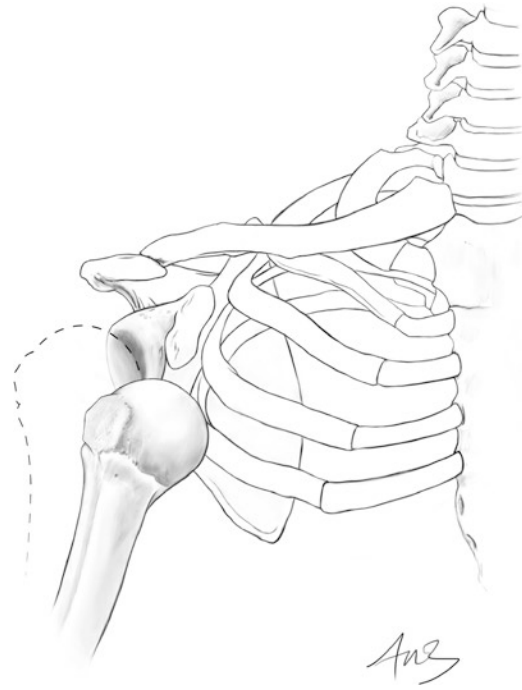

Fig. 9.3 Dislocated shoulder

The usual anterior dislocation occurs in contact sports and from falls. Not uncommon, however, is dislocation in the elderly person from minor trauma because of degenerative changes in the ligaments and cartilage in the glenoid "collar." The condition may be overlooked in an elderly person who complains not of pain but only of difficulty in moving the arm.

The far less common posterior dislocation of the humeral head may appear as a bulge over the scapula. The finding, however, is subtle and is easily missed if present at all. Limited movement of the shoulder in the elderly that is attributed to "old age" may actually be such a posterior dislocation [1].

Dislocation of the shoulder at any age can injure regional nerves. Consequently, a neurological evaluation of the arm is indicated when this injury is diagnosed. The axillary nerve, supplying the deltoid muscles, is most often injured [2]. If uncorrected, subsequent atrophy of the deltoids restricts abduction of the upper arm.

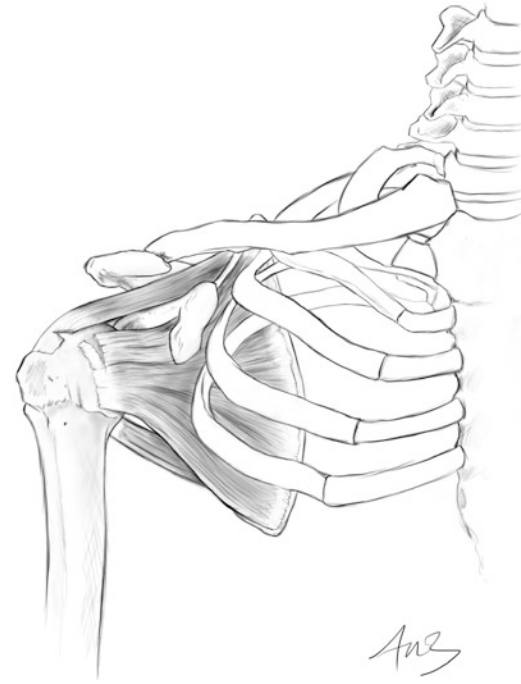

Fig. 9.4 Tear of rotator cuff

Restricted Range of Motion

Pain or physical limitation on trying to touch the scapula indicates bursitis, arthritis, dislocation, or fracture within the humero-acromial joint.

Rotator Cuff Disease

Pain emanating from the rotator cuff is the most common problem of the shoulder presenting in a primary care setting. The cuff is made up of the convergence of tendons of four muscles arising from the scapula that merge to form a broad ligament and insert on or near the head of the humerus, allowing rotation of the upper arm. Three of the muscles originate on the posterior surface of the scapula. These are the supraspinatus, infraspinatus, and teres minor muscles. Coming from the anterior surface of the scapula is the subscapularis muscle.

The term rotator cuff disease encompasses a wide range of conditions that include injury, inflammation, and degeneration. The usual cause is repetitive strenuous and abrupt shoulder motion, especially with an overhead component requiring a strong hand grip [3]. It is the tendon of the broad supraspinatus muscle that is most often affected. Notorious is the partial or complete tear of the rotator cuff of professional baseball pitchers. Injury also occurs in the aged when tears may be asymptomatic and nontraumatic (Fig. 9.4).

The patient can abduct the upper arm to near horizontal (a deltoid muscle function) but will experience increased pain and sometimes a sensation of grating on raising the upper arm above this level (an action requiring muscles attached to the scapula). Pain may be experienced in the area of deltoid insertion as the arm moves overhead. Tenderness, if present in the rotator cuff syndrome, is just below the tip of the acromion.

The "drop arm test" is a reliable indicator of rotator cuff disease [4]. The arm is first raised to full abduction. The patient then slowly lowers the arm. The test is positive if the patient is unable to maintain a smooth descent at about 90 degrees of abduction. At this point, the arm may suddenly fall.

Supraspinatus Tendonitis

Tendinitis of the supraspinatus tendon is a degenerative process complicated by calcium depositions. The condition is suggested when a patient keeps the arm close to the side and when all motion of the upper arm is limited by pain.

Biceps Tendonitis

Tendons of the biceps pass the humeral head to insert on the upper scapula (the coracoid process and the cusp of the glenoid fossa). Bicipital tendinitis is noted by tenderness at the bicipital groove. Pain occurs at this site on external rotation and abduction of the arm. The condition is often accompanied by rotator cuff disease (Fig. 9.5).

Adhesive Capsulitis

Fibrosis of the glenoid capsule can severely restrict mobility of the shoulder in all planes in both active and passive range of motion [5]. External rotation of the upper arm is particularly painful. Known as the "frozen shoulder," the patient keeps the arm tucked in to prevent pain.

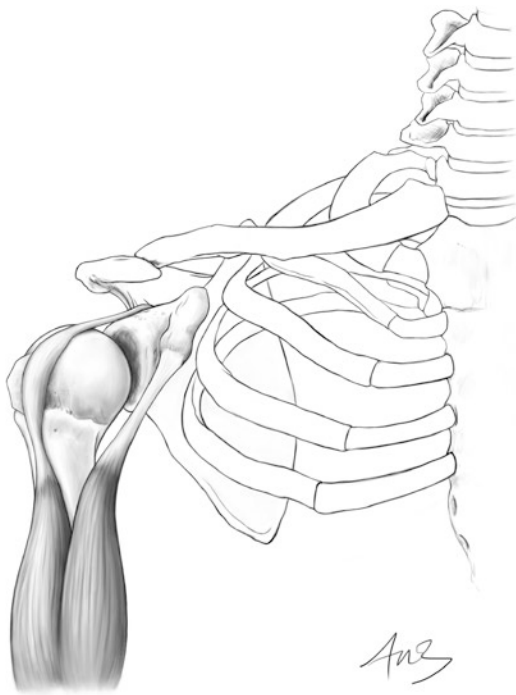

Fig. 9.5 Biceps tendinitis

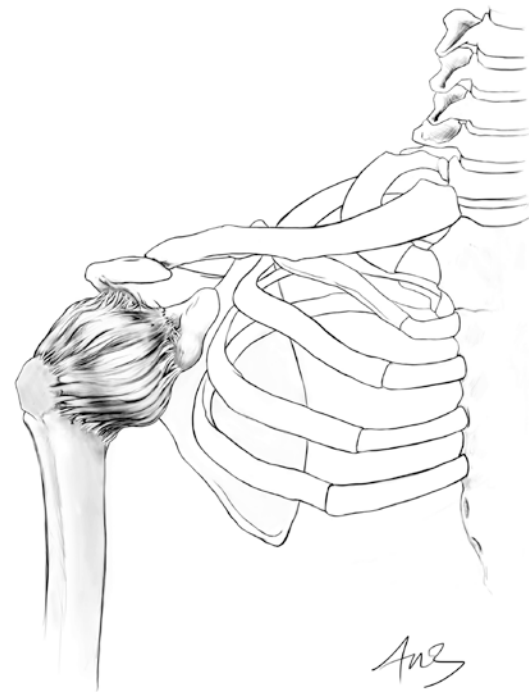

Fig. 9.6 Adhesive capsulitis

This protective carriage actually promotes further immobility. In all cases, the capsule around the glenohumeral joint becomes thickened and tight. Synovial fluid is reduced (Fig. 9.6).

The causes of adhesive capsulitis are usually quite evident: prolonged immobility following an arm fraction or regional surgery. There are also enigmatic associations in which diabetes accounts for the large majority. Disorders of the thyroid gland and Parkinson's disease are also implicated in some cases.

Osteoarthritis

The wear and tear of aging results in degenerative changes in cartilage that disrupt the smooth articular surfaces between the humoral head and the glenoid fossa. Fracture or dislocation of the shoulder can also produce posttraumatic arthritis. Crepitus – clicking or creaking on motion – is a notable feature.

Acromioclavicular Disease

Pain and tenderness on top of the shoulder from stretching or tearing of ligaments may be incurred

where the clavicle joins the acromial process. Violent collision injuries may be responsible as well as degenerative changes from osteoarthritis. Typical of acromioclavicular disease is pain in the superior shoulder region when the arm reaches across the chest to touch the opposite shoulder.

Crystal Arthropathy

Acute gouty arthritis and calcium pyrophosphate deposition disease (pseudogout) are common in the shoulder joint, but they do occur. What distinguishes them is the rather sudden onset of pain that is excruciatingly severe and exacerbated by the slightest movement. The diagnosis is further suggested by a history of acute arthritis of either type in other more commonly affected joints.

Avascular Necrosis

The prolonged use of corticosteroid hormones and sickle cell disease are the most likely causes of avascular necrosis of any bony components of the shoulder. As there are no specific physical findings, the diagnosis is made intuitively and then backed up by an imaging procedure.

Septic Arthritis

Infection localized to the shoulder is rare and difficult to diagnose. Physical signs including fever are variable. Treatment, however, is urgent since a delayed diagnosis can result in serious complications. Infection localized within the shoulder joint should be suspect when a patient with an immune deficiency disorder or who self-injects drugs develops a febrile condition with regional pain and/or tenderness. Rheumatoid arthritis is also a predisposing condition. Reported cases without such predispositions are in infants and the very elderly [6].

Neoplasm

The head of the humerus and the scapula are uncommonly the site of primary or metastatic cancer. Indeed, prolonged pain or stiffness of the shoulder girdle from neoplasm can be attributed to a treatment-resistant "frozen shoulder" [7]. Sarcoma, malignant fibrous histiocytoma, and multiple myeloma are reported to be primary or metastatic tumors.

SLAP

An acronym for "superior labrum, anterior to posterior," SLAP refers to an injury of the labrum (Latin for lip) [8]. The labrum is a strong, fibrous band that forms a rim around the glenoid fossa. It is an anchoring band composed of a proximal tendon of the biceps muscle superiorly and a tendon of the triceps muscle inferiorly. Competitive athletes who throw or who lift weights are particularly susceptible to a tear here. Physical signs of SLAP are tenderness over the origin of the biceps muscle and pain in the area of lifting overhead. Persons with a tear in the labrum are familiar with hearing a pop or click or feeling a grinding or catching when they raise the arm.

Motor Function

Weakness

Central Nervous System

The "pronator drift" sign is a sensitive and reproducible test for strength of muscles in the upper arm. Normally, the outstretched arms can be held with palms up for at least 10 s. Weakness is demonstrated by a slow rotation toward palms down within the time [9]. The drift will usually start when the thumb begins to turn down (Fig. 9.7).

Muscles that support supination are somewhat weaker than those supporting pronation, thus

Fig. 9.7 Pronator drift

accounting for the hand rotation of the fully abducted arms. The test simply establishes that it is easier to hold the arms out with the palms facing down. Because the pronator drift test requires activation of both cerebral hemispheres, demonstration of premature pronation indicates a central neurological abnormality.

Interruption of the pyramidal (or corticospinal) tract from cerebral cortex to motor effectors, when it involves the arm, tends to weaken the extensor muscles more than the flexors. Subsequent paralysis or weakness from such a lesion typically leads to holding the upper arm in a somewhat flexed position. In addition, there is resistance to passive motion as well as exaggerated deep tendon reflexes of the biceps and triceps. Muscle atrophy tends not to occur in the territory of the pyramidal tract in contradistinction from that of peripheral nerve or primary muscle disease. A lesion confined to the pyramidal tract has no associated sensory defect. Details of this pure motor lesion are provided in Chap. 11.

Peripheral Nervous System

The causes of peripheral neuropathies in the upper arm and shoulder are many. Overuse (from occupational tasks or athletics, such as tennis and pitching) is common. Arthritis occurs in the glenohumeral joint (especially in rheumatoid arthritis) and in the acromial-clavicular joint (usually from osteoarthritis). Subacromial bursitis and tendinitis, apical lung cancer, dermatomyositis, polymyositis, and vasculitis come up in the differential diagnosis. Dislocation of the humeral head may cause injury to any of the regional nerves.

Lesions of the peripheral nerves in the upper limb are readily detected in the hand, as already covered. Loss of the peripheral motor innervation causes weakness in addition to atrophy of muscles, disturbing the natural "balance" of flexors and extensors and resulting in deformities. Specific nerves causing them are readily identified. See Chap. 3.

More proximal lesions of the ulnar nerve may come from vertebral compression in cervical spine disease and from thoracic outlet restriction. Distal pressure on the palm, as it may come from long-sustained grip on an oar or power tool, can cause ulnar nerve injury with motor but no sensory impairment.

Complex Regional Pain Syndrome

A painful condition, long known as "reflex sympathetic dystrophy", is associated with inflammation and disturbed autonomic functions. The syndrome rarely follows physical injury to the neck or shoulder. It results in weakness and muscle atrophy in the upper arm, forearm, and hand. Initially, the inflammation causes increased warmth and moisture in the affected limb along with swelling and erythema. As the condition progresses, however, cooling, pallor, and decreased sweating occur when autonomic dysfunctions dominate. With the pathogenesis of reflex sympathetic dystrophy obscured, this condition is now known by the updated term: "complex regional pain syndrome" [10].

Sensory Function

Central Nervous Functions

Compression or inflammation of the spinal cord can produce neurogenic pain in the upper arm. Deep tendon reflexes are helpful in localizing the lesion. These are actually superficial reflexes that carry stimuli from the acutely stretched tendon to the cord and then to the flexor muscles. A reduced or absent DTR reveals the cervical location of cord pathology:

C5 – biceps deep tendon reflex
C6 – brachioradialis deep tendon reflex
C7 – triceps deep tendon reflex

Peripheral Nervous Function

Sensory defects in the upper arm and shoulder may involve the ulnar, medial, or radial nerves. A fracture or dislocation of the humerus can cause such injury as can persistent pressure in the axilla from crutch. The distribution of these nerves and patterns of anesthesia have been described in Chap. 3.

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Neck

10

Essentials

Mobility
Trachea
Thyroid
Lymph nodes
Salivary glands

Mobility

Examination of the neck begins with testing for mobility. The full range of motion is quickly performed, including checking for neck flexion. If habitually observed in the routine examination, chances are that this critical sign of meningitis in the febrile patient will not be overlooked.

To evaluate flexion, extension, and lateral rotation of the neck, the examiner should add slight resistance against each movement with a fingertip on the patient's chin. Going through the full range of motion is likely to bring out any significant cervical pathology that may involve the disks, joints, ligaments, muscle, bone, or innervation. Any directional limitation of the neck will often be associated with pain and/or weakness.

Flexion of the neck requires a consortium of muscle action, most prominently the sternocleidomastoid muscles. The trapezoid muscles extend the neck. The same muscles provide the ability to shrug the shoulder, innervated by the accessory cranial nerve. Lateral rotation enlists the sternocleidomastoid muscle on the side contralateral to the direction of turning. This range of motion of the neck, if normal, can be completed in less than a minute.

Limited Range of Motion/Pain/Weakness

Meningeal Disease

First in importance: any patient with fever or history of recent fever or suspected infection dictates testing of flexion of the neck! Without checking for meningeal inflammation, the diffuse symptoms of meningitis can easily be passed off as "a touch of flu." In the intensity of completing a complex evaluation of the febrile patient with influenza-like symptoms, neck flexion may be forgotten if not performed at the outset.

The term "nuchal rigidity" is often applied to the physical signs of meningitis. Lateral motion of the neck in meningitis, however, is often normal, while flexion is stiff and painful. Thus, a preferred way of referring to the meningeal sign is to record "chin to chest." Keep in mind, too, that persons with major immunological defects and the elderly may have severe infections including meningitis without ever developing fever. Also, there are other causes of pain and stiffness on neck flexion, for example, subarachnoid hemorrhage as well as chronic osteoarthritic conditions.

R.E. Phillips, *The Physical Exam*, https://doi.org/10.1007/978-3-319-63847-8_10

Muscular-Skeletal Disease

The most objective clinical sign of neck disease is limitation of motion, although pain and/or weakness are usually associated. Together, they are among the most familiar symptoms in the general population. To some degree, they are virtually universal in the aged population.

Disorders of the cervical spine tend to restrict motion in all directions (in contradistinction to irritation of the meninges that produces restriction mainly on flexion of the neck). Causes of stiffness and limited range of neck movement are numerous: inflammation (rheumatoid diseases), diseases of the bone (including spurs), abnormal postural strain, and acute or recurring trauma (whiplash, repetitive actions, kyphoscoliosis, lordosis). Osteomyelitis, epidural abscess, and neoplasm should also be considered when the cause of neck stiffness and pain are not readily identified.

Neuropathy

Neurological complications of neck disorders are evaluated by distribution of symptoms and findings. They may involve motor, sensory and/or tendon reflex functions. Neuropathies from forward herniation of a cervical disk are most likely to involve cervical roots 5, 6, and/or 7. When herniation is posterior, however, there is an increased chance of injury to the spinal cord, resulting in upper motor neuron disease (spastic paralysis) and sensory disturbances in all the limbs.

Distal manifestations of cervical lesions may involve the medial, ulnar, or radial nerves, neuropathies described in the Chap. 3. They also cause damage to the nerve roots and to the muscles that are detected in the neck and shoulder. The sequelae of cervical disease may be unilateral or may affect both arms.

In an oversimplified version, the telltale signs of specific nerve root involvement are:

Cervical Disc Lesion

C5: Motor. Weakened deltoid and supraspinatus muscles, limiting abduction power of the upper arm
Sensory. Pain or neuropathic reduced sensations along the shoulder and lateral area of the upper arm
Reflex. Dampened deep tendon reflex of the biceps

C6: Motor. Reduced flexion power of the upper arm from a weakened biceps muscle
Sensory. Accompanying pain or hypesthesias over the lateral forearm to the thumb and index finger
Reflex. Depressed deep tendon reflex of the brachioradialis muscle

C7: Motor. Compromised straightening of the arm by extension of the upper arm owning to weakness of the triceps muscle
Sensory. Disturbance of sensations in the posterior upper arm and the dorsal forearm
Reflex. Diminished triceps deep muscle tendon jerk

The above criteria must be considered somewhat imprecise owing to the overlapping of neural territories. Also some muscles of the neck have bilateral cortical innervation. Where restricted cervical mobility is not associated with pain or a sensory defect, the clinician should consider the diagnosis of a pure motor neuron disease such as amyotrophic lateral sclerosis and multiple sclerosis. Weakness of neck flexion is common in patients with both conditions.

Trachea

Palpate the trachea at the sternal notch. A rapid technique is to simply place the tips of the third and fourth fingers at the sternal notch so that the trachea is between them. The normal position of the trachea is precisely mid-center. This test will assuredly be normal on almost every routine examination. Because, however, it can be performed in a few seconds and because an abnormality of major significance might be discovered, habitually checking the trachea for position is included here as an integral part of the routine physical examination. Furthermore, once having gained extensive experience in judging the normal alignment of the trachea, the clinician will have confidence of recognizing even the slightest deviation from midline (Fig. 10.1).

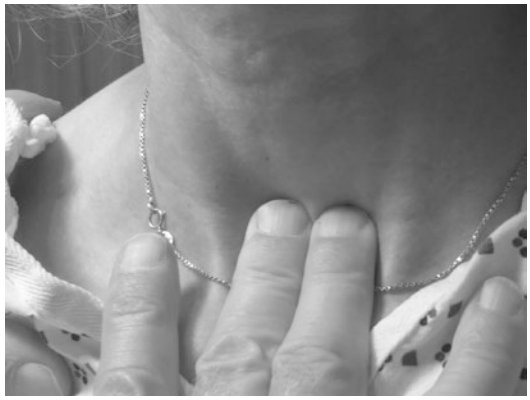

Fig. 10.1 Trachea, palpation for midline

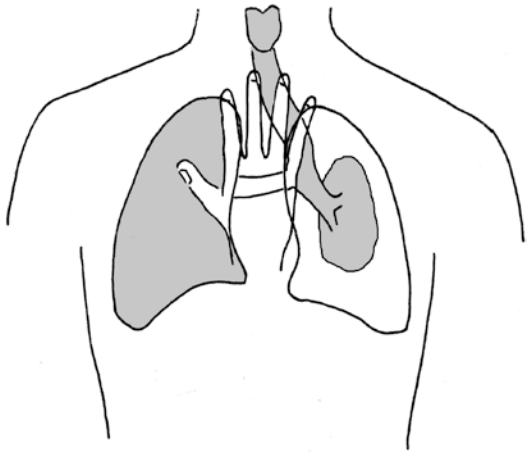

Fig. 10.2 Trachea, deviated

Even a slight deviation of the trachea from midline may reflect a prominent scoliosis or rib cage malformation. More important, it may be the result of a shift of the mediastinum due to a mass lesion, tension pneumothorax, a substernal goiter, or major atelectasis. Bear in mind that a major large shift of the mediastinum to one side will produce a very small deviation of the uppermost trachea from its midline position (Fig. 10.2).

Thyroid

Normally, the thyroid gland is so soft that it is perceptible by palpation only in the most sensitive hands. The isthmus may be felt with light palpation immediately after locating the upper trachea, as described above. The fingertips can then "crawl" from the center laterally to examine each lobe for size, texture, and nodules.

Enlargement

If diffusely enlarged or if a lump is found, observe and feel the gland while the patient swallows. Only if an abnormality is found (or suspected) does the examiner need take the time to walk behind the patient and palpate the gland from another vantage point. (Remember, the hospital bed may have been raised so that walking around should not be done unless another person stands directly in front of the patient or unless the examiner can keep one hand firmly on the patient's shoulder as he or she moves to the back.) Palpation from behind is done with both hands simultaneously. Swallowing a sip of water can help expose the thyroid isthmus to palpation.

Goiter is a general term referring to enlargement of the thyroid gland that may be diffuse or multinodular. It occurs in states of both over- and underactivity. There are many causes of both. Sometimes, the gland is grossly enlarged yet produces sufficient and appropriate thyroid hormone.

Thyroiditis

Autoimmune damage to the thyroid gland is the most common cause of goiter in the developed world. Called "Hashimoto's thyroiditis", the gland enlarges from increased release of thyroid-stimulating hormone (TSH) from the pituitary gland, initially producing hyperthyroidism. Eventually, however, there is enough atrophy of thyroxin-producing cells that occur to cause hypothyroidism; it is the most common cause of hypothyroidism [1].

Thyrotoxicosis, Autoimmune

Also having an autoimmune pathogenesis, Graves' disease differs from Hashimoto's in causing an increase in the synthesis and release of thyroid hormone. It is the most common cause of thyrotoxicosis [2]. Ocular manifestations, in particular proptosis from postorbital fat infiltration, lid lag, and lid retraction, frequently occur

in Graves' disease. In iodine-poor areas of the world, the fortification of salt with iodine can precipitate Graves' disease. Lithium and amiodarone (an iodine-containing drug) can also induce hyperthyroidism.

Hypothyroidism

Lack of iodine is the classical form of goiter worldwide. Nutritional aberrations, such as an extreme soy or peanut diet, can cause iodine depravation and with it goiter. The enlarged thyroid gland may or may not be able to synthesize enough thyroxin to maintain normal function. Viral thyroiditis, enzymatic defects of thyroid hormone metabolism or reception sensitivity, and infiltrative diseases such as sarcoidosis and cancer are additional causes of goiter.

Texture

A diffuse goiter may feel firm and rubbery throughout. This texture is typical of Hashimoto thyroiditis. Graves' disease has a soft texture. The nodular form of goiter has a granular feeling. On the other hand, a hard texture of tissue anywhere in the gland – whether localized or diffuse – is more in keeping with thyroid cancer. Goiter may be large enough to impair breathing and swallowing, particular if there is a retrosternal component.

Vascular Sounds

The high blood flow of hyperthyroidism is confirmed by auscultation. A bruit, if present, is heard throughout systole and diastole. The sound is not to be confused with the transmitted systolic sound of aortic valve stenosis or carotid artery stenosis. If a bruit is also pulsatile, an aneurysm or kink in the carotid arterial complex is suspect. A continuous "venous hum" heard just below the right clavicle varies with changes in the neck and body position and with compression of the jugular veins; it is a normal finding that is prevalent in children.

Nodule

Single or multiple adenomas in the thyroid gland may produce hormones that function independent of normal, physiological feedback. Called "Plummer's disease," these toxic adenomas produce symptoms and signs of hyperthyroidism.

"Silent" nodules – that is, hormonally inactive lumps in the thyroid – can be benign or neoplastic. Most benign thyroid nodules remain unchanged in size over long period. A thyroid nodule that is firm and fixed in position suggests a malignant etiology. Hoarseness and dysphagia of recent onset are corroborating findings. Exposure of the head or neck to irradiation is an addition warning sign. An enlarged cervical lymph node neighboring a thyroid nodule suggests a metastatic malignancy [3].

Many nodules detected in the thyroid area are non-thyroid tissue. These include metastatic tumors, infiltrative diseases (such are sarcoidosis and hemochromatosis), lipomas, parathyroid adenomas or cysts, and paragangliomas.

Lymph Nodes/Salivary Glands

These structures in the head and neck are considered together since they are palpated in a continuum. Run the tips of the fingers up the anterior cervical lymphatic chain in a crawling motion from the lower aspect upward toward the angle of the jaw and then forward to the submandibular and submental areas palpating for adenopathy or other masses. With a similar touch for detecting lymph nodes, palpate up the medial neck then down the posterior neck and finally over the supraclavicular areas. Here is another example where both hands can be efficiently used simultaneously. This full sequence, rapidly performed, should become habitual in the routine physical examination. Certainly, proceeding in a standardized progression reduces the possibility of missing a palpable abnormality while it facilitates remembering where it was. Routinely palpating for pre- and postauricular nodes is not deemed necessary unless there are symptoms suggesting the possibility of mastoiditis or parotitis.

Lymph Nodes

The great majority of lymph nodes are in the thorax and abdomen. Those most easily accessible ones, however, are in the neck. Meticulous palpation for lymphadenopathy here is particularly important for evaluating a patient with fever or

unexplained weight loss. It is appropriate to include an overview of the subject of lymphadenopathy at this point in the text.

The clinician's challenge on palpating lymph nodes is sorting out benign lesions from serious ones. By "benign" means adenopathy will resolve spontaneously or after appropriate therapy. Serious lesions refer to those associated with conditions that are chronic or neoplastic. There are some clues to help with this differential, but they must be considered in a larger context of being helpful, not diagnostic.

Benign Lymph Nodes

The vast majority of enlarged lymph nodes in the neck are entirely benign. They are, of course, a defensive response to a wide variety of infections. Findings that suggest a benign cause are (1) tenderness, (2) freely mobile, (3) less than 2 cm in greatest dimension, and (4) localized to one region of the neck. Furthermore, the chances of enlarged lymph nodes being benign are when they are associated with a sore throat (whether a viral or streptococcal origin) and if the patient is a child or young adult. Prominent lymph nodes in the posterior neck together with palatal petechiae and a palpable spleen are highly suggestive of infectious mononucleosis.

Neoplastic Lymph Nodes

One sign that a palpable cervical lymph node – singular or in clumps – has a serious implication is the texture: if it is rocklike hard, the node is consistent with metastatic cancer. A firm, rubbery texture (similar to the eraser at one end of a pencil) is more typical of lymphoma. The presence of palpable lymph nodes throughout the neck increases the possibility of lymphoma. Young children who commonly harbor many small cervical lymph nodes are perhaps exceptions.

A lymph node in the neck that is greater than 2 cm in its longest diameter deserves further evaluation for possible infiltrative, infectious, or neoplastic origin. A lymph node that appears fixed to the underlying tissue suggests malignancy. Usually a neoplastic lymph node is not tender, but necrosis or hemorrhage may produce tenderness. Chances of cervical lymph nodes being malignant rise sharply in the older population. This concern is redoubled in the patient who smokes.

A palpable supraclavicular lymph node (just behind and above the clavicle) is uncommon, and it merits special attention owing to its strategic site. Nodes here drain from the arm and thorax. On the left, they also receive lymph from the abdomen and pelvis. A metastasis from any of these regions to a supraclavicular lymph node has given it the name "sentinel node." Sometimes, it is the first sign of a malignancy. The supraclavicular lymph node is more easily felt by having the patient perform Valsalva's maneuver during palpation.

Granulomatous Lymph Nodes

When lymph nodes feel matted together, a chronic inflammatory disease (such as tuberculosis or sarcoidosis) may be responsible. A historical term for localized tuberculosis in the neck is "scrofula"; it was common in children during the "Industrial Age." The infection can extend into the skin and form a chronic, draining sinus. It is a diagnosis to contemplate in any significant cervical adenopathy that is persistent, especially if the patient is from a country where tuberculosis is quite common [4].

Generalized Lymph Nodes

Lymph nodes found in the axilla, groin, or elbow in addition to cervical lymphadenopathy imply a systemic disorder. The causes vary from infectious mononucleosis, HIV, and syphilis to sarcoidosis and connective tissue diseases. Coinfection of tuberculosis and HIV can cause widespread lymphadenopathy. Lymphoma may present with extensive cervical, axillary, and inguinal lymphadenopathy.

Lymph Nodes with Ulceration

A lymph node infection that tends toward ulceration is from the bacteria *Francisella tularensis* [5]. Tularemia (also known as "rabbit fever") is more like to affect "outdoors" people. The organism is largely harbored in the rodent population. Infection comes by way of fly or tick vector, by inhalation, and by direct contact with contaminated water or surfaces.

Lymph Node Mimics

Several lumps in the neck region can simulate a lymph node. Such benign lesions include an

enlarged salivary gland and a cyst lesion. Cysts can be of thyroglossal duct or sebaceous or branchial cleft origin. The thyroglossal duct cyst occurs close to midline and is usually discovered in childhood [6]. It moves on swallowing and rises on protruding the tongue. At an older age, its discovery may depend upon an attention-gaining infection that causes the cyst to become tender and warm. The branchial cleft cyst is a congenital remnant of the apparatus that enables fish to develop gills [7]. The cyst, which is smooth, not tender, and fluctuant appears on the lateral neck. As a persisting indolent cyst, it has the potential of becoming infected and creating a draining cutaneous sinus or fistula. An anomalous configuration of a cervical rub or a synovial cyst of rheumatoid arthritis on it can be mistaken for a supraclavicular lymph node.

Salivary Glands

Most pathology of the salivary glands involves parotid and submandibular glands. Significant enlargement of either usually renders them palpable. On finding a mass within the mouth, palpating it with a gloved finger requires a special precaution. Roll up a facial tissue or washcloth, and place at the angle of the jaw to prevent the patient from closing the mouth. The clinician is reminded that the masseter muscle, gram for gram, is the strongest muscle in the body.

Ductal Obstruction

Plugging of the salivary glands by mucus or calculi causes them to swell and become tender. Eating can become painful.

Inflammation

Ipsilateral sialadenitis occurs from obstruction of the salivary ducts and from dental or other oropharyngeal infections. *Staphylococcus aureus* is a common invasive organism.

Characteristically, mumps causes swelling of parotid glands bilaterally along with cervical lymphadenopathy. The clinician may not think of mumps when parotitis is unilateral and in an adult. Drugs, including propylthiouracil and iodine compounds, are also etiologically culpable.

Granuloma

Palpable salivary glands can commonly occur in widespread tuberculosis and sarcoidosis. They may also complicate chronic sicca (Sjögren's syndrome).

Neoplasm

Tumors arising from the salivary glands can represent primary or metastatic cancer. Most tumors are in the parotid gland and most of these are benign [8]. Malignant tumors can occur in any of the salivary glands, including the smaller glands of the sublingual area. In many cases, the salivary neoplasms are not reported by the patient, only to be discovered as an incidental finding when the cancer is far advanced. Certainly, the clinician is prudent to practice looking beneath the tongue in the periodic physical examination.

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Face

11

Essentials
Shape
Symmetry
Color
Pigmentation
Texture
Hair
Motion
Sensation

A glance at the face described at the outset of these guidelines was meant to give an impression of the patient's general well-being and attentiveness. A more studied approach is presented here.

Observe the face for general shape, distribution of hair (including eyebrows), and obvious scalp problems. Although sometimes taught as a beginning of the top-to-toe physical examination, ruffling through the hair to feel for bumps is probably not an efficient use of time unless there are relevant symptoms or there has been recent head trauma; it may leave the patient with abdominal pain a bit baffled.

Shape

Much has been made of attributing the shape of one's face to personality. Considered in a nutshell are round (emotional), square (analytical and fiery), oblong (practical), and triangular (artistic). These interesting, folk generalizations, however, have no place in medical diagnostics. Here, the cardinal observations of the face are presented as "full" and "thin." A commonly used term to describe the head – "normocephalic" – is too indefinite to be meaningful.

Full Face

Edema

Gravity has an important role in the body fluid distribution. Edema of the normal face after an overnight sleep is subtle but accounts for *morning puffiness* (particularly in the loose tissue beneath the eyes) as well as the timbre of the "morning voice." Normally, these disappear rapidly with upright activity and gravitation-dependent redistribution of interstitial fluid. On the other hand, facial edema from severe *hypoalbuminemia*, as in the nephrotic syndrome or protein deprivation, may persist throughout the day. Swelling in renal diseases is most noticeable beneath the eyes where the venous-rich tissue is loosely matted. Edema from myocardial failure, by contrast, is more gravity-affected; that is, it principally affects the position-dependent lower body during upright activity.

Obesity

The facial distribution of adipose tissue in obesity is principally in the cheeks and below the

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chin. The upper half, including the temples, is relatively spared, differing from the typical "moon facies" of Cushing's syndrome as described below.

Trauma

On examining a patient after head trauma, the skull must be palpated carefully. Feel for possible consequences of a subgaleal hematoma, subcutaneous crepitus, or depressed fracture. Otherwise, taking time to palpate the head is time probably not well invested.

Osteitis Deformans

Noting an abnormally enlarged head should bring to mind the possibility of Paget's disease of bone (osteitis deformans), especially when the patient realizes that his or her hat is "getting too small." The enlargement is most prominent in the forehead. This condition – overactive remodeling of bone with excessive osteoclastic function and excessive osteoblastic response – produces bigger and weaker bones. The affected skull may have corrugated ridges, the forehead enlarged, and the facial bones misshaped, all symmetrical or not. In some patients with Paget's disease of bone, the frontal bone is so prominent, that the head has the shape of a triangle standing on end. Bony overgrowth at the base of the skull crowds emerging neurological pathways that can result in reduced hearing and vision. A common corroborative finding is the anterior bowing of the tibiae.

Endocrinopathy

Three unusual forms of facial fullness are mentioned here: acromegaly, myxedema, and Cushing's syndrome. Each is a systemic disease with insidious onset so that family and friends – and even periodic healthcare providers – may simply ascribe typical changes from endocrine diseases to "aging." Here, comparison with old photo portraits is particularly helpful.

Furthermore, comorbid conditions are often associated with each of these three syndromes: hypertension, diabetes type 2, and obesity. These conditions are so prevalent in the general population that they may be overlooked as part of a syndrome. Yet, each has a serious underlying cause in which there are many corroborating findings, findings awaiting the perspicacity of the "fresh" examiner.

1. *Acromegaly*. Bony overgrowth is a characteristic feature of acromegaly with two notable areas in the face: (a) an enlarged mandible causing a serious underbite and (b) thickening of the frontal bone resulting in prominent supraorbital ridges. In addition, the facial skin appears coarse and oily, the nose large and fleshy.
2. *Myxedema*. The face in myxedema is typically puffy, particularly around the eyes. The thickened skin is non-pitting in contrast to other forms edema. It has a dough-like consistency, is excessively dry and rough, and may have a pale, yellowish hue. The skin feels cold owing to reduced blood flow. The hair is coarse and brittle. It is diffusely sparse, most tellingly in the lateral eyebrows. An apathetic demeanor along with a husky voice further supports the diagnosis. Whether or not the thyroid gland is enlarged depends upon the primary cause of myxedema, that is, of pituitary, thyroid, or nutritional origin.
3. *Cushing's syndrome*. Prominent facial fullness with a ruddy complexion in the presence of hypertension and diabetes merits consideration of Cushing's syndrome. Cortisol-induced fat distribution favors the cheeks and the temporal fossa, causing a more round appearance, the so-called moon facies that is characteristic of the syndrome.

It is speculated that the rounded face is from deposition of "brown fat" that fills out the temporal area of the face and is distinctly different than the distribution of adipocytes in obesity. Metabolism of brown adipose tissue yields more heat than does ordinary white fat; it is more abundant in infants and in mammals that hibernate. "Browning of Subcutaneous White Adipose Tissue in Humans after Severe Adrenergic Stress" has been demonstrated in severely burned children [1]. Circumstantial evidence, then, suggests that the distribution of brown fat is largely controlled by corticosteroid activity.

An additional facial characteristic of Cushing's syndrome is the flush of vascular plethora, owing to thinning of the skin. Acneiform lesions in patients well beyond the adolescent years are often present as well. Hair tends to grow in an abnormal distribution, particularly over the forehead and the upper cheeks. These signs of virilization reflect the underlying cause of the syndrome: hyperactivity of the pituitary gland that induces not only overproduction of cortisol by the adrenal gland but also increases androgenic hormones.

Generalize weakness, common in Cushing's syndrome, may be erroneously attributed to being "out of shape." The actual causes – musculopathy and/or hypokalemia – are common features of the disease.

Thin Face

Weight Loss

There is something unsettling about the face of the seriously ill patient, although there are rarely identifying signs of the cause. Weight loss may be evident in the hollowed cheeks, pinched nose, and sunken eyes. The neck shares the emaciated appearance. The coincidental finding of pallor, cyanosis, or jaundice provides strong diagnostic clues. Scleral icterus coupled with loss of facial fullness, for example, may reveal painless biliary obstruction from pancreatic carcinoma. Severe emaciation of anorexia nervosa presents no identifying physical signs, although lowered blood pressure, heart rate, and core temperature are commonly associated.

The causes of thinning of the face encompass the entire spectrum of vital organ failure: circulatory, metabolic, hypoxic, neoplastic, infectious, nutritional, and inflammatory. Allowances must be made for the tissue-losing normalcy of aging.

Symmetry

Symmetry of the face is quickly determined. Look for the palpebral fissures, the nasolabial folds, and the corners of the mouth. They provide readily spotted clues of facial pathology and may have been detected in the first impression.

Palpebral Fissures

Normally, the margins of the upper lids just barely overlap the uppermost part of the iris.

Even a slight degree of difference, if unilateral, will disrupt the obvious symmetry of the palpebral fissures.

Retraction of the eyelids is evident when the sclera is visible above the iris. Most instances involve elevation of the upper eyelid and are associated with Graves' disease [2]. The underlying pathology is an autoantibody response, causing chronic inflammation of extraocular muscles and accumulation of retro-orbital mucopolysaccharides. The subject, ophthalmic complications of thyroid disease, is expanded in Chap. 12.

Ptosis ("fall" in Greek) indicates that the upper lid droops enough to cover a large area of the iris. Ptosis has many causes: infectious, neurologic, connective tissue, trauma, and vascular. Paralysis of the oculomotor nerve that supplies the levator palpebrae superioris muscle will result in ptosis. Since this cranial nerve has a parasympathetic action, the pupil on that side will likely be dilated (mydriasis). A second and subdominant muscle (Müeller's muscle) contributes to lid opening and is under control of the sympathetic nervous system. A disorder of this system, as in Horner's syndrome, will cause mild ptosis and here the pupil is constricted (miosis).

If ptosis is bilateral, the lesion may be in the central nervous system, specifically in the nucleus of the oculomotor nerve. Myasthenia gravis will cause bilateral ptosis but at the neuromuscular junction; the abnormality is one of the earliest signs of this condition. An acute ptosis-inducing disorder is botulism when diplopia, facial weakness, and dysphagia will probably also be present. These serious problems of eyelid control considered the most common cause of ptosis is relatively benign: the droopy eyelids of the very old.

Nasolabial Fold

Flattening of the nasolabial fold on one side reflects denervation of the maxillary branch of the facial nerve.

Corners of the Mouth

A motor defect of the mandibular branch of the facial nerve will allow the stronger muscle on the contralateral side to drawn toward the mouth toward that normal side. This finding is exaggerated on smiling or showing the teeth. For further description of disorders affecting these three regions, refer to "Motor" in this chapter.

Color

Discoloration in reference to the face refers to a global distribution. Abnormalities of color in the face that are localized are covered under the subheading RASH. The causes of an abnormal hue of the complexion are either (1) disturbances in vasomotor control, namely, an imbalance of vascular neural control of cutaneous blood vessels, or (2) quantitative abnormalities of the red blood cell mass. These abnormalities produce either erythema or pallor, their appearance modified by natural skin pigmentation and thickness.

Check facial skin for color, including erythema (blush) or inflammatory discolorations. Some patients may have cyanosis or pallor that is more easily detected in the lips than in the palms, nail beds, or conjunctival sac.

Erythema

Generalized vasodilation of the cutaneous blood vessels in the face causes bright red erythema. The discoloration vanishes on slight external pressure but returns instantly on release.

Vasodilation

The blush is a familiar and benign, momentary vasodilation from emotional stimuli, sudden exertion, alcohol, spice ingestion, or heat exposure (such as a hot beverage or ambience). The trigger point for blushing varies greatly among individuals.

Intermittent or persistent flushing in the face occurs with various forms of vascular instability, including states of falling estrogen production, thyrotoxicosis, and niacin therapy. Rare forms of facial flushing are the carcinoid syndrome in which excessive serotonin released from a splanchnic neoplasm induces generalized vasodilation. There are also individuals who have very red faces and who have no documentable vasomotor disorders.

Solar injury (or, more specifically, overexposure to ultraviolet light) to the skin causes sunburn. Telltale signs are revealed by lines of clothing. Aside from the bright red appearance, the skin is painful and edematous. The development of blistering transforms the burn from first to second degree.

Overdose from an atropine-like agent causes overheating from inhibited sweating and an increased heart rate. These changes in turn produce cutaneous vasodilation. Typical of atropine toxicity is a brightly reddened face, with dry skin and mucous membranes along with tachycardia and dilation of the pupils, all signs that the parasympathetic nervous system is inhibited.

Rosacea is a persistent flush over the cheeks from vasodilation. The nose and chin are often involved, as well. The ruddiness tends to highlight further when stimulated by spices, alcohol, and changes in temperature. Vasodilated areas are prone to develop telangiectasias and pustules. Rhinophyma is a hyperplastic complication of the nasal rosacea.

Blood Volume Excess

Polycythemia of sufficient degree will cast a deeply ruddy complexion to the face; the color may be mistaken for cyanosis. It is likely most noticeable in the lips but can be evident in the hands and feet and most especially the nail beds.

Obstruction of the Superior Vena Cava

A darkly reddish complexion of the face is a clue that the superior vena cava is obstructed from

thrombosis, mediastinal neoplasm, or fibrosis. In contradistinction to polycythemia, plethora in this syndrome is confined to the head. Distention of the veins in the neck and chest wall is corroborative evidence of the obstructed superior vena cava.

Carbon Monoxemia

The classical description of the "cherry-red" face from carbon monoxide toxicity occurs but not until the condition has developed into the advanced stages. Discoloration of the skin from lesser degrees of carbon monoxemia results in cyanosis from hypoxia. See below under section "Cyanosis."

Pallor

Pallor is not synonymous with anemia and is not an accurate indicator of mild anemia. Indeed, vascular tone contributes to skin color as well as the amount of circulating hemoglobin.

Vasoconstriction

There are individual variations of vascular beds for dissipating the heat of metabolism. These variations account for the warmth or coolness felt on shaking hands. The degree of vasomotor tone in the cutaneous bed is certainly a component of one's complexion.

Acute vasoconstriction induces pallor of facial skin under strong adrenergic stimulation. Emotional reactions, such as high anxiety, fear, or intense pain will induce facial pallor through this mechanism. Tobacco smokers have a persistent vasoconstriction that gives the face a pale, grayish appearance. A change in complexion is clearly noticeable in those who have recently quit smoking.

Vasoconstriction also occurs as part of the generalized compensatory mechanism in response to some compromise in the central or regional peripheral circulation. The cause might be a benign, presyncopal event (such as a hypotensive episode induced by hyperventilation). Sudden shifting of circulation by vasodilation in the viscera (as in one form of anaphylactic reaction) will produce pallor from a reactive cutaneous vasoconstriction. Vasoconstriction-induced pallor can be a reaction to an acute cardiac decompensation from myocardial ischemia or sudden-onset and severe tachycardia. Of course, this appearance will accompany any abrupt and profound peripheral vascular event such as serious blood loss or pulmonary embolism.

Decreased Blood Volume

Severe anemia will results in generalized facial pallor. Lesser degrees of anemia, however, are more easily observed in the tongue, palms, nail beds, conjunctival sac, or the lips. Of these sites, a comparative study concluded that pallor of the tongue was more accurate than other pallor sites [3]. Distinguishing mild anemia requires optimal lighting and a well-practiced eye; even then it is difficult to be sure [4].

Cyanosis

Hypoxia

Facial cyanosis, the result of hypoxia, is most easily observed in the lips and lobes of the ears. Often cyanosis is associated with a compensatory increased rate of breathing (hyperpnea), although it must be remembered a reduced rate and depth of respirations can also be responsible. Either way, cyanosis indicates hypoxic stress from any of a myriad of causes: central nervous system and pulmonary, cardiac, and peripheral vascular.

Vasoconstriction

The vasoconstriction of cold exposure, causing pallor initially, may result in cyanosis owing to slowly moving cutaneous blood. Swimming in cool water will often cause cyanotic lips in healthy persons. Paradoxical are the ruddy cheeks and lips of persons accustomed to intense cold.

Excessive Deoxyhemoglobin

Cyanosis occurs in severe polycythemia, even without tissue hypoxia. In addition, inherited hemoglobinopathies, such as sickle cell disease, are responsible although, usually, the pallor of associated anemia usually dominates.

Carbon Monoxidemia

Exposure to carbon monoxide displaces oxygen in hemoglobin, resulting in cyanosis. The mechanism is the affinity of carbon monoxide to hemoglobin, a factor of more than 200 times that for oxygen, and to myoglobin and cytochrome oxidase, a respiratory enzyme within the mitochondria. Keep in mind that it is only in the advanced stages of toxicity that the characteristic "cherry-red" appearance of carbon monoxidemia occurs. The most frequent source of this condition is the emergency use of heating units in improperly ventilated rooms.

Arteriovenous Fistulas

Anatomical shunts, such as arteriovenous fistulas, should be considered when more common causes of cyanosis have been reasonably excluded. Signs of "high cardiac output" may or may not be present. Places for careful palpation and auscultation for detecting an A-V fistula are over areas of surgery or injury, whether recent or remote.

Pigmentation

Melanin-containing cells in the epidermis release pigment on direct exposure to ultraviolet radiation. Tanning is the body's way of protecting the skin from prolonged exposure to harmful sunlight. It occurs only at those sites so exposed.

Increased Pigmentation

Hyperpigmentation: Diffuse

Diffuse hyperpigmentation occurs when the pituitary gland is stimulated to secrete melanin-stimulating hormone for a prolonged time. This results in the "summer tan that lasts into the winter" and involves areas not normally exposed to the sun.

Three rare but critically important causes of hyperpigmentation in the face and beyond should be considered: (1) hemochromatosis, (2) primary adrenal insufficiency, and (3) hyperpituitarism.

1. *Hemochromatosis* is the result of iron overload, either excessive inherited dietary absorption or acquired by excessive administration of iron over a long period. The face develops a brownish cast from deposits of iron in the skin. This discoloration is a cosmetic issue, whereas excessive iron deposited in the liver, heart, and pancreas causes reactive inflammation. When advanced and compromising insulin production, the condition is referred to as "bronzed diabetes."
2. *Adrenal insufficiency* typically produces the facial tan "that just won't go away." Confirmation of the abnormal pigmentation is found on mucus membranes and along the creases of the palms. Easy fatigue, low blood pressure with orthostatic light-headedness, and salt craving are frequently associated problems.

   Hyperpigmentation from adrenal insufficiency indicates primary failure of the adrenal gland. The pituitary gland responds by increasing its ACTH output and, concurrently, its release of melanin-stimulating hormone.
3. *Cushing's syndrome* from overproduction of ACTH and with it melanin-stimulating hormone commonly results in generalized hyperpigmentation. It is usually most noticeable in the face. Hyperpigmentation does not occur if excessive cortisol is from an adrenal adenoma or is iatrogenic.

Hyperpigmentation: Spotty

Freckles

Spotty hyperpigmentation is best known as freckles. These are tiny concentrations of melanin, most prominent on fair-skinned persons. In part genetic, their prominence correlates directly with exposure to sun, tending to fade by avoiding sunlight.

Liver Spots

Liver spots ("age spots" or lentigines), on the other hand, are confluences of localized hyperpigmentation. Larger than freckles, they also tend to occur in more uncovered areas and do not fade when sunlight is avoided. They are usually seen in the second half-century of life and seldom

before. Both freckles and liver spots are totally benign. There is a danger of misidentification, however, on assuming that a precancerous lesion is one of them.

Chloasma

During the second half of pregnancy, most women develop some degree of hyperpigmentation. Freckles enlarge and converge. Brown splotches, or chloasma, appear on the forehead, nose, cheeks, and upper lip in a symmetrical pattern. Known as the "mask of pregnancy," the darkened facial skin will usually fade within several months after childbirth.

Melasma

Melasma refers to tan to brown patches appearing on the forehead, cheeks, temples, and upper lip. The patches are generally symmetrical. The hyperpigmentation – generally induced by estrogen therapy and often occurring during pregnancy – is entirely benign.

Moles

Moles are dense concentrations of melanocytes. They may be benign or malignant or somewhere in between, termed "dysplastic."

1. *Benign moles*. Most adults have between 10 and 20 small moles, and these usually appeared in their teens or twenties. Moles are generally less than a ¼ inch in diameter and are round or oval with well-defined border. They vary in degrees of pigmentation, ranging from pale to black, but each maintains a homogeneous color. Some are flat-topped; others are raised or dome-shaped. A few can be pedunculated. Hair growing from within is occasionally present and is, in fact, reassuring of its benign nature (Fig. 11.1).

   Moles occur virtually anywhere on the body. They are more prevalent, however, in sun-exposed areas and are particularly common on the face. Once they appear, moles are lifelong and generally do not change in color

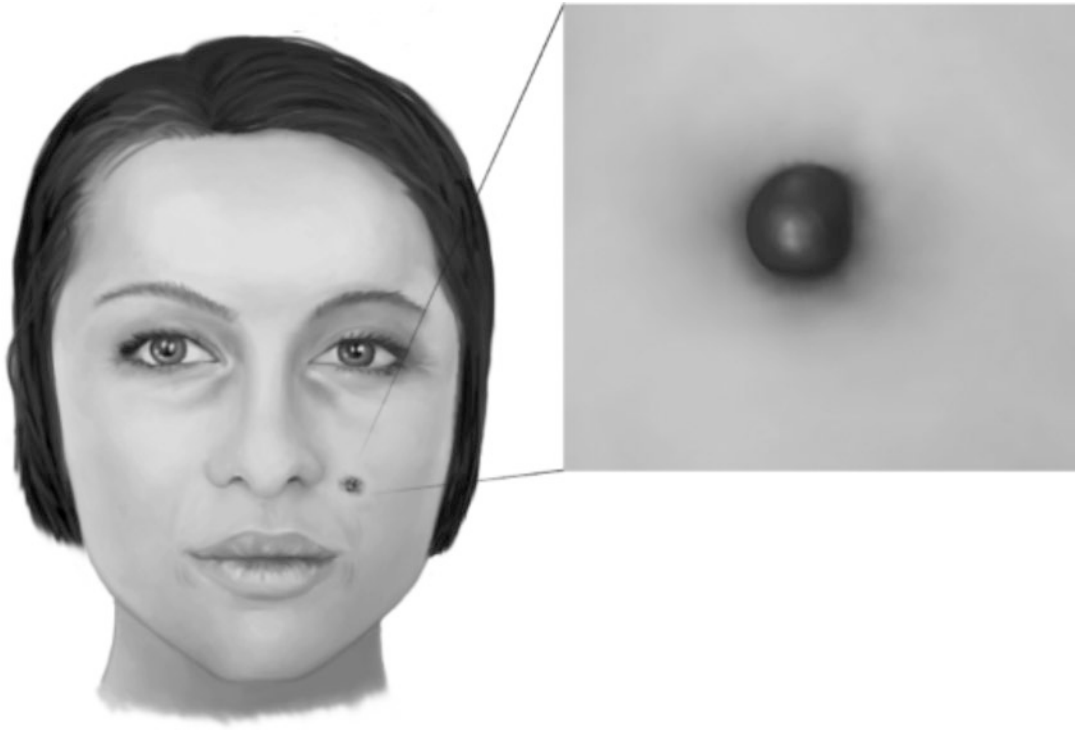

Fig. 11.1 Mole of face

or size. For reference to any suspicious mole, it is prudent to take a color photograph with a ruler alongside.

2. *Melanomas*. These lesions are a form of skin cancer. Most often, they arise in areas of chronic sun exposure, most commonly, of course, in the face.

   Some features of melanomas help distinguish them from benign moles, although the differentiation can be challenging. One feature is size: a pigmented lesion greater than half a centimeter in diameter is suspicious. A mole that is unlike all the others (the "ugly duckling") is highly suspect of a melanoma, as does any degree of growth: broadside or raised. A more advanced degree of malignancy is revealed when itching, pain, ulceration, or bleeding occurs within or around the mole (Fig. 11.2).

   The clinician, examining the face, must observe each mole for features of malignancy. The mantra for suspicious signs is referred to as the diagnostic "A B C D E rule":

   A. Asymmetry rather than roundness is dominant.
   B. Border is irregular.
   C. Color is quite variable throughout.
   D. Diameter is greater than 6 mm.
   E. Evolution in size or pigmentation.

   Of course, any mole with crusting and/or bleeding, however minor, is strong evidence of a malignant component.

3. *Dysplastic moles*, also known as "atypical moles" tend to be larger than benign moles; indeed, they can be huge. They exhibit transitional pathology between benign and malignant lesions. Pigment may be distributed unevenly in dysplastic moles, causing some variation in color. Borders that may be irregular, notched or scalloped can be somewhat indistinct. These characteristics stated, it is the dysplastic mole that tests the diagnostic acumen of the most experienced dermatologist.

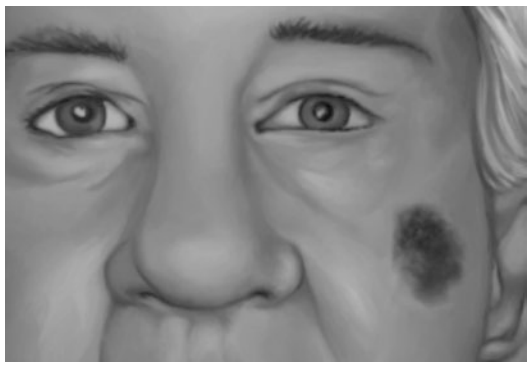

Fig. 11.2 Melanoma of face

Decreased Pigmentation

Albinism

The most extreme form of diffuse hypopigmentation is congenital homozygous albinism in which the face is strikingly pink-white. The hair of both eyebrows and scalp is pure white. The irises appear red, owing to the lack of pigment and to the penetration of the retinal image. The glare of ambient light causes photophobia and nystagmus.

Albinos lack (or are deficient in) tyrosinase. It is an enzyme necessary for production of melanin from its precursor compound, tyrosine.

Far more common that pure albinism is partial albinism with varying degrees of hypopigmentation. Most individuals are blond, light-skinned, and blue-eyed and are unaware of their inherited condition. Sensitivity to light and sometimes nystagmus provide corroborating clues.

Vitiligo

Vitiligo is depigmentation in patches of the skin. Predisposed to sun-exposed areas, it often occurs in the face, particularly around the mouth, nostrils, and eyes and in the lips. Shocks of white hair in the scalp and eyebrows often coexist. While vitiligo is a benign condition, some autoimmune diseases are commonly associated. Among these conditions, consider in particular thyroiditis, pernicious anemia, and alopecia areata.

Texture

The texture of the skin is predominantly determined by its elasticity. Elastic fibers decrease with aging, one cause of wrinkling.

Taut Skin

Systemic Sclerosis

A most striking facial feature of systemic sclerosis (scleroderma) is its "pinched" appearance, especially noticeable in the nose. The taut skin greatly limits the capacity of facial expression, as if the patient were wearing a mask. The drawn mouth moves little when speaking and eating.

Slack Skin

Wrinkles

Here, conditions are included in which the elastic component of the skin is reduced. Naturally acquired wrinkles (rhytides) of aging are, of course, universal. Wrinkles that occur prematurely and with exaggerated extent and depth are a product of a damage-prone lifestyle. Excessive exposure to ultraviolet radiation damages the dermis and causes photoaging (the "weathered" face) so typical of those who are long exposed to the raw elements of outdoor life. Chronic hypoxic states, such as pulmonary insufficiency or living at high altitude, promote wrinkling. Smoking is a notorious cause of premature wrinkling.

Dehydration

Dehydration in the face is evident only when severe. Then, the eyes appear sunken, the cheeks withdrawn, and the lips parched. A more sensitive sign of dehydration in the face is dryness of membranes in the conjunctival sac and in the mucous membranes of the tongue and oropharynx. Because mouth breath can cause some degree of dryness, the most advantageous place to evaluate dehydration is in the gingival fossa between the lower lip and the gum.

The gradual reduction of the sense of thirst with aging probably explains the frequency of occult dehydration in the elderly [5].

Weight Loss

Substantial weight loss, intentional or not, causes the jowls to sag. Especially noticeable in persons who were obese is redundant skin hanging loosely below the chin.

Spots

Covered here are speckled, nonpigmented cutaneous lesions that occur predominantly or at least are prominent in the face.

Exanthems

By exanthem is meant a diffuse skin eruption that is a component of an acute systemic and usually a febrile illness. A reaction to a drug can also cause an exanthem (from Greek, to burst forth, to blossom). Sometimes the cause is the combination of febrile illness and a drug such as occurs in almost all patients with infectious mononucleosis who are given ampicillin. There is evidence that the illness induces formation of an IgM and IgG immunoglobulin, and these proteins form a complex with ampicillin. The result is a dissemination vascular reaction [6].

The rash may be seen as small flat or raised lesions (morbilliform) or large, confluent, erythematous patches (scarlatiniform). Here mentioned are common, widespread spots – typically from viral infections – that produce a characteristic pattern on the face. While these infections are typically associated with childhood, clinicians are seeing them with greater frequency in young adults whose parents had chosen to spare them as children the perceived hazards of vaccines.

The onset and progression of a rash are important factors in diagnosis. These infections are acute in onset; they present with varying degrees of malaise and fever. Diagnostic clues of two of the most well-known exanthems are readily identifiable.

Rubeola

Spots in measles (rubeola) first erupt on the face near the hairline and behind the ears, then spread downward. Lesions may appear on the palms or soles. Bright red at first, the lesions are flat (or slightly raised). They tend to merge into one another and turn dark brown. High fever and profound malaise, occurring 2–4 days before the eruption, are typical. Cough, coryza, or conjunctivitis (at least one of these findings, if not all)

appears 2–3 days after the onset of symptoms. Clearing of the spots occurs in a similar sequence – head to foot. All manifestations in uncomplicated measles last about 1 week. Predisposing complications of rubeola are meningitis, encephalitis, and permanent damage to the cochlear apparatus. These potential sequelae pose a serious challenge to parents who choose not to immunize their children against measles for fear of an unproven side effect from the vaccine.

Rubella

The skin lesions of *German measles* (rubella) begin as pink spots. As with measles, they begin on the face and spread from there. The spots are not the bright red of measles, and the magnitude of "sickness" is usually not so severe. Also differing from measles, the lesions do not merge but rather remain separate. Regional or generalized enlargement of lymph nodes may be present along with coryza.

Vesicles

Varicella

The eruption of chicken pox (varicella), usually starting in the scalp and spreading to the face, tends to appear in crops as slightly raised red spots. A typical lesion soon develops a central vesicle, described poetically as a "dewdrop on a rose petal." Each lesion soon undergoes desquamation, forming a crust from exuded serum before fading, all within 2–3 days (prompting the name: "the three-day rash"). Some lesions, on the contrary, may become pustules and are more persistent. The skin lesions of chicken pox remain separate and appear in various stages of development.

Finding a few scattered lesions typical of chicken pox may represent a limited outbreak in a patient partly immunized by natural infection or vaccination. Rarely, such vaccination expressions are found in older children and adults.

Herpes Zoster

Shingles (herpes zoster) is a localized recurrence of a varicella infection, caused by reactivation of the same virus hibernating in a dorsal nerve root. Thus, the outbreak involves the proximal sensory nerves. Lesions of shingles appear in clusters of tiny vesicles and are always unilateral in the immune-competent person.

In the face, shingles can appear in the cutaneous distribution of one of the branches of the trigeminal nerve – ophthalmic, maxillary, mandibular – or in any combination. H. zoster affecting the cornea is an ophthalmic emergency requiring urgent treatment.

In the pre-eruption stage of herpes zoster, the diagnosis is puzzling. For 2 or 3 days before vesicles appear, the patient may have a low-grade fever and experience malaise while becoming aware of hypersensitivity to touch over the affected area. A continuous burning, itching, or stabbing sensation is described. It is the astute clinician, using a corner of a facial tissue to demonstrate exaggerated skin sensitivity over a dermatome, who will suspect shingles during this pre-eruption period.

Herpes Simplex

A cold sore (herpes simplex) is a localized eruption of multiple minute vesicles, usually grouped along the lateral lip at the vermillion border. The lesions begin as flat erythematous spots that become slightly raised, fluid filled with later excoriation, and development of a serum crust. The whole process, from onset to healing, takes about 2 weeks.

Cold sores when they recur tend to do so in the same area. They are generally more widespread in persons with compromised immunity, tending to occur in the nose, cheek, and chin. Also in the immunocompromised, herpes simplex can become a widespread cutaneous eruption and produce encephalitis [7].

Petechiae

Petechiae are less common in the face than on the limbs. Their presence on the face should alert the clinician to the possibility of a major illness involving a reduced population or dysfunction of platelets or an underlying vasculitis. These

conditions have been described in Chap. 6. Differentiating palpable from non-palpable petechiae is critical diagnostically.

Angioma

Spider Nervi

A spider nevus is a small cluster of cutaneous blood vessels having a central arteriole and knurled capillaries that radiate out to venules. It is characterized by pulsations that can be blunted with slight pressure directly over the central arterial, causing the entire nevus to blanch. While spider nevi can sometimes be found in healthy persons, they are more common in chronic liver disease and in conditions with increased estrogens, including pregnancy.

Telangiectasia

A telangiectasia is a persistent dilation of capillaries in the superficial dermis, with the face as the most likely location. They are bright red and do not pulsate. Telangiectasias may be punctate, linear, or radiating. They are ubiquitous in the older population (senile telangiectasias), over areas of sun-damaged skin, and in rosacea and scleroderma. Telangiectasias themselves are benign, although conditions in which they are incidental may not be, as noted next.

Hereditary Telangiectasia

A troublesome exception to the benign character of scattered telangiectasia is "hereditary telangiectasia" (Osler-Weber-Rendu syndrome). In this syndrome, some capillaries are not merely dilated, but they are also grossly malformed and bulky, producing lesions that are dark red and slightly raised with "arterialization" of venules. In reality, these are tiny A-V fistulas. The lesions are most prominent on the lips but occur in the nasal mucosa and throughout the gastrointestinal and respiratory tracts. Lesions in the nasal mucosa cause unprovoked nose bleeds, while those in the gastrointestinal tract or lung pose the danger of spontaneous bleeding which may be occult or symptomatic. For additional description, see section "Lips" in Chap. 15.

Rash

A rash is an inflammatory reaction in the skin, producing a bright red appearance from markedly dilated blood vessels. Variations of this reaction are extensive. Only some of the more common rashes that affect the face are included here.

Allergy

In contact dermatitis, the face along with the hands is the most likely site for direct contact of allergic substances. The offending agent is usually a cosmetic (mostly fragrances or preservatives in facial creams). Metallic substances, particularly those containing nickel, frequently cause contact dermatitis; offending metals are found in jewelry, wrist watches, and eyeglass frames. Traditionally the ear lobes were most often exposed although contact dermatitis can occur virtually anywhere in keeping with current trends in body piercings.

Seborrheic Dermatitis

Dermatitis of seborrheic origin is a chronic inflammatory condition characterized by erythematous, greasy areas, particularly along the nasolabial folds and around the nose. Yellowish scales may partially cover the rash and appear in the scalp and eyebrows as dandruff. Areas affected tend to have an oily cast where small scales are continuously shed.

Lupus Erythematosus

A "malar rash" (referring to the cheeks) is often associated with systemic lupus erythematosus (SLE). It is known as the "butterfly rash." Characteristically, the bilateral rash crosses the bridge of the nose and may even extend up into the forehead. This feature helps distinguish a lupus dermatitis from rosacea (Fig. 11.3).

Intensely erythematous, the lupus rash is diffusely raised by edema. Scaling occurs as an acute manifestation. Atrophy of the skin in affected areas is a long-term consequence. There is a strong element of photosensitivity. See the section on HAIR in this chapter for additional clues of SLE.

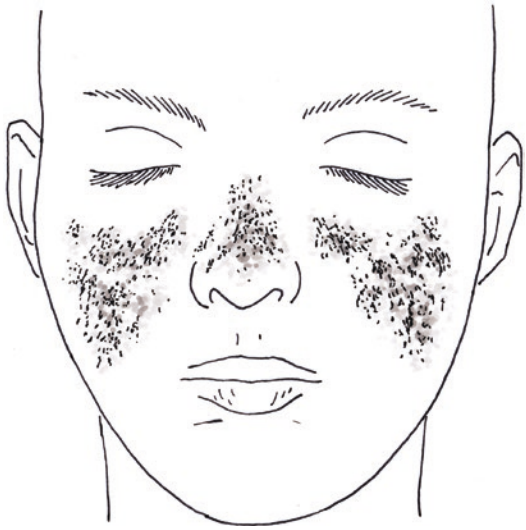

Fig. 11.3 Lupus erythematosus (facial)

Carcinoid Tumor

In this syndrome, vasoactive substances released from a gastrointestinal carcinoid tumor produce intermittent flushing of the face. The flush tends to be violaceous. Usually, metastases to the liver have already occurred by the time flushing appears [8].

Sarcoidosis

Sarcoidosis may present a facial rash similar to that of lupus erythematosus. An identifying feature of sarcoidosis may be a brownish infiltrate within the eyelids and ears.

Dermatomyositis

The facial erythema of dermatomyositis may be diffuse or mottled. It appears as a dusky reddish area over the nose, cheeks, and periorbital regions. The erythema can be most prominent in upper lids, its shape resembling a ring around the sun, prompting the name "heliotrope." This finding is highly suggestive of dermatomyositis. The rash can be highly pruritic in contrast to the cutaneous lesions of lupus erythematosus. An additional tip-off to the diagnosis is Gottron's sign: purplish papules on the knuckles described in Chap. 3 (Fig. 11.4).

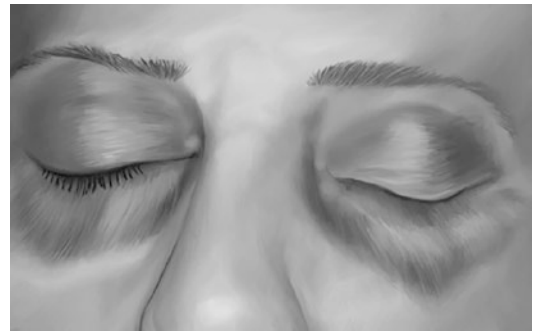

Fig. 11.4 Dermatomyositis (heliotrope)

Bumps

Papules

Bumps that frequently involve the face are an assortment of benign and malignant lesions. Usually these can be differentiated by considering of the basic clues. Any diagnostic uncertainty, however, dictates further investigation.

Acne

Who is not familiar with the zits of adolescence? These are small cysts in hair follicles where normal secretions from sebaceous glands are blocked. "Open" cysts exposed to air turn black (comedos) through oxidation. "Closed" cysts develop an inflammatory reaction and become pustules. The condition, referring to both forms, is known as acne vulgaris. Enhanced activity of sebaceous glands is an underlying condition. There is a strong androgenic component.

Seborrheic Keratosis

Thickened patches of light tan to deep brown with irregular outlines arise from the superficial layers of the skin. They may have a waxy, granular, or scaly surface that is slightly raised onto a thickened plateau. Non-cancerous, they occur mainly in sun-exposed areas and seem to be pasted on the skin. The lesions usually begin in those of middle age (although the first can appear at a much younger age), thus accounting for the term "senile

keratosis." They become more numerous with aging; an elderly person may have several.

Actinic Keratosis

The term "actinic keratosis" refers to small red bumps in sun-exposed areas ("actinic" is a Greek word for "ray" or "radiating"). The face and a bald scalp are most susceptible. They range from half a centimeter to the size of a quarter. Actinic keratosis may start as red macules, later becoming raised pink, tan, or flesh-colored lesions. Some eventually become thickened, dry, and scaly, developing in rough crusted patches and likened in texture to sandpaper. Less commonly, actinic keratosis lesions become wart-like and even develop horny projections. The keratinized skin can be sensitive and annoying, as if stinging or burning.

Immunosuppressive drugs greatly increase the susceptible to developing actinic lesions. Such lesions should be considered precancerous lesions. Look for tenderness and formation of nodules within the keratotic lesions, two warning signs that the lesions are transitioning to a squamous cell carcinoma.

Xanthoma

Xanthomas are raised and flattened, yellowish lesions near the eyelids, most often the inner canthus. They are usually bilateral, appearing almost symmetrical and sometimes in clusters. Of variable size, each lesion is notable for its sharp edges. Xanthelasma palpebrarum is a variant in which lesions arise directly on the lids (Fig. 11.5).

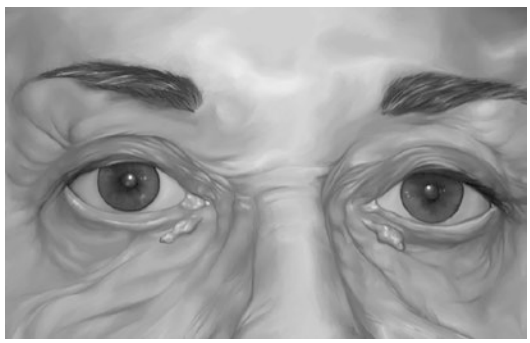

Fig. 11.5 Xanthoma of face

Xanthomas are lipid-rich foam cells from deposition of fatty material in subcutaneous tissue. There is an inherited propensity, but often the underlying problem is marked hyperlipidemia, particularly in VLDLs and triglycerides.

Urticaria

Urticaria (hives) of acute onset is a common allergic reaction. Hives are considered here because their predilection is for the face although, in fact, they may appear over any part of the body.

A typical urticarial lesion begins as erythema. Within minutes, it can swell into a raised and pale wheal owing to the leakage of plasma into the local interstitial space. Individual wheals vary in size from a few millimeters to gigantic. They can be intensely pruritic.

When urticaria occurs and is accompanied by any other rapid-onset manifestation of an acute allergic reaction – angioedema, bronchospasm, voice change, dysphagia, symptoms of hypotension – it is considered anaphylaxis, truly an emergency situation. To restate, anaphylaxis is any acute allergic reaction other than cutaneous and urgent intervention is indicated.

Neoplasm

The most common form of cancer is basal cell carcinoma which tends to occur on sun-exposed areas: the face and neck. The appearance can be highly variable, from a reddish or colorless patch to a wax-like, translucent papule to an open sore that oozes and crusts. In a dark-skinned person, a basal cell carcinoma may be dark. Well-developed lesions exhibit a "rolled" border within which there is central indentation (Fig. 11.6).

Squamous cell carcinoma is usually found on the lower lip. Typically, the lesion, sitting on a red base, has an irregular border. It is indurated and hyperkeratotic. Late warning signs of this dangerous neoplasm are ulceration with crusting and intermittent bleeding.

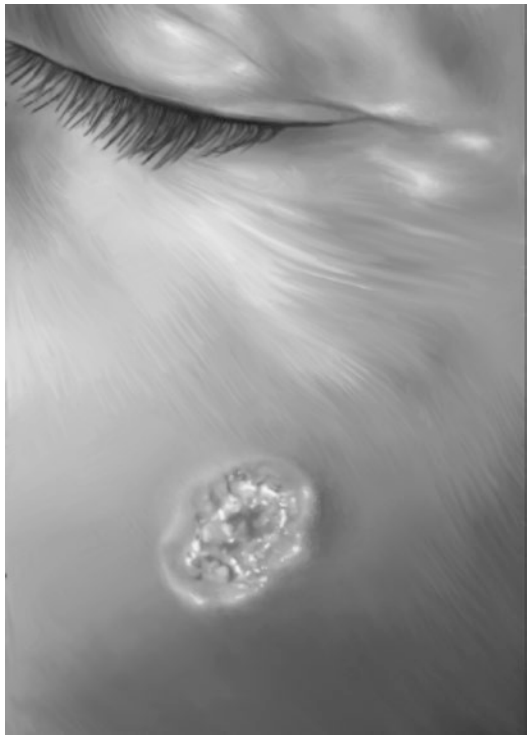

Fig. 11.6 Basal cell carcinoma

Hair

Precise diagnostic conclusions on disorders of the hair are complex, often requiring laboratory analyses. Mentioned here are those that are relatively easy to recognize and those that may reflect systemic diseases.

Vellus hair is the barely visible, ultra-fine, soft, short hair present in both sexes and throughout all ages but most obvious in the very young. It has an important role in exposing the surface area of sweat for evaporation and thereby contributes to the dissipation of heat. This function is particularly important in infants.

Terminal hair arises from the same follicles as vellus hair, but, in contrast, the tufts are long, bristly, and pigmented. The growth and distribution of terminal hair is regulated principally by androgens, although hormones of pregnancy, melanin, and thyroid have some influence. The eyelashes, in distinction, are resistant to hormonal control. On the face, terminal hair appears during puberty at first just above the upper lip and on the center of the chin.

Increased Hair

Hirsutism

Hair distributed in women in the adult male pattern is denoted hirsutism. Vellus facial hair becomes long and coarse, particularly in the mustache and beard areas. There may some receding of the frontal hairline. A tendency to become hirsute has a strong familial component.

When hirsutism is of recent onset, the clinician should look for clues of an endocrine disorder: pituitary, adrenal, or ovarian. Consider also drugs with androgenic properties; "body building" agents are commonly responsible. If excessive facial hair is accompanied by cystic acne and oligomenorrhea, the possibility of polycystic ovarian syndrome is particularly suspect.

Hypertrichosis

Hypertrichosis is terminal hair growing in extraordinary places, such as the forehead and upper cheeks, and that is independent of androgenic regulation. The finding may be a clue to the presence of Cushing's disease or to thyrotoxicosis, anorexia nervosa, and medications (phenytoin, cyclosporine, and anabolic steroids).

Decreased Hair

Balding

Balding in men reflects a genetic pattern in which the underlying cause is not well understood. Paradoxically, balding is partly dependent upon androgen and, in fact, is known as the "androgenic male pattern." Despite ongoing speculation, there has been no decisive evidence that there is a link between male baldness and any systemic disease. On the other hand, balding in women suggests some degree of virilization, most frequently polycystic ovarian syndrome.

Alopecia

Alopecia areata affects the scalp, usually with a rather abrupt onset of hair loss in patches, often in a circular pattern. Terminal hairs may be broken. The hair follicle is generally preserved so that regrowth of hair is likely. The condition is

thought to be autoimmune in origin. Fingernails may have dystrophic changes. Tinea capitis, psoriasis, seborrheic dermatitis, and head lice are putative comorbidities.

When patches of alopecia cause scarring and atrophy of the scalp, the term cicatricial alopecia is used. Here, the scalp has a violaceous discoloration. Loss of hair follicle structure is irreversible. These findings suggest discoid lupus or systemic lupus erythematosus as the underlying pathology although herpetic, bacterial and fungal infections may be responsible.

Texture Changes

In hyperthyroidism, the hair becomes fine in texture. Many people with Grave's disease will develop some degree of alopecia.

Hair in myxedema tends to be dry and brittle. The eyebrows are thinned out, particular in the lateral aspects.

Sinuses

Physical signs of acute sinusitis of the face are admittedly insensitive. Palpation is futile. Fluid accumulated in the sinuses is detectable by percussion and transillumination only when the volume is large. These techniques are unproductive in most cases of acute facial sinusitis.

Certainly, maxillary sinus is by far the most site of common site of facial sinusitis; drainage requires the action of cilia to convey mucus uphill to the ostia and entrance into the nasal chamber. Flow of mucus from the ethmoid and frontal sinuses is augmented by gravity. Acute respiratory illnesses not only produce a greater volume and viscosity of mucus secretions in the sinuses, but the inflamed and engorged mucus membranes tend to block the ostia. In addition, the cilia that conveys mucus secretions from the sinuses into the nasal cavity (the "ciliary escalator") is lost when the sinus epithelium is shredded. For completeness, mention is made of the clinical rarity of sphenoid sinusitis; it is associated with serious and diffuse systemic complications such as headache and ocular symptoms.

Acute sinusitis is such a frequent complication of the common cold that the term "rhinosinusitis" has been suggested. To make a diagnosis of acute facial sinusitis, the clinician, short of imaging, is left with a history: a recent common cold with rhinitis or reactivation of an allergy. One symptom highly suggestive of acute sinusitis is pain in the face from the jarring of walking downstairs. Additional information on sinusitis can be found in Chap. 14.

Nervous System

This section is intended to provide a foundation for performing a credible neurological evaluation as it concerns the face, helping to localize a lesion and to assess its relative significance. It is not meant to cover the fine points expected of a neurologist but rather to provide enough direction for the nonspecialist to derive some satisfaction and confidence in further evaluation of a neuromuscular symptom or finding. The descriptions presented here can serve as a basis for understanding the complex neuroanatomical principles that may also affect the extremities.

Motor Function

The Face at Rest

Aside from blinking, the normal relaxed face projects a level of alertness even when relatively immobile in the clinical encounter. Blinking normally occurs about ten times a minute. It speeds up when talking and slows down while listening. Anxiety increases the frequency of blinking as do drugs that have a dopamine-like action such as those used to treat psychoses and Parkinson's disease. Bradykinetic conditions decrease blinking, tending to cause discomfort from dry eyes. Such is the appearance of the relatively expressionless face of Parkinson's disease, especially when coupled with a persistent stare from retraction of the orbicularis palpebrarum.

Testing for motor function of the face is quickly done. The patient wrinkles the forehead,

squeezes the eyes shut, and exaggerates a smile. If these observations appear normal, if the ability to speak is natural, and if there are no symptoms or physical suggestions of neurologic disorders, there is probably no need to pursue other neurologic testing of the face.

Increased Movement

Tic

A tic is an abrupt contraction and relaxation of a muscle, often in the face. Its twitch is repetitive but nonrhythmic. It is habitual but semi-voluntary; that is, the patient may have some control during an urge to resist its suppression. The contraction somehow relieves momentary tension.

The stress of the physical examination or of stimulants, including caffeine and cocaine, can increase the frequency of a tic. When such motor events occur along with simultaneous vocalizations, the term Tourette's syndrome is applied.

Spasm

A brief, involuntary and forceful muscle contraction is denoted a *spasm*. The hemifacial spasm is repetitive, occurring on one side of the face and involves one, two, or sometimes all three branches of the facial nerve. A possible cause is partial recovery of the nerve following an acute facial neuropathy (such as Bell's palsy).

Chorea

Choreiform movement consists of sudden, nonrhythmic, non-repetitive contractions that can happen virtually anywhere in the body as they skip unpredictably from one place to another. In the face, these hyperkinetic movements occur in the jaw, around the mouth, and in the tongue. They can disrupt speaking and swallowing. The disorder is attributed to abnormal impulse regulation between the cerebral motor cortex and the basal ganglion.

Rarely, chorea-form movements that are temporary occur during pregnancy. They may also be a complication of acute rheumatic fever (Sydenham's chorea) and systemic lupus erythematosus. Certain neurotropic drugs can be causal as can an infarct of the basal ganglion. When chorea-form movements are chronic, they most likely represent an inherited disorder, such as Huntington's chorea.

Dyskinesia

Dyskinesia in the face involves frequent or continuous involuntary, twisting movement around the eyes or mouth. Facial grimacing, twitching, or writhing is characteristic. The dyskinesia may include repetitive pursing of the lips, tongue protrusion, and movement of the jaw. The cause is usually a disorder of the extrapyramidal system or a side effect of a neuroleptic drug.

Tremor

Tremor of the face is most commonly a component of Parkinson's disease, called in the nineteenth century "shaking palsy." The tremor is generally a slow, side-to-side rhythmic motion of the jaw. In addition, the lips and tongue may be tremulous. Although tremor is usually the most conspicuous feature of Parkinson's disease, the major problem is, paradoxically, reduced spontaneous movement and rigidity, described more fully in the section "Decreased Movement" that follows.

Other causes of facial tremor are withdrawal from many drugs (including anticonvulsive and antidepressant agents such as lithium) and from alcohol. When an unexplained tremor with rigidity is discovered in a young adult, the possibility of copper overload from Wilson's disease should be considered. The iris is a good place to look for an identifying sign: a partial or complete yellowish brown ring at the periphery (limbus) of the iris, the so-called Kayser-Fleischer ring. Typical signs of advanced liver disease from chronic active hepatitis and cirrhosis may be present. Varying manifestations of psychiatric or neurologic disturbances are common in Wilson's disease.

Fasciculations

Fasciculations are fine twitches of muscle fibers that indicate disease of lower motor neurons. These result from the spontaneous discharge from anterior horn cells of the spinal cord. The muscle can respond to the erratic stimuli. In primary diseases of muscle, by contrast, there are no fasciculations.

Fasciculations are most readily observed in the tongue. They can also occur in any distribution of the facial nerve. Such twitches can occur in

healthy persons and are only important when associated with muscle weakness. Any of the demyelinating diseases, including multiple sclerosis and amyotrophic lateral sclerosis, may cause fasciculations. When coupled with rhythmic contractions of the palatine muscles, the voice takes on a tremulous nature.

Bobbing

Head bobbing is a sign of aortic valve regurgitation. It occurs immediately after ventricular systole during the rapidly collapsing diastolic pressure. Another possible cause of bobbing is an extremely high cardiac output as might occur in a major arteriovenous fistula.

Decreased Movement

Bradykinesia

In Parkinsonian's disease, the face shares in the generalized bradykinesia and rigidity as the condition advances. That is, some movements (such as walking) are slow to initiate and slow throughout the action. The result is asynchrony in muscle contraction and relaxation. A physiological basis is the loss of dopamine-producing cells in the substantia nigra. The substantia nigra is a part of the basal ganglion where the execution of learned movement is controlled. The function of this center is to attenuate excitation of muscles from impulses coming from the cerebral cortex. Dopamine facilitates this inhibitory action. A deficiency of dopamine-generating cells results in poorly modulated impulses of the pyramidal tract. Basically, there is a compromised ability to initiate purposeful movement.

An immobile face (the Parkinsonian "mask") is characteristic of advanced Parkinson's disease. Blinking decreases in frequency as does general mobility. The dynamics of voice and facial expression are reduced, as is oculomotor function (namely, convergence of gaze). There is often a tremor of the protruded tongue, while tremor of the jaw occurs but is less common.

Certain drugs can induce bradykinesia and tremor, thus mimicking Parkinsonism. Those culpable include the antipsychotic drugs such as chlorpromazine or neuroleptics, lithium, amiodarone, pesticides, and ethanol. The akinetic-rigid syndrome may also follow infections (mainly viral infections) or be associated with infectious or autoimmune damage to the basal ganglion complex.

Weakness

Facial weakness at rest is most noticeable by comparing the corners of the mouth. The asymmetry is increased with an exaggerated smile with the stronger pull toward the unaffected side. For simplification, the location of neuronal lesions causing weakness is presented here as (1) upper, (2) middle (or brain stem), and (3) lower motor neuron syndromes.

1. Upper motor neuron syndromes (also known as supranuclear, corticobulbar, or central motor syndromes)

   Syndromes originating within fibers of the upper motor neuron syndromes can occur anywhere from the motor area of the cerebral cortex (forward of the central or sagittal sulcus, also known as the Rolandic fissure) to the midbrain. They largely affect the skeletal muscles on the contralateral side (although about 10% of these fibers do not decussate).

   (a) Motor cortex

   The portion of the cortical motor area that supplies the facial muscles is located on the most lateral aspect. Vascular lesions (thrombosis or hemorrhage) here are more likely to induce weakness in a relatively limited area of the subtended neural territory. They mainly cause weakness of the lower face on the contralateral side. The upper face is relatively spared owing to cortical innervation by both ipsilateral and contralateral fibers. Wrinkling of the forehead on frowning, consequently, is not affected. Put in another way, when facial palsy is from a lesion in the central nervous system, wrinkling of the forehead is preserved. Wrinkling does not occur if the paralyzing injury of the facial nerve is peripheral.

   When lesions that affect the motor cortex also produce sensory loss in a similar distribution, the question arises of a vascular

event or breaching of the central sulcus by a mass lesion.

(b) Pyramidal (corticospinal) tract

Fibers leading from the broad motor cortex merge to form the compact bundle, the pyramidal tract (also known as the internal capsule). Lesions in this area are more likely to produce pure motor loss and involve a large subtended territory. Even very small hemorrhages or thrombi here cause devastating hemiparesis or hemiplegia, often affecting the face and the upper and lower extremities.

Persons incurring hemorrhage of the internal capsule will typically have lost no sensations. The arm is held flexed owing to weakness of the muscles of extension. The leg is extended because the muscles of flexion are more compromised.

The middle cerebral artery supplies the pyramidal tract through tiny branches (the striate arteries) that emerge at right angles. These branches are more susceptible to rupture from high blood pressure than ordinary Y-branching arteries that provide a gradual step-down of hemodynamic pressure. A look at the anatomical relationship of this neural tract and at the arteries that supply it provide an clear understanding of the pathogenesis of the most common stroke (Fig. 11.7).

Hemiplegia without sensory loss from hemorrhage of a striate artery was by far the most common cause of major strokes. This cerebrovascular accident has become uncommon since effective measures to control hypertension have become available.

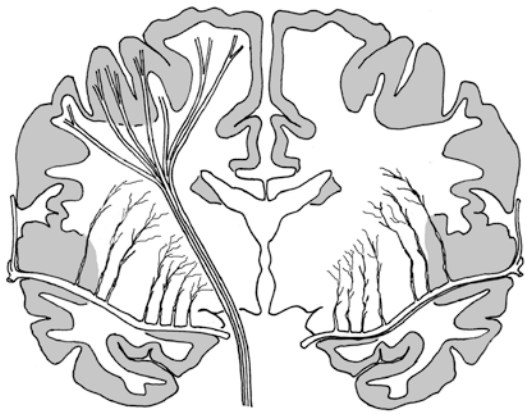

Fig. 11.7 Blood supply of internal capsule (pyramidal tract)

Cortical motor lesions that affect the whole body tend to spare the face to some degree, owing to bilateral representation within the pyramidal tract pathways. The exception is the mandibular branch of facial nerve. Consequently, chewing is not effected even with a massive disruption of the corticostriate tract.

Interruption of fibers in the pyramidal tract to the face results in tightening of the jaw. This increased muscular tone is demonstrated by tapping the chin. It is a deep tendon reflex (masseter reflex) equivalent to the exaggerated knee jerk on striking the patella ligament.

(c) Bilateral upper motor neuronal lesions

When lesions in the upper motor neuronal system are bilateral, generalized facial and limb weakness occur, along with disabling complications of dysarthria, dysphagia, dysphonia, and emotional lability. Muscles for chewing (masseter, temporalis, and pterygoid) are markedly impaired. Toxic or infectious encephalopathies are the usual causes. Because the syndrome resembles lesions within the nuclei of the brainstem, it is known as "pseudobulbar palsy."

2. Middle motor neuron (brain stem or nuclear) syndromes

The middle nuclear syndromes occur from lesions within the brain stem nuclei or in nearby pyramidal tract fibers. This portion of the brain is referred to as the bulb. It is where the large majority of fibers of the tightly packed pyramidal tract cross over to enter the spinal cord. Deeper within the bulb are the nuclei that control the cranial nerves (Fig. 11.8).

A bulbar lesion can disrupt the flow of motor stimuli within the decussating pyramidal tract at the pons, producing weakness that is principally on the ipsilateral side. Because of their proximity, lesions within or near the pons frequently affect nuclei of the cranial nerves, individually or in multiple. A lesion within the area

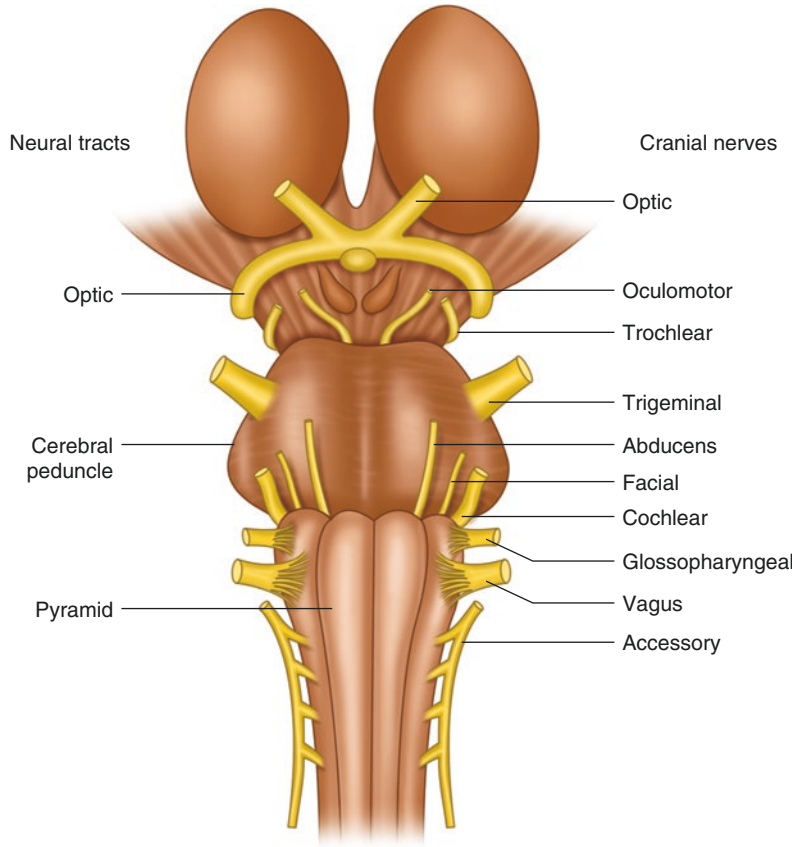

Fig. 11.8 Ventral surface of brain stem

should be suspected when facial paresis or paralysis is associated with any disturbance of conjugate gaze, such as weakness of lateral rectus muscle on the opposite side.

With bulbar lesions, the power of eye closure and spontaneous blinking may be reduced, caused by a defect of the facial nerve. Food tends to accumulate within the buccal fossa. Sensory tracts of the trigeminal nerve may be involved as well. Thus, lesions in this part of the brain produce profound disturbances of neurological function in a great variety of syndromes. Vascular insults within the basilar-vertebral arterial systems – either hemorrhage or thrombosis – are usually the underlying event. The general clinician need not remember all the names of these many syndromes but should be mindful of the implications of facial weakness along with signs and symptoms of cranial nerve deficits.

3. Lower motor neuron syndromes

Motor fibers that are distal to the pons are referred to as lower motor neurons. Lesions of these fibers make up the peripheral neuropathies. These may occur within the skull, at the emerging site from the skull or exterior to it, including the terminal portion of the nerve.

Facial Nerve

Peripheral neuropathies and neural injuries can involve any or all branches of the facial nerve. Lesions of the more distant portion of the facial nerve tend to involve a single division. If affected at or near the bony canal as it emerges from the stylomastoid foramen, all three branches of the nerve are likely to be involved: ophthalmic, maxillary,

and mandibular. Inflammation or injury of the facial nerve distal to the stylomastoid foramen can result in weakness in any one of the branches.

(d) Ophthalmic branch. Weakness on closing the eyelid indicates damage of the ophthalmic branch of the facial nerve. Dryness of the eye occurs from infrequent blinking, with cornea ulceration a possible outcome.
(e) Maxillary branch. Flatness of the nasolabial fold and sagging of the corner of the mouth on the affected side characterize maxillary branch paralysis disease.
(f) Mandibular branch. Involvement of the mandibular branch interferes with chewing. It innervates the masseter muscle that, gram for gram, is the strongest muscle in the body.

Causes of proximal facial nerve injury may be local inflammation or infection. Parotitis, Lyme disease, sarcoidosis, infectious mononucleosis, and AIDS are possible offenders. When the cause of weakness of the facial nerve is not apparent (as occurs in about half the cases), the condition is referred to as Bell's palsy. Thought to be viral in origin, Bell's palsy at the time of onset may present with vesicles behind the ear. This finding helps identify the leading suspected cause: herpes simplex [9]. Sjögren's syndrome has been reported as an unusual cause of Bell's palsy [10].

In addition to the mimetic muscles of the face, the facial nerve also serves the stapedius muscle. The stapedius contracts in response to loud noises, serving to protect the inner ear by dampening severe vibrations within the chain of auditory ossicles. When a lesion occurs in the proximal section of the facial nerve, the patient may experience hyperacusis, that is, the perception of increased loudness on the affected side, mostly for sounds of low pitch. The symptom is common in Bell's palsy.

It should be remembered that the facial nerve, while having predominantly a motor function, also carries sensory and parasympathetic fibers. Involvement of the sensory component of the facial nerve blunts taste in the anterior tongue where, normally, sweet and salt are tasted. Disabled parasympathetic axons in the facial nerve reduce tearing and moisturizing of the palate and nasal mucosa.

Chronic palsy of the facial nerve leads to atrophy of muscle, as is typical of peripheral neuropathies. At rest, the cheek is sunken, and the face is pulled toward the affected side, paradoxically giving the impression that it is the normal side. An exaggerated smile, however, will cause the mouth to shift toward the innervated side.

Trigeminal Nerve

Predominantly a sensory nerve, the trigeminal nerve does have an essential motor functions. It contributes to innervation of the muscles of mastication: the masseter, temporalis, and medial and lateral pterygoids. Problems of chewing – when coupled with loss of sensation in the territory of the trigeminal nerve – identifies peripheral neuropathy of this fifth cranial nerve.

On testing, the rapid opening and closing of the mouth, there will be a slight lag on the side where there trigeminal nerve is weakened. The face may appear misaligned. The diagnosis presupposes that there is no coexisting weakness in the distribution of the facial nerve.

Systemic Motor Diseases

Mention is made of three forms of weakness from peripheral nerve diseases that are rare but are nevertheless important to recognize: Guillain-Barré syndrome, botulism, and myasthenia gravis. Signs of these dangerous conditions that may be revealed in the face are covered briefly.

Guillain-Barré Syndrome

Guillain-Barré syndrome is an acute demyelinating disease, causing both motor and sensory changes. Weakness of facial muscles is bilateral and is reflected in loss of facial expression, difficulty in speaking and swallowing, and numbness. Actually, the neurologic symptoms typically begin in the lower extremities and within hours ascend to affect the arms, then the face. It is thought that the syndrome is autoimmune, perhaps initiated by a viral infection. Rarely, it occurs late in pregnancy.

Botulism

The toxin that causes botulism is produced by the anaerobic bacterium, *Clostridium botulinum*, which may contaminate "preserved" food or necrotic wounds. An early sign of acute botulism is ptosis, singular or bilateral. Inhibitions of parasympathetic innervation rapidly progress to facial weakness, mydriasis with blurred vision, diplopia, and dry mouth, and dysphagia. Weakness descending to the limbs and diaphragm indicates the life-threatening complications. The defect, in brief, is an acute blocking of acetylcholine-producing nerve endings at the neuromuscular junction.

Human ingenuity has recruited botulin toxins for a cosmetic purpose. A controlled injection into a muscle prevents excessive contraction and, thus temporarily, removes wrinkles.

Myasthenia Gravis

Myasthenia gravis is another defect within the acetylcholine-dependent junction. It involves resistance of the muscle receptor to a neural stimulus. Characteristic of myasthenia gravis is the gradual tendency of weakness to progress with sustained or repeated contractions. Ptosis of one or both eyes may be the earliest finding. It is often associated with oculomotor weakness, manifested by intermittent diplopia.

The signs to identify myasthenia gravis are brought out by holding the gaze upward and outward or by repetitive rapid blinking. As the condition advances in intensity, weakness becomes apparent in reduced facial expression and in difficulty with continuous talking, chewing, and swallowing.

Sensation

Stimuli from sensory receptors in all three branches of the trigeminal nerve converge at the trigeminal ganglion. From here, they enter the brainstem to form the nucleus of the fifth cranial nerve. This nucleus is long and slender; it extends from the brainstem into the upper cervical spinal cord. Nerve fibers from the nucleus decussate to the contralateral side where they enter the thalamus. From there, they ascend to the somatosensory gyrus of the cerebral cortex (Fig. 11.9).

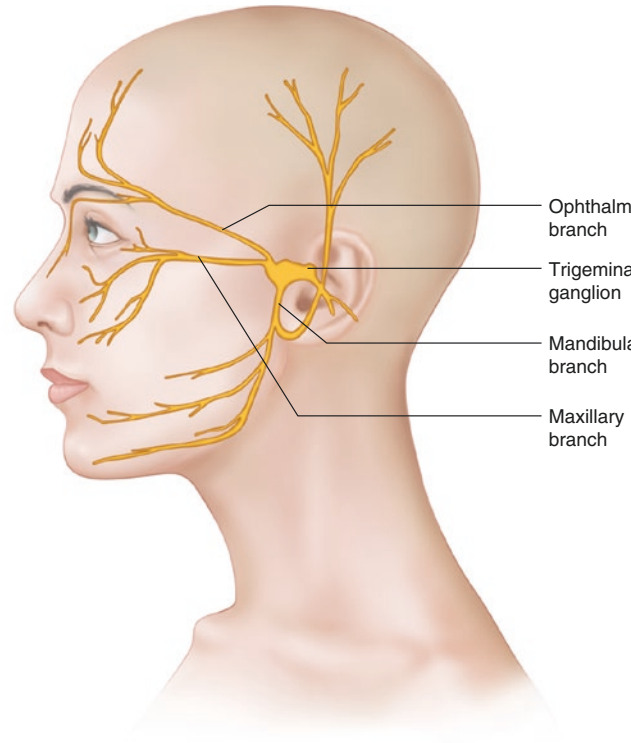

Fig. 11.9 Sensation: trigeminal nerve

The neuroanatomy of sensation is complex. For example, fibers from various sensory receptors of the trigeminal nerve terminate in various parts of the elongated nucleus of the facial nerve. For our purposes, the explanation of this system has been oversimplified.

Peripheral fibers subtending the face comprise the trigeminal nerve. It divides into three divisions: ophthalmic, maxillary, and mandibular. While the trigeminal nerve is predominantly a sensory nerve, the mandibular branch innervates muscles involved in chewing; this action was described earlier under motor function.

Increased Sensation

Paroxysmal Neuralgia

Sharp stimulation of the trigeminal nerve sends an electrical or shooting sensation into one side of the jaw, cheek, or periorbital area. The cause of paroxysmal facial pain could be a demyelinating process within the trigeminal nerve from vascular disease or from multiple sclerosis. When these attacks are repetitive and there is no demonstrable neurological injury, the condition is known as trigeminal neuralgia or "tic douloureux." The term "tic" refers to the facial twitch that is a response to a paroxysm of sharp pain. This condition must be differentiated from postherpetic neuralgia and cluster headache as well as from dental pathology.

Herpes Zoster Neuralgia

Discomfort or outright pain in the trigeminal distribution can be caused by herpes zoster from reactivation of the varicella virus. If affecting the trigeminal nerve (or any of its branches) and in the "pre-eruption stage," patients may describe the sensation of stinging or burning in the subtended territory. For a day or two prior to the outbreak of typical vesicular lesions, the patient may experience mild constitutional symptoms (such as fever and malaise) along with regional pain or hypersensitivity. While perplexing at this stage, the diagnosis can be supported by detecting hypersensitivity to touch along the affected region, as described above under section "Vescicles."

Any sign of herpes zoster that occurs in the area of the eye should be considered an ophthalmic emergency. See Chap. 12 for details.

Thalamic Disorders

Symptoms from injury to the thalamus are difficult to describe other than exceedingly unpleasant and unremittent, but they may have a burning or tearing character. A thalamic lesion might be suspect as the cause of sudden unexplained tingling, numbness, or excruciating pain over the cheek or periorbital area on one side. A lesion within the thalamus is difficult to diagnose by physical examination. One clue is exacerbation of the discomfort by light touch, as with a facial tissue, in the affected area. An additional clue that a sensory lesion is central is that the sensations affected tend to be at more complex levels of development than those for touch and sharp. These levels, for example, are two-point discrimination and recognition of shapes or written numbers on the skin.

Decreased Sensation

The source of a sensory defect causing numbness or paresthesia of the face may be anywhere from the sensory cerebral cortex, in the thalamus or trigeminal ganglion to the most peripheral twig of a trigeminal nerve branch. Localization is often difficult owing partly to the complexity of the sensory system pathways and partly to the sustained attention required of the patient.

Light touch is an adequate screening test. Admittedly, it may be the weakest of all neurologic testing since it depends upon keen perception of the patient. If a defect is found by light touch in any of the trigeminal nerve branches, follow-up testing is indicated, to include checking for perception of sharpness and/or temperature. More discriminating functions can be tested as well such as two-point discrimination and recognition of numbers "written" on the face. These last mentioned, however, are beyond the scope of these guidelines.

Lower Sensory Neuron Syndromes

Injury resulting in anesthesia in one or more of the three branches of the trigeminal nerve can be

mapped by testing either sharpness or temperature. If the latter is done, using a cool object (such as a vibratory fork), the affected side will feel cooler.

Trigeminal Nerve

1. *Ophthalmic branch*. This division transmits sensations from the nose, supraorbital area, and anterior scalp. It transmits the afferent arm of the corneal and palpebral reflexes.

   Corneal and palpebral reflexes are tested by applying the twisted end of a facial tissue to the sclera and slowly dragging it until it touches the cornea. The sclera is relatively insensitive to touch, the cornea exquisitely sensitive. The patient will wince with discomfort, and the lids will tighten the instant the cornea is stimulated. This test, need it be said, is not performed routinely and is not well liked by patients.
2. *Maxillary branch*. The infraorbital, upper cheek and upper lip provide sensory fibers within this division. Like the ophthalmic branch, the maxillary branch contains carries only sensory stimuli.
3. *Mandibular*. The chin, lower lip, and lower cheek are within the territory of this division. Unilateral numbness in these areas, if not from obvious trauma, may arise from a dental problem. Otherwise, a careful search for an underlying, occult malignancy of the jaw or mouth is appropriate.

The mandibular branch of the trigeminal nerve, in contrast to the ophthalmic and maxillary branches, also has motor actions. It supplies the set of muscles involved in chewing. The jaw may be aligned slightly away from the weaker side. Specific testing of facial motor function is described above.

One of the distal nerve syndromes is "trumpet player's neuropathy." Persistent pressure of the mouthpiece against the anterior-superior alveolar nerve can result in midline pain or anesthesia and may affect playing. A causal factor in this "embouchure entrapment neuropathy" related to wind instruments may be a protruding tooth [11].

Trigeminal Ganglion

Injury of the trigeminal ganglion may occur from infection or result after neurosurgery. It causes reduced sensation on the same side of the face and is likely to affect all three of its branches.

The trigeminal ganglion may be the site where varicella viruses reside. On activation, they induce cutaneous herpes zoster or herpes simplex lesions in the distribution of the trigeminal nerve.

Middle Sensory (Brain Stem) Neuron Syndromes

The nucleus of cranial nerve V, receiving impulses from the trigeminal ganglion, is a long, slender tract that extends from the rostral part of the brainstem to the upper cervical spinal cord. Vascular or compression injuries can cause a wide range of clinical manifestations because of the location of the termination of different sensory receptors. The defects are on the same side as the lesion.

Considering its length, lesions of the cranial nerve V (trigeminal) nucleus will often extend to adjacent cranial nuclei. Most often, the extended injury affects the extraocular muscles with diplopia and nystagmus evident. Disturbances in other nuclei within the brain stem may result in severe nausea and vomiting, dysphagia, hoarseness, or ataxia in the limbs.

If hemianesthesia of the face occurs in the presence of ipsilateral hearing loss, tinnitus, and/or vertigo, the possibility of an acoustic neuroma (vestibular schwannoma) should come to mind. Of all the tumors of the brain, the acoustic neuroma is one of the most common.

Because of the extended length of the trigeminal nerve nucleus, damage in one part can impair function in varying subtended territories, a phenomenon called "sensory dissociation." For example, a reduction in sharp and temperature sensation occurring in the face may cause a similar defect on the opposite side of the lower body (called "Wallenberg's syndrome"). Often associated are signs and symptoms of acute brain stem disease: dysphagia,

nausea, and nystagmus. The most likely cause is an occlusion or hemorrhage of a branch of the vertebral artery.

Upper Sensory Neuron Syndromes

Cerebral Cortical Lesion

Sensory fibers from the trigeminal nucleus cross over to the thalamus. From there, in the supranuclear pathways, impulses are relayed to the cerebral cortex. Facial sensations are represented in the lateral aspect of the sensory gyrus.

Lesions of the sensory cortex are readily recognized by the highly specific territory involved on the contralateral side. Often, hypesthesia from injury to the sensory cortex is coupled with weakness of the same area because of its proximity to the motor cortex. The combination suggests a vascular event or a mass lesion that bridges the central sulcus.

Thalamic Lesions

Injury to any part of the thalamus generally causes sensory defects in indefinite territory. They involve impairment of all forms of sensation on the opposite side. Thalamic disorders have been described above under section "Increased Sensation."

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Eye

12

Essentials
Palpebral fissures
Pupils
Sclera
(Visual acuity)

The viewpoint is taken here that the general clinician can manage many of the common eye problems while recognizing when care by the specialist is indicated. Furthermore, abnormal findings in the eye may actually be the clue as an early manifestation of a systemic disease. A tremendous amount of information about the eye can be gained within a few minutes. Some guidelines are offered here.

The clinical implications of a basic but reliable eye examination are great. For example, finding normal vision in a "red eye" of recent onset is strong evidence for a benign viral infection, a foreign body speck, or irritation from contact lens cleansing fluid. If instead, vision is compromised in the eye without obvious explanation, the illness constitutes an ophthalmic emergency in which inflammatory disease of the ciliary body, acute glaucoma, or other serious condition – ophthalmic or systemic – is suspected.

Other abnormal findings in the eye, even the incidental ones, may be critical clues to the possibility of systemic diseases of great variety. Certainly, the well-practiced clinician can find a careful examination of the eye to be an interesting and fruitful everyday practice. It is an organ easily assessable to accurate and rapidly assembled information that is organ-specific as well as being a sensitive mirror of general health.

How detailed should the examination of the eye be in the patient with no symptoms referable to the eyes? If palpebral fissures are equal, pupils are of normal size and equal, and the sclerae have a normal color, then the clinician has performed the essentials. Even so, testing of visual acuity by reading aloud, described later under the section "Vision," can add a wealth of information within seconds.

Palpebral Fissures

The palpebral fissures are considered first because they are the most obvious features on the initial observation. An important exception: if the examination is being done because of an eye complaint (illness or injury), it is good practice to start with visual acuity.

The normal symmetry of the palpebral fissures indicates intact lid innervation and position of the globes. Opening of the eyelids is mediated by the oculomotor nerve with a minor contribution by the sympathetic nervous system, the superior tarsal muscle. Closure is a function of the facial nerve. Thus, the palpebral fissures are determined by the continuous interactions of opposing forces.

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Keeping this relationship in mind is helpful in sorting out the various abnormalities that affect the size of the palpebral fissures.

The upper lid normally rests just below the superior margin of the iris so that sclera here is not visible at rest. A small arc of sclera, however, is normally visible just beneath the iris. Lack of symmetry of the palpebral fissures is definitely abnormal. It may be widened or narrowed by various mechanisms: neurologic, mechanical, and myogenic.

Widened Palpebral Fissure

Sclera observed above the iris usually indicates a wider palpebral fissure. Retraction of the eyelid(s) is most often responsible with Graves' disease, the preeminent cause. (See below under Section "Hyperthyroidism.") Neurogenic causes include the "eye-popping reflex" of infants, myasthenia gravis, and a host of lesions of the central nervous system. Mechanical force can push the eye forward: retro-orbital tissue expansion from connective tissue or neoplasm or "blowout fracture" of the orbital floor [1] (see below under section "Proptosis") (Fig. 12.1).

Hyperthyroidism

The most common ophthalmic complication of Graves' disease is retraction of the eyelids [2] which is usually bilateral and which produces a gaping stare. The pathogenesis involves antibodies bound to thyroid-stimulating hormone receptor that react with ocular muscle proteins and produce a chronic inflammatory reaction. The ensuing changes include fibrosis as well as thickening of muscles and the retro-orbital connective tissues. Retraction of the lid is an added complication. These manifestations often do not appear until after the hyperthyroidism is brought under control.

Lid lag is a sensitive test for assessing reduced mobility of the eyelids as might be caused by restrictive fibrosis of the orbital muscles. It consists of observing the palpebral fissures as the eyes follow a descending object. The object in focus should be lowered slowly from just above the head to below the chin. In an abnormal test, some exposure of sclera above the iris will occur because of a slight delay in the flexion of the upper lid as it follows the descending globe.

Proptosis

Exophthalmos – protrusion of the globe or "proptosis" – is a less frequent complication of Grave's disease. It is usually bilateral. The mechanism of exophthalmos is deposition of fat and thickened connective tissue within the retrobulbar space.

Also, a widened palpebral fissure may be from the eye being forced forward by a mass behind the globe. In the absence of thyroid disease, a neoplasm – particularly ocular lymphoma – is suspected. The possibility of a retro-orbital mass should be considered strongly if the proptosis is unilateral.

Mild degrees of proptosis may be difficult to detect and could appear as simply retraction of the upper lid. It is best detected by observing the eyes from the side or from above, looking down on the seated patient.

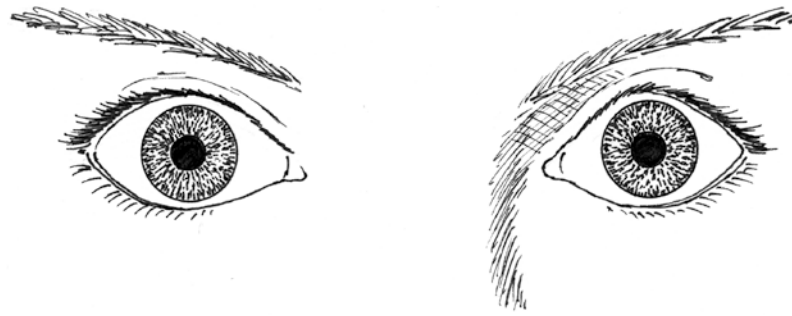

Fig. 12.1 Increased palpebral fissure (exophthalmos) (bilateral)

Ectropion

If the lower lids sag outward, the palpebral fissure is widened. The usual cause is advanced aging from relaxation of the orbicularis oculi. Scaring of the tarsal plate from infection is a second if far less likely etiology. Besides the cosmetic issue, ectropion can be associated with dryness and, paradoxically, excessive tearing.

Entropion

When either the upper or lower lid is distorted inward, the width of the palpebral fissure may be altered. Entropion is a condition in which every blink causes the eyelashes to abrade the cornea. Laxation of supporting tissue from aging is the preeminent cause in the hygienically modern world where the condition is rare. In warm climates where sanitation practices are meager, blindness from trachoma caused by entropion is endemic. A species of the bacterium *Chlamydia* is the infectious agent in trachoma.

Narrowed Palpebral Fissure

The upper lid is maintained open by the levator palpebrae superioris muscle innervated by the oculomotor nerve. In addition, a contributing lid-raising pathway is through a branch of the sympathetic cervical chain, the less dominant Müller's muscle (or superior tarsal muscle); it is a branch that passes from the neck to the upper lung and then upward, ultimately into the ocular chamber.

Ptosis

Weakness of either parasympathetic or the sympathetic control results in some degree of ptosis (from Greek "fall") (Fig. 12.2).

Weakness of the ophthalmic branch of the oculomotor nerve results in drooping of the upper lid. Other signs of oculomotor nerve defects are likely to be present as well: a defect in extraocular movement and dilation of the pupil. The mydriasis is caused by unopposed sympathetic tone. If ptosis is unilateral and an isolated finding, the lesion is probably confined to the ophthalmic branch.

Because of the action of sympathetic control of the levator muscle of the eyelid is subdominant, ptosis from paralysis of the sympathetic cervical chain is relatively mild. In Horner's syndrome, one may find slight ptosis (along with a small pupil) on the affected side. Oculosympathetic palsy may also be an intermittent component of recurring cluster headaches [3]. Recent-onset ptosis should prompt a search for apical lung cancer that interrupts the cervical portion of the sympathetic cervical chain.

Ptosis in both eyes is a sign of systemic illness. As an early sign of botulin toxicity, it is a critical sign of a life-threatening disease [4]. Myasthenia gravis, on the other hand, is a chronic and slowly progressive condition. It is detected by repeated rapid closing of the eyelids that induces or exaggerates palpebral narrowing. Patients with myasthenia gravis have more difficulty holding their eyes open when they are tired.

Fig. 12.2 Decreased palpebral fissure (ptosis) (unilateral, right eye)

Vision

Central Vision

Testing visual acuity is a much-neglected procedure in the routine physical examination. As already stated, testing may be omitted in the patient if there are no symptoms related to the eye, if palpebral fissures, pupils, and sclera appear normal and if there are no systemic diseases such as diabetes that tend to affect the eye. Should any of these findings be present, it is mandatory that a definitive examination for visual acuity be performed. In addition, examinations for certain occupations dictate that visual acuity is evaluated whether or not there are eye symptoms.

Vision is what the eye does! Its acuity can be quickly and reliably evaluated – in fact, within seconds by reading aloud – while providing reliable information related to visual comprehension, all within the context of the medical examination. More precise measurements (those involving charts, various lenses, standardized lighting, and predetermined distances, required for prescribing corrective lenses) fall within the realm of the optometrist.

Focused vision depends upon about 1 mm of highly concentrated cones within the retina in the central macula. It is the perceptive area for color vision and fine detail. Dependent upon a high level of metabolism and high blood flow, central visual acuity and color vision are rapidly compromised with reduced systematic blood flow, as in pre-syncopal events. The first thing to go in a typical hypotension-induced faint is the loss of color, soon followed by loss of vision altogether.

The optics of the eye is also dependent upon concentrations of ions and molecules between refractory surfaces. In uncontrolled diabetes with marked hyperglycemia, the increased optical density within the aqueous humor causes the cornea to refract images to a greater power. In this way, patients with recent onset severe hyperglycemia experience decreased vision in the form of myopia. By contrast, we become extremely farsighted underwater, owing to greatly decreased refraction by a water-to-cornea interface. Goggles worn while swimming merely preserve the air-to-cornea relationship.

Reading Aloud

"Diagnostic" reading aloud can rapidly demonstrate accurately and sensitively many components of seeing and speaking. The method, simply put, is outlined:

1. Hand the patient a sheet of ordinary reading material, the letter size typical of newspaper or magazine print (headlines or text). The print on a box of gloves or facial tissues will do. It must be language appropriate. Using reading glasses, if customary, is encouraged. For patients with significantly impaired vision, larger letter size can usually be found at the examining site.
2. Request in a soft voice that the patient read aloud, starting anywhere. The patient can hold the material at whatever distance is optimal. Proper lighting is essential.
3. Observe that both eyes are directed at the page. There will normally be some degree of convergence and narrowing of the pupils, although these changes are small and often difficult to detect. An inward or outward divergence of either eye during near vision indicates a weakness of an adducting extraocular muscle.
4. After a few seconds of the patient reading, cover one eye with your hand for a few seconds while the patient continues to read. Then move the cover to the other eye for several more words.
5. Listen carefully for subtle abnormalities of articulation, intonation, and fluidity.
6. For further assessment, ask the patient to give some idea of what was read.

This test of reading can be performed within 10–15 s. Its performance is, of course, not applicable if visual acuity is markedly reduced, if the patient is not literate, or if language-appropriate material has not been presented.

The commonly designated summary in medical records "cranial nerves 2–12 intact" does not

reveal the details, although it implies that all these neurological functions had been tested and found normal. Actually, such an evaluation would be too time-consuming for the general physical examination. Writing the statement or one equivalent without having performed all the components is worse. Preferred here is a more descriptive but brief statement that "read aloud = N. – R/L/B" (Normal – Right/Left/Both eyes).

Visual acuity can be normal even when there is significant pathology in the retina. Examples are early macular degeneration and diabetic microaneurysms. Indeed, these conditions can be far advanced by funduscopic examination long before they disrupt visual acuity. The key determinant is where the pathology occurs in the retina and whether or not it involves the central macula. By this principle, even a small area of retinal degeneration or rupture of a venous microaneurysm near the fovea can have a devastation effect on visual acuity. Also, marked degrees of papilledema can develop without any noticeable compromise of eyesight.

Chapter 16, placed after the all the cranial nerves have been covered, could be applied at this point. The "simple" task of reading aloud establishes the ability to see fine print, to interpret these symbols as words, and to articulate them. These tasks bring into action nearly all of the cranial nerves and a huge sampling of the higher intellectual functions. There is a good chance that the perceptive observer/listener gains some appreciation of any pathology of the visual apparatus, of most other cranial nerves, or of cognitive functions during this brief testing. The high sensitivity of reading aloud as a test of cerebration can be demonstrated readily by listening to someone who is even slightly intoxicated from alcohol attempting it.

Peripheral Vision

Peripheral vision can be rapidly determined. Occlude one eye with the back of a hand (or a card) and have the patient fix his or her eye on your nose. With the other hand, place a small light behind one ear of the patient and slowly bring it forward. The patient is instructed to report the instant it is seen. Normally, this stimulus is detected by rods at the nasal outer rim of the retina (the ora serrata). The stimulated pathways cross over at the optic chiasma and lead to the occipital visual cortex. A defect in lateral vision may indicate a lesion affecting the chiasm (such as a pituitary adenoma) or injury to the optic nerve (as by glaucoma). A vascular event within the retina would be pre-chiasmal (a complication of hypertension or diabetes). The most common cause of a peripheral visual defect is post-chiasmal, usually from an infarct of the occipital cortex [5]. Such lesions are more likely to be present in the vertical and oblique fields of vision.

If lateral peripheral vision is intact and if there are no ocular symptoms, assessment of nasal, vertical, and oblique peripheral vision is unlikely to disclose an abnormality. This statement applies only to those patients who have no visual or neurological complaints or findings. Visual field defects are complex; any suggestion of such warrants full visual field testing by perimetry by the specialist.

Pupils

The normal pupil in ordinary ambient light is 4–6 mm across. Pupils may be somewhat larger in the very young and somewhat smaller in the very old. Abnormal size can occur in one or both pupils. A difference in diameter of more than 0.5 mm is significant, indicating a unilateral defect anywhere along the pathway between ciliary body and the brain stem nuclei. If both pupils are either constricted or dilated to a similar degree, the cause is presumably a systemic abnormality.

In a complex system, it is helpful to think of pupillary size as determined by the yin and yang of autonomic control: stimulation of the sympathetic system producing dilation (mydriasis) and stimulation of the parasympathetic system producing constriction (miosis) by way of the oculomotor nerve. Conversely, the absence of sympathetic control leads to constriction; the absence of parasympathetic control causes dilation.

The clinician, having tested lateral peripheral vision, can readily move on to examine the pupils in the single motion. This is accomplished by continuing to bring the light source forward to fall directly on the near pupil from the lateral side. As soon as pupillary constriction is noted, withdraw the light and observe the pupil on the opposite side. If this far pupil is observed dilating, it must have been constricted from stimulation of the near pupil. In this maneuver, both direct and indirect (consensual) pupillary functions have been documented. The sequence of testing for lateral peripheral vision and pupillary reflexes is then applied from the opposite side.

Sometimes, pupillary action is not easily observed, especially in a person with dark irises and where ambient light is bright. If the response to light is questionable, a simple maneuver can be used to accentuate a pupillary constriction. Have the patient close his or her eyes while shining the light directly onto the lid. Within seconds, the pupils will dilate somewhat behind the closed lid. In opening the eyes, constriction is exaggerated in the light-exposed pupil and is thus more easily detected.

Light-Accommodation Interrelation

Pupillary reactions were very important to the physician of an earlier era when complications of syphilis were common. In those days, the clinical presentation of this infection was extremely variable, while its diagnosis by laboratory testing was unavailable. The finding of an "Argyll Robertson pupil" secured an "ah ha" reaction by defining the diagnosis of such a confounding disease. The positive sign is failure of the pupils to contract with direct exposure light but with preservation of constriction during accommodation. Incidentally, the effort back then was entirely academic for the physician of the time had no effective treatment for syphilis.

Accommodation can be assumed to be intact if the pupils constrict with exposure to light. Exceptions to this rule are vanishingly rare. Therefore, there is no practical need to check accommodation when the pupil constricts to light. If, on the other hand, there is no papillary light reflex, it is imperative to determine if the pupils constrict in accommodation. Parenthetically, in addition to syphilis, multiple sclerosis and other demyelinating diseases can produce the Argyll Robertson pupil by injuring the parasympathetic nuclei (the Edinger-Westphal nucleus) that innervate the ciliary body.

Accessing accommodation requires evaluating three specific functions: (1) the eyes converge when looking at an object close up; (2) both pupils constrict during this action; and (3) the patient can read up close. All three should be checked on those rare occasions when the pupils fail to react to light.

Recording

A few words address the ubiquitous acronym "PERRLA", standing for "pupils equal, round, and reactive to light and accommodation." It is a convenient expression for a very complex set of observations that may lead to overuse. For example, the "A," representing accommodation, is often added to the abbreviation habitually without the appropriate tests being performed. Indicating PERRLA without completing all functions places a misstatement in the record. When viewed from a legal perspective, such omission or unintentional untruth could compromise the integrity of the physical examination and, therefore, cast doubt on the validity of all the entries. As mentioned previously, it is not necessary to test for accommodation if there is a pupillary reflex to light – and the "A" in the acronym can be appropriately omitted.

Dilated Pupil

Photographs taken with the camera lens at its smallest aperture are sharpest; they just require more light. Ideally, the image focused on the retina is pinpoint. The image striking the retina becomes more diffuse as the pupil enlarges. One common experience is that focus is reduced in dim ambient light. Reduction in vision occurs in conditions that produce mydriasis, such as in the fundus examination when a local drop is applied to dilate the pupil.

Fig. 12.3 Dilated pupil (unilateral) (left eye)

Unilateral Mydriasis

Suspect a lesion of oculomotor nerve when the pupil is dilated only on one side. Mydriasis is caused by the unopposed tone of sympathetic innervation of the iris. Because of the multiple functions of the oculomotor nerve, ptosis and a defect in lateral movement of the eyes would be commonly associated. Thrombosis of the central retinal artery is another cause of unilateral mydriasis. It may also be a solitary and transient finding from rubbing an eye after contact with a plant containing parasympatholytic alkaloids of the atropine class [6] (Fig. 12.3).

Where the afferent parasympathetic pathway for the light reflex has been compromised, shining a light into the dilated pupil will not induce constriction. The light directed at the normal eye, however, will produce constriction of the dilated pupil. This is the "consensual response," innervated by sympathetic control of the ciliary nerve.

Bilateral Mydriasis

Bilateral dilation of the pupils is assumed to be a systemic reaction from either (1) extreme sympathetic stimuli or (2) parasympathetic paralysis. Each has a wide range of both pathological and pharmacological causes.

1. *Adrenergic stimulation*
   Widened pupils from adrenergic stimuli may arise from strong emotional reactions (such as from fear) or from a host of commonly used drugs. Such drugs include amphetamine and the serotonin-stimulating antidepressants. Cocaine is particularly powerful mydriatic agent.
2. *Cholinergic blockade*
   Pupillary dilation caused by anticholinergic agents can be attributed to any of the atropine-like drugs that are so widely taken for gastrointestinal complaints. Drops used to cause mydriasis for an eye examination are of this class; that is, these "cycloplegics" inhibit the parasympathetic pathways to the ciliary muscles.

Trauma

A more ominous explanation of mydriasis is increased intracranial pressure. When acute following trauma to the head, it is known as the "blown pupil." One or both eyes may be dilated. In a cardiopulmonary resuscitation effort, bilateral dilated pupils that do not react to light stimulation suggest that hypoxic damage to the midbrain control centers has occurred.

Photophobia

Patients with mydriasis – whether if affects one or both eyes – avoid the discomfort of bright light (photophobia). After all, the evolution of readily changeable pupil size is to adapt to the sudden changes in intensity of light and focus, a phenomenon that has not been overlooked in the animal kingdom. The pupil in the form of a slit, for example, opens and closes more rapidly than a rounded pupil, giving an edge to flying or stalking predators.

There are causes of photophobia not all related to pupil size. A preeminent example is the photophobia experienced by the large majority of people with migraine headaches. It may be experienced with or without the headache. The cause is not well understood but enhanced excitability within the visual cortex in part may be responsible.

Constricted Pupil

Narrowing of the palpebral fissure can result from (1) inhibition of adrenergic activity or from (2) overstimulation of the parasympathetic system. Drug reactions are high on the list of bilateral miosis-producing causes.

Unilateral Miosis

Constriction of the pupil in only one eye is most likely the result of disruption somewhere along the sympathetic pathway. The injury may be central (hypothalamic neurons), preganglionic (spinal cord neurons), or postganglionic (superior cervical ganglion neurons).

Unilateral constriction of the pupil most often indicates disruption of sympathetic innervation within the cervical sympathetic chain. Known as "Horner's sign," it generally presents with mild ptosis on that side, as noted in this illustration (Fig. 12.4).

Bilateral Miosis

1. *Adrenergic blockade*
   Bilateral constriction of the pupils can be caused by blunting of adrenergic stimulation. Opioids, such as morphine and heroin, cause extreme miosis, aptly termed "pinpoint" pupils (Fig. 12.5).

   When an overdose of an illicit drug is suspected, finding bilateral miosis implicates an opiate etiology. The observation has a compelling application now that opiate-neutralizing agents are readily available. Lesser degrees of miosis occur from products containing nicotine.
2. *Cholinergic stimulation*
   Agents – such as pilocarpine – are used to narrow the pupils (for the treatment of glaucoma). They enhance parasympathetic tone to the ciliary muscle by blocking acetylcholinesterase, thus protracting the action of acetylcholine.

Ocular Motion

Within the perspective of the routine physical examination, a patient who has no ocular symptoms or difficulty reading and who does not experience vertigo, tinnitus, or neurological symptoms does not require an assessment of extraocular muscle function. Nevertheless, the range of eye movement is frequently tested in the general physical examination.

If done at all, the range of gaze should be performed with accuracy, not casually, with broad

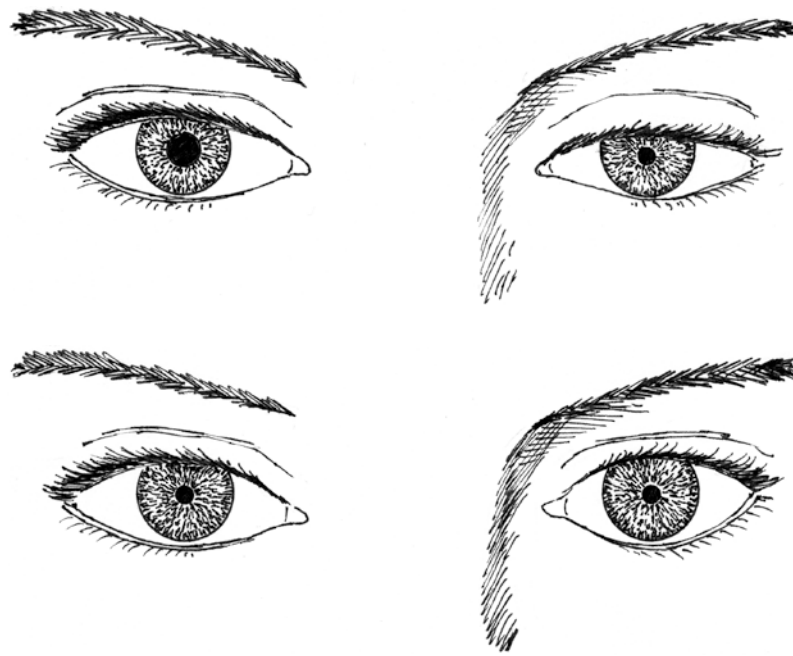

Fig. 12.4 Constricted pupil (unilateral) (Horner's syndrome) (left eye)

Fig. 12.5 Constricted pupils (bilateral)

and slow sweeps of a target. A small penlight point is preferred because it allows the examiner to observe the position of the light reflex on the pupil. The light on an iPhone, incidentally, is a LED; its brightness directly on the eye can cause irritation.

With the patient looking straight at the light head held about a foot away, the light will normally reflect in the center of each pupil. Observe the position of this reflection as the light slowly moves laterally – first in one direction, then the other – throughout the extremes of gaze.

Ophthalmoplegia

Even a slight deviation of the mid-pupillary light reflection from the center indicates some degree of abnormality of extraocular motion. When this evidence of oculomotor muscle weakness happens, the patient loses binocular vision and may experience two images of the light. The second image is often habitually repressed when this abnormality occurs frequently. Terminology for this weakness only on deviations of gaze is "phoria" (exophoria and esophoria). Strabismus (i.e., a deviation of the eyes at rest) is referred to as "tropia" (exotropia and esotropia).

The lateral rectus is responsible for achieving lateral gaze in the eye on that side. The muscle is supplied by the abducens cranial nerve, its only function. The long pathway from this nerve's origin in the pons renders is more susceptible to neuropathic injury, and it is not an uncommon neuropathy in diabetes. Weakness of the lateral rectus muscle is expected to cause limitation of extreme lateral gaze, while the opposite eye, depending on the oculomotor nerve for adduction, exhibits normal traction. When the lateral rectus muscle is severely weakened, the affected eye may turn inward (esotropia) at central gaze.

Nystagmus

For efficiency, the clinician looks for nystagmus as the eyes move through their full range of motion. Normal (or "physiological") nystagmus may be observed as slow undulations at the far range of lateral gaze. The eye on the side of gaze tends to slowly drift toward center and then snaps back to full deviation. Fatigue of the lateral rectus muscle is responsible.

Abnormal nystagmus is generally apparent as the eyes move about half way into the extreme range of lateral gaze. On finding significant nystagmus, vertical and oblique tracking should be performed for completeness. Nystagmus is an important sign associated with vertigo of any cause. This subject is expanded in Chap. 13.

Lid Lag

The opportunity to check for lid lag in the routine examination presents itself here. The test consists of observing for a delay of the upper eyelid to close as it follows the slow downward movement of the eyes. A slight degree of sclera just above the iris will be exposed briefly. It is an important test for ophthalmopathy in Graves' syndrome. While both are usually seen together, the autoimmune disorder causing them has a common but separate etiology. Note that eye complications typical of Graves' disease, including lid lag, sometimes occur without thyroid disease.

Corneas

The transparency of the cornea is truly one of the most remarkable of the infinitely remarkable features of nature. Its curve contributes the major refraction mechanism of the eye, involving transmission of light from air to fluid.

Detection of foreign bodies and infectious opacifications of the cornea are described later. These cornea lesions are included in the magnified view using the ophthalmoscope.

Opacification

While the cornea generally heals rapidly after trivial trauma, some loss of transparency can occur, usually transiently and rarely permanently.

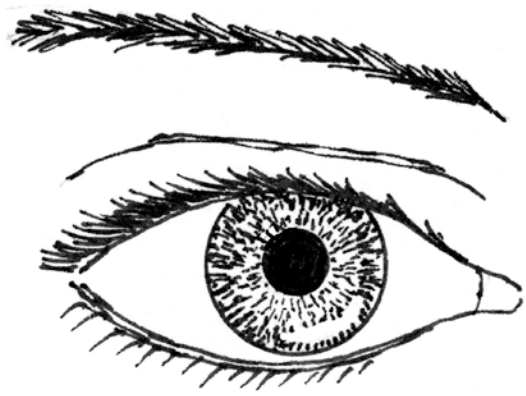

Fig. 12.6 Limbus sign

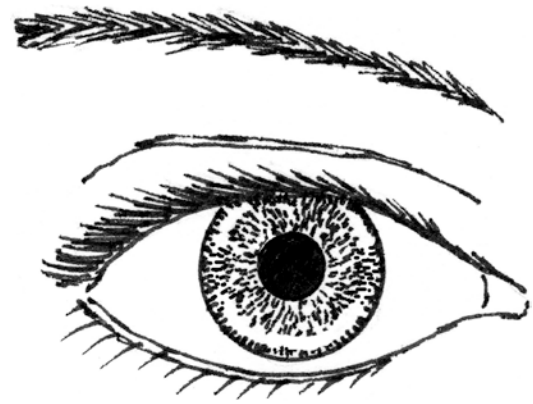

Fig. 12.7 Arcus senilis

Limbus Sign

Virtually any cause of hypercalcemia may lead to deposition of calcium salts at the limbus, that edge of the pupil between the cornea and the sclera. The precipitation, usually along the light-exposed, lower portion of the cornea, is milky white. It is referred to as a "band keratopathy" or "limbus sign." The limbus, anatomically, is a normal, extremely narrow, dark ring around the interior margin of the iris (Fig. 12.6).

Conjunctiva

The conjunctiva lines the sclera (the bulbar portion) and inside the eyelids (the palpebral layer). It is thin and translucent. Conjunctivitis may be caused by viruses, bacteria, allergy, physical contact, and systemic diseases [7]. Because the clinical features of inflammation and infection of this membrane involve the underlying tissue, not the invisible conjunctiva, the subject of conjunctivitis is covered later under section "Sclera."

Iris

Discoloration

Arcus Senilis

Deposition of lipid material at the outer rim of the cornea is common in older people in good health. It is, however, more prevalent in those with hyperlipidemia or hypercholesterolemia. The thin rim seen at the edge of the pupil is white or gray. It usually begins at the upper or lower margin and slowly increases until it forms a complete ring (Fig. 12.7).

Copper Deposit Ring

An inherited disorder in the metabolism of copper leads to a visible and highly diagnostic accumulation of the element in the iris and cornea. It appears as a rust-colored, brownish-green ring at the periphery of the iris, not like the off-white color of arcus senilis. This finding, designated the "Kayser-Fleischer ring," is diagnostic for Wilson's disease. More importantly, deposition of copper in the liver and brain causes jaundice and a wide range of cognitive, behavioral, and movement disorders, problems that are usually apparent in the child or young adult. Identification of the disease early in life can lead to treatment that may ameliorate development of such complications.

Coloboma

A missing part of the iris is clearly evident. The defect may be a notch at the edge of the iris or a slit completely across the iris. Usually congenital, a coloboma (from Greek, meaning "defect") can be bilateral, giving a "cat's eye" appearance. Similarly, a keyhole-shaped defect from cataract extraction surgery was once standard, although newer approaches circumvent the need (Fig. 12.8).

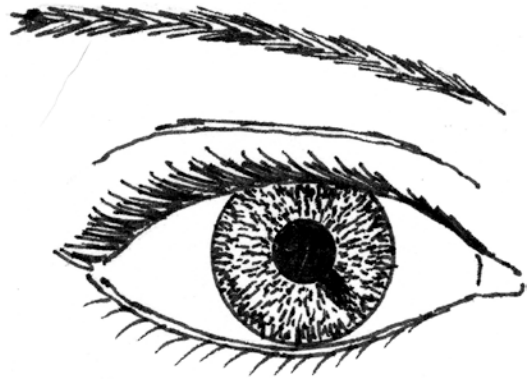

Fig. 12.8 Coloboma

Sclera

Color

In the normal sclera (Greek, for "hard"), the color ranges from pure white to a hint of "eggshell" white. Normal sclera, however, can also be heavily pigmented with melanin. These deposits may be miliary but can also be present in patches.

Jaundice

The sclerae are the most advantageous place to look for jaundice. It may be helpful to have the patient turn the eyes downward while holding up the upper lid. Icteric sclerae can be identified under ideal natural light when the total serum bilirubin is above 2.0 mg/dl. A combination of neon light and infrared light approximates this ideal of "white light." Sometimes mild jaundice is difficult to detect in sclera that are heavily pigmented.

A tinge of green observed in the yellow sclera indicates the presence of biliverdin, a precursor to bilirubin. Its presence in high enough concentration to alter the hue of jaundice suggests long-standing chronic obstructive biliary tract disease.

Mentioned in Chap. 3 is the yellow staining of excessive carotene ingestion. The pigment tends to be deposited in callous tissue, not in the highly elastic tissue of the sclerae.

Blue Sclera

Thinning of the sclera allows the deeper pigment of the uveal apparatus to show through, giving a bluish cast. The red end of the color spectrum emitted by the inner vascular tissue is absorbed as it passes through the thinner outer layer, much like veins in the skin appear blue. The classical example of a disease causing blue sclerae is osteogenesis imperfecta, a defect of collagen synthesis. It is a congenital disease associated with brittle bones prone to fracture, with lax ligaments, and hearing loss. The syndromes of Ehlers-Danlos and Marfan's can also include blue sclerae.

Owing to their deposition of blue-staining pigments or thinning of connective tissue, some drugs are known to cause blue sclera. These include amiodarone, phenothiazines, and minocyclines [8]. Also, the prolonged use of high-dose corticosteroids may be responsible. Silver deposits from silver-containing medications or from industrial exposure can also give the sclera a bluish tinge.

Nodules

Pinguecula is a benign, fleshy growth over the sclera. It consists of degraded collagen and fat-laden tissue deposited into the overlying conjunctiva. The name in Latin refers to it appearing "somewhat fatty." These lesions have no pathological implications nor do they encroach into the pupil (Fig. 12.9).

Pterygium (Greek for "wing") is similar in appearance to a pinguecula, but it is a fibrous

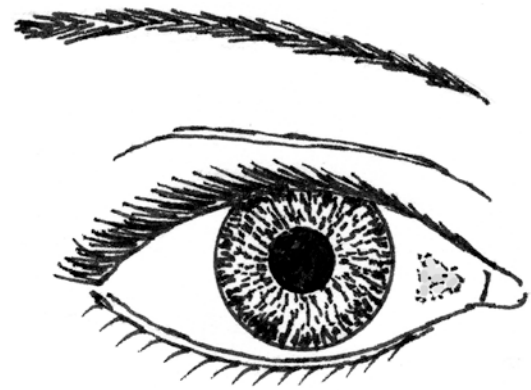

Fig. 12.9 Pinguecula

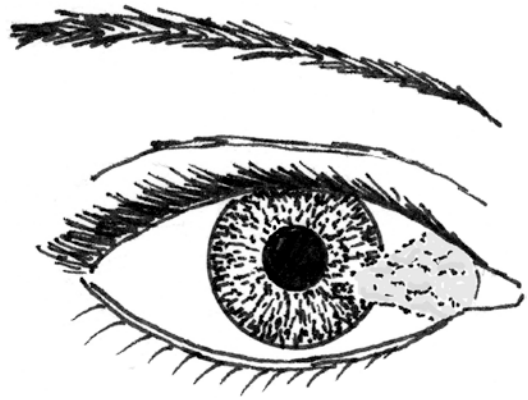

Fig. 12.10 Pterygium

structure with a more obvious vascular bed. It tends to be somewhat triangular in shape with the apex pointed toward the iris. When advanced, a pterygium can extend onto the cornea and affect vision. Otherwise, it is a benign lesion (Fig. 12.10).

Red Eye ("Conjunctivitis")

Dilated blood vessels that supply the sclera give the appearance of "red eye." The condition is usually referred to as "conjunctivitis" although it is the sclera that gives visibility.

There are many causes but the vast majority is totally benign and self-limiting. A few distinctive features, however, hint that there may be serious underpinnings. This determination depends on whether or not visual acuity is reduced.

Conjunctivitis with:

Normal Visual Acuity

Most causes of red eye with normal visual acuity are entirely benign and self-healing, although treatment can relieve symptoms. Finding acuity normal, however, does not always mean the inflammation is benign. Additional evidence by history or physical examination may point to a more serious cause and provide reason for further testing and a specialist's attention. Although they may not require urgent care, some of these more worrisome conditions are noted here.

Allergy

Seasonal conjunctivitis from environmental allergens comprises the vast majority of cases. In addition to "red eye," itching is the preeminent symptom.

Viral Etiology

Viral conjunctivitis (usually from adenovirus) is a highly infectious but generally benign condition. Tears are watery and clear. Dilated arterial branches tend to be in the lateral aspect of the visible globe and do not extend as far as the iris. Pharyngitis and periauricular lymphadenopathy are common cofeatures.

The presence of vesicular lesions around the eye, nose, or ear likely has a viral origin, either herpes zoster or herpes simplex. A herpetic ("snakelike") lesion on the cornea confirms the diagnosis. Urgent ophthalmic management is indicated when vesicular eruptions accompany the signs and symptoms of conjunctivitis – even if visual acuity is normal.

When red eye is coupled with dilated pupils, a possible relationship to cocaine should come to mind.

Bacterial Etiology

In bacterial conjunctivitis, the discharge is purulent. Mattering of eyelids on awakening strongly suggests a bacterial etiology. Other associated symptoms are highly variable. Generally, visual acuity is unaffected; when advanced, however, bacterial conjunctivitis can impair vision. Although infections by *Staphylococcus*, *Streptococcus*, and *Haemophilus* bacteria are the predominant causes, those caused by gonorrhea and chlamydia are generally more aggressive.

Trauma

Injury to the cornea (keratosis) with secondary conjunctivitis is most commonly caused by wearing contact lenses. The explanation may be direct injury or from irritating material in the irrigating solution. *Chlamydia trachomatis* – the leading worldwide cause of blindness – causes scaring of the eyelids, conjunctiva, and cornea. The cause of trachoma is inversion of the eyelids (entropion)

and chronic irritation of the cornea by mechanical irritation by the eyelashes.

Systemic Diseases

Prolonged or recurring conjunctivitis is common in sicca disease (Sjögren's syndrome), Kawasaki disease, and systemic lupus erythematosus.

Cellulitis

Ocular cellulitis, usually a complication of sinusitis, affects not only the conjunctiva but also causes redness and swelling of the lids.

Hemorrhage

The "bloodshot" eye appearing "out of the blue" causes great alarm but is almost always harmless. Coughing or another action that suddenly increases venous pressure is likely responsible for rupturing a small blood vessel within the sclera or the underlying choroid layer. More serious conditions include direct trauma, obstruction of the superior vena cava, or severe hypertension.

Conjunctivitis with:

Reduced Visual Acuity

A recent reduction in visual acuity in the eye with conjunctivitis is a strong warning of intraocular disease. The patient will have noticed that reading material is more blurred in the affected eye.

Acute glaucoma and uveitis (inflammation of the ciliary tract muscle) must be considered as causes of vision-loss conjunctivitis. Arterial dilation in the sclera in these conditions usually extends more centrally, sometimes to the edge of the iris. The findings define an ophthalmic emergency.

In addition to reduced visual acuity with conjunctivitis, reasons for prompt ophthalmological consultation are clouding of the cornea (chemosis), abnormal pupil size or shape (anisocoria), swelling of the eyelids and bulging of the globe (proptosis), the presence of corneal vesicles, severe pain, and conjunctivitis that lasts for several days.

The seriousness of reduced visual acuity in acute eye symptoms is reemphasized. It underscores the importance of having a previous assessment on hand for comparison.

Ophthalmoscopy

Overview

Should the general clinician perform ophthalmoscopy during the routine physical examination? Nonspecialist clinicians may admit that they lack the skills to perform a productive examination of the fundus and find futility in trying. Another view is that appropriate skill can be readily acquired and rapidly performed. The results can prove extremely useful, especially in those patients who are most susceptible to developing retinopathy. As an example, it is well established that many patients with newly diagnosed diabetes type 2 probably have had hyperglycemia for years. During this long asymptomatic period, treatable microvascular complications within the retina could already have developed [9].

Proficiency gained from frequent use of an ophthalmoscope early in one's clinical training can prove a valuable asset throughout a career. Indeed, the ophthalmic examination can become an important tool in the day-to-day practice of a clinician. The basics of a systematic examination can be quickly acquired and effectively applied in the routine physical examination.

It can be performed selectively on patients with diabetes or hypertension and certainly on those with neurologic or ophthalmic disorders.

The ophthalmoscope also provides a handy tool for examining external lesions under magnification that are on the lids, cornea, and sclera. Indeed, the rotating lens system allows the degree of power, in some instruments up to 20 times magnification. With each increase in power, the focal distance between instrument and object moves closer.

For perspective, only the fundamentals of the ophthalmic examination are mentioned here; covering details of a comprehensive examination is well beyond the scope of this text.

Yet, the basic procedure is useful in the clinician's assessment, especially in the diabetic or hypertensive patient. It is imperative for the

patient with obscure neurological complaints and in recent significant head trauma.

It is worth mentioning that the fundus is the only place where one can directly observe functioning arterioles and venules. The fundus also provides the unique opportunity of looking at an extension of the brain. After all, the optic nerve is not a nerve but the visible part of the brain. Nerves start in the retina.

To be reasonably successful at ophthalmoscopy, the technique requires a methodological approach. Good internal visualization of the eye's interior will not happen by just spinning the dial. Further, the exam must be performed in a semi-darkened room, allowing a minute of two for the pupils to dilate naturally. Neither patient nor examiner needs to wear glasses because the instrument lenses, when properly adjusted, will correct for problems of refraction. An exception, however, is severe astigmatism.

There are mixed attitudes about the use of pharmacological agents to render the pupil maximally dilated for optimal viewing of the retina. An attack of acute glaucoma may be precipitated by inducing mydriasis. Some, therefore, argue that a dilated eye examination is only within the realm of the ophthalmic specialist [10]. In a contrary view, others hold that a local mydriatic drug can be safely used by the general clinician [11]. Certainly, the clinician can get a far better view of the retina when the pupil is fully dilated. What approach is most appropriately practiced by the general clinician?

To answer this question, several issues are considered. Fear of precipitating acute glaucoma is probably overstated, although it has a basis in reality. Furthermore, induced mydriasis can be quickly reversed by application of a pupil-constricting topical agent. This approach, in fact, does require that a locally administered counter-agent is on standby for emergency use. Indeed, the onset of signs and symptoms of acute glaucoma induced in this way may be the first indication, alerting the clinician to the diagnosis.

The case for using a pupil-dilating drug for non-specialist's periodic examinations can be supported for patients (1) who are most susceptible to retinopathy and (2) who cannot or will not consult an ophthalmologist. It is particularly important that the diabetic and the prediabetic have the fundus looked at least once a year; twice a year is actually preferable. The retinal lesions that chronic hyperglycemia predisposed to namely, venous aneurysms – can be easily recognized by a well-practiced clinician. An effective technique along with determination to use the instrument is all that is required.

An important consideration when using a mydriatic drug is the aftermath. The dilated pupil increases optical aberration (by diffusing the focus) in the same way a wider lens on a camera does. This effect, which can last an hour or more, sharply reduces visual acuity. The risk of falling from an out-of-focus pathway rises sharply. Certainly, driving is out of question. Either a miotic topical agent to counteract the mydriasis or an escort to help the patient navigate is essential during this period of recovery.

Of course, the use of a mydriatic is contraindicated in cases of acute neurological injury. The size and reactivity of pupils are markers too critical to lose in serial evaluations.

Procedure

It is helpful to place an easily seen marker on a wall directly behind the examiner. It serves as a target for the patient to gaze steadily at a distance. A Band-Aid with an "X" penned on it makes a good, improvised, and wall-friendly marker.

To begin, the dial of the scope is set at *plus* 5 diopters (+5). At this setting, this convex lens will focus parallel beams of light to a point at 20 cm. (By standard terminology, a 1 diopter convex lens has a focal point at 100 cm.) Convex ("plus") lenses on most ophthalmoscopes are indicated by green or black. For the patient without a replacement lens after cataract surgery, begin with the more powerful lens of +10 diopters.

With the scope firmly against the examining eye – the right eye looking at the patient's right

eye – begin at about a foot from the patient and point the light directly on the upper lid margin. Slowly close in until the magnified lid and lashes come into sharp focus. If the examiner has normal visual acuity (i.e., 20/20), the distance will be 20 cm. It will be closer in examiners with myopia. At this distance, the patient's external eye can be examined in detail: lids, sclera, and cornea. Any surface opacity on the otherwise invisible cornea will stand out.

To examine the inner eye, direct the light beam onto the pupil and slowly advance, keeping the lens at 5 diopters and the instrument still firmly pressed against one's own eye.

The aqueous humor and then the lens will come into focus. As the focal point moves beyond the patient's lens, it will add to the instrument's lens and the image will blur. With the index finger used to scroll the wheel, advance the instrument until the middle finger is placed firmly against the cheek of the patient. Note: unless this uncompromising proximity is insured, the retina will be difficult and more likely impossible to visualize. The examination now depends on changing the instrument lenses until the retina is in sharp focus, as described below.

What one has done with this approach is, in effect, visually "dissect" the ocular content. Although these structures are normally invisible, the approach allows the clinician to sequentially pass through one structure at a time, looking for opacities from cornea to the retina.

With some lens changes, described below, the retina can be examined (Fig. 12.11).

Cornea

The +5 diopter lens suggested provides a good magnification to detect "a speck in the eye." Minute foreign bodies over the cornea or subconjunctival surfaces are easily visualized as are those on the turned-up or turned-down inner surface on an eyelid. If greater magnification is desired, turn the lens wheel to +10.

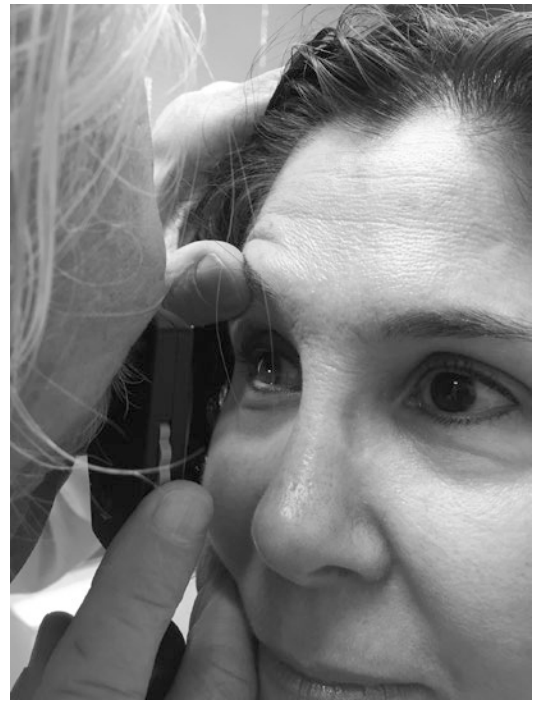

Fig. 12.11 Ophthalmoscopy

Herpes Simplex

Herpes simplex is the most common cause of infectious keratitis. The typical lesion on the cornea is a linear white opacity that can be solitary, multiple, or diffuse. The term "dendritic" is used to describe the lesion because of its tree-like branching. The lesion may be visualized with magnification alone but are best seen after staining with a fluorescein dye. In most cases, the replicating virus affects only the outer cornea (epithelial) and is self-limiting. The virus, however, can invade the deeper cornea (stroma) and result, rarely, in severe scarring (Fig. 12.12).

The differential diagnosis of acute corneal disease includes herpes zoster, *Acanthamoeba* infection, abrasion, and irritation from a topical medication or contamination of contact lens fluid.

Because herpes simplex virus (HSV) can also cause conjunctivitis, the infection may be attributed to ordinary conjunctivitis from the adenovirus, overlooking the primary corneal lesion. Rarely, the acute infection is further com-

Fig. 12.12 Herpes simplex (dendritic lesion)

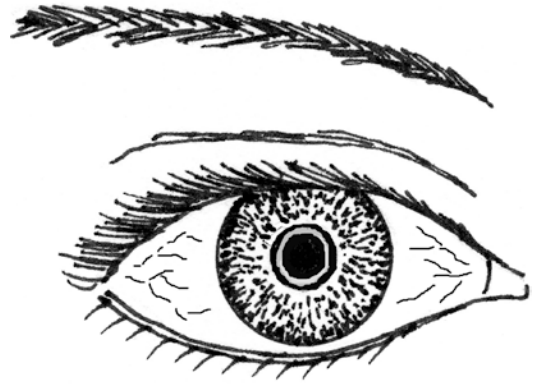

Fig. 12.13 *Acanthamoeba* keratitis

plicated by inflammation of the lids (blepharitis) and uveitis with pain and reduced vision. The need for urgent management of these cases has been stressed.

Herpes Zoster

"Singles" affecting the eye involves the ophthalmic branch of the trigeminal nerve. Typically the keratitis is associated with the vesicular rash with crusting within the dermatome. Punctate lesions may dot the cornea and these in aggregate can produce a branching "pseudodendrite" lesion. A sight-threatening complication can ulcerate the cornea.

While herpes zoster may be confined to the cornea, it usually has a wider distribution in the skin, involving the upper lid, the nose (down to the tip), and the forehead. The skin lesions begin with red spots that, with a few days, evolve into vesicles. These eventually break down and crust over. An important identifying point of herpes zoster is that the rash does not extend beyond midline.

The ophthalmic complications are some of the more common manifestations of the herpes zoster. At times, it involves only a tiny part of the dermatome, confounding diagnosis. When the rash is on one side of the nose, the nasociliary branch of the trigeminal nerve is involved; keratitis is a frequent concurrent manifestation (Hutchinson's sign).

About half the cases of ophthalmic herpes zoster develop uveitis, accompanied by reduced vision, pain, and photophobia. In addition, the infection can affect the optic nerve as well as the extraocular muscles. Clearly, any suggestion of ocular infection by the varicella virus dictates prompt treatment.

Acanthamoeba Keratitis

A common, tap water-borne protozoa can infect the cornea. Almost always, the condition is limited to wearers of contact lenses. Discomfort over the cornea along with diffuse redness of the conjunctiva is a typical feature. In addition, a late sign of corneal irritation and impending ulceration is a ring around the inner margin of the iris (Fig. 12.13).

Lens

Normally invisible, ophthalmoscopy will reveal any part that is opacified. Aging incurs varying degrees of lenticular opacification and is the preeminent cause of cataracts. In long-standing diabetes, cataracts commonly occur prematurely. Various patterns can be identified on ophthalmoscopy. The magnification of the +5 diopter lens will allow a clear view of the cataract. Dense cataracts will prevent getting a clear view of the retina (Fig. 12.14).

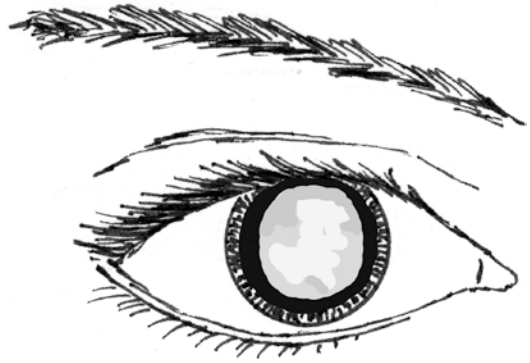

Fig. 12.14 Cataract

Vitreous Humor

Abnormalities of transparency in the vitreous humor may be suspected only by the inability to visualize the retina. Vitreous hemorrhage, producing sudden reduction of visual acuity and massive floaters, is the most serious form and has many causes. Neovascularization – that is, development of new blood vessels into an area of hemorrhage – occurs after a retinal bleed diffuses into the vitreous humor.

Retina

Once direct contact of the examiner's middle finger to the patient's cheek is established, slowly turn the lens wheel with the index finger, reducing the power diopter by diopter until the retinal details are clearly visualized. If both examiner and patient have 20/20 vision, the details of the retina should come into view when the dial reads "zero." If either is hyperopic (far-sighted), focus will occur before the "zero" lens comes into the indicator window. To compensate for myopia (near sightedness) in either person, the wheel continues to move in the same direction, bringing into play the minus (and conventionally red) lenses. Finally, some adjustments back and forth may be necessary to achieve optimum visualization. Since the clarity of focus has priority over the actual number of the lens used, there is no need to bother checking the sequential numbers dialed during the procedure.

Optic Disc

The optic disc is the most conspicuous retinal structure. It is the landmark to first locate. The color is normally slightly yellow. Atrophy of the axons in the optic disc causes them to turn white. Pallor confined to the temporal aspect of the disc is an indication of a degenerative nerve disease, most likely multiple sclerosis.

Next, look at the disc margins. The lateral margin is normally sharp, while the nasal may appear slightly fuzzy. This difference is only because the optic nerve enters the eyeball at an angle directed laterally.

Papilledema

Papilledema obliterates the sharpness of the edges as the optic disc is projected forward. The cause is increased intracranial pressure that pushes the optic disc forward. The center of the bulging disc can be brought into focus by reducing the power of the lens a diopter or two from that in which the periphery of the disc was in focus. A more obvious sign of increased intracranial pressure, however, is blurring of the lateral margin of the disc (Fig. 12.15).

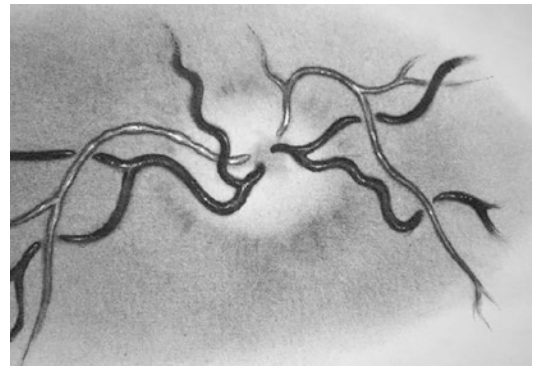

Fig. 12.15 Papilledema

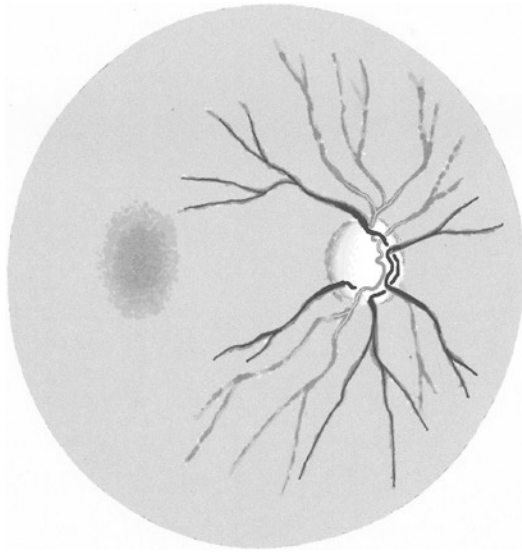

Fig. 12.16 Glaucoma

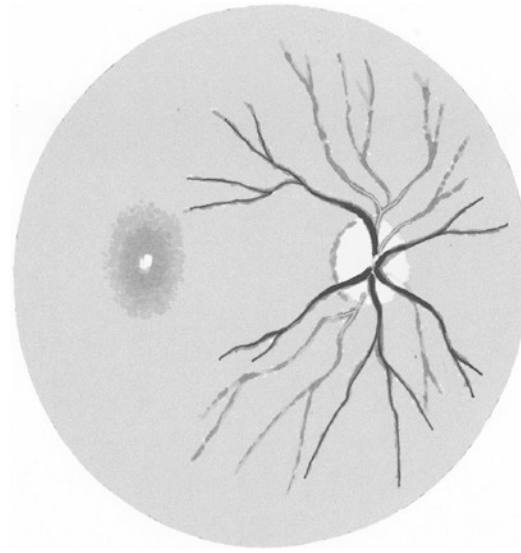

Fig. 12.17 Macula

Glaucoma

In chronic glaucoma, the increased intraorbital pressure pushes the optic disc away. The appearance is typically of very sharp disc margins with a cup-shaped disc surface. Blood vessels appear to emerge abruptly from the disc at the medial edge (Fig. 12.16).

Fundus

Begin by observing the blood vessels as they emerge from the optic disc. At this point, the veins can be seen pulsating. This slight pulsation occurs during cardiac diastole when venous flow increases. The observation is useful in evaluating head injury because the venous pulses disappear when intracranial pressure is increased substantially.

Next, follow the upper nasal blood vessels to the peripheral and then the lower nasal blood vessels. The lateral blood vessels are likewise traced to their visible termination. The lateral ones are examined last because the examiner's head will get in the way of the patient's line of vision to the target.

The critical area for visual acuity is at the macula where there is a high concentration of cones. The macula can be found a distance of two optic disc breadths lateral to the disc. It is a red spot about 0.1 mm across, about the same size as a typical venous aneurysm. The macula is easily differentiated from them, however, by appearing a much brighter red. Also, the patient will experience a much greater sensitivity to light when the beam strikes directly onto the macula. The importance of looking in this area for a microaneurysm in a patient most likely to develop any (i.e., the diabetic) cannot be overemphasized (Fig. 12.17).

Several tiny, light spots may be seen around the macula. Generally, these are white to yellow precipitations of extracellular protein and fat and are not microaneurysms. They are fairly commonly seen in the elderly and do not usually interfere with vision.

At the center of the macula, there is a bright white spot. This is the reflection of the light on the fovea. The fovea is a dimple comprised exclusively of cones. It is the region of the retina responsible for keen eyesight, making reading possible.

One hint for visualizing the fovea is to have the patient look directly into the light. The bright light reflection will pop into view but only for an instant because of discomfort, causing the patient to look away. Before examining the fovea, it is strongly recommended that the intensity of the instrument's light be turned to a dimmed setting. The only point in looking for this central light reflection is to ascertain that the structure is actually the macula and fovea and is not a microaneurysm.

For a look at the extreme periphery of the fundus, the ora serrata, have the patient gaze laterally. Vessels in this area have narrowed to microscopic size and the image, if normal, is an uninterrupted pink field. A microaneurysm in this area can easily be visualized from this angle.

The major vascular abnormalities observable in the fundus are described below. It is prudent to keep in mind that the fundus is a unique opportunity to examine blood vessels of this size and that the same vascular pathologies visualized here are likely taking place in other critical organs. At the same time, we now live in an age when the leading risk factors for their development – diabetes, hypertension, and hyperlipidemia – can be identifiable early and treated effectively.

A-V Nicking

"Nicking" of veins at arteriovenous crossings is a sign of hypertension. The narrowed arterioles associated with hypertension partially obstruct venous flow. The explanation is that the vessels at these junctions share the same connective tissue wrapping (Fig. 12.18).

This illustration also depicts "flame-shaped" hemorrhages, resulting from an arteriolar bleed. In addition, "hard exudates" are present where serum has leaked from the blood vessels. These lesions together (A-V nicking, flame hemorrhages, and hard exudates) are commonly found in severe hypertension.

Arteriolar Hemorrhage

The typical small arteriolar hemorrhage is recognized by its "flame shape." It is widest at the site of rupture and thins out as it extends beyond, taking on the appearance of a candle's flame. The configuration of the dissection tends to follow the linear pattern of neuro-fibers within the retina. If well confined, a small hemorrhage may appear more like a splinter. Retinal hemorrhages can be, however, amorphic, large, and multiple. There is a strong correlation between arteriole hemorrhage and hypertension.

Hard Exudates

Flecks of sharp-edged, deep yellow densities over the retina are deposits of lipid and protein that have leaked out of damaged arterioles. They may have a partial ring-like shape. Vascular disease from diabetes and hypertension are the preeminent causes.

Arteriolar Occlusion

Thrombosis of the central retinal artery produces marked pallor throughout the retina. Of course, visual acuity is lost. Even so, the macular stands out in sharp contrast as "cherry red"; the blood supply of the macula does not depend upon the central retinal artery.

Depicted here is the proximal occlusion of the inferior, lateral branch of an arteriole. Ischemia in the lower lateral quadrant is evident. Microembolism is highly suspected in the obstruction of such a small blood vessel (Fig. 12.19).

Fig. 12.18 Arteriolar-venular nicking

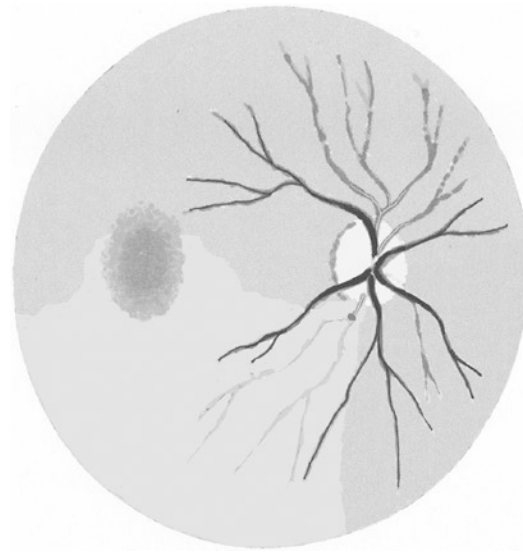

Fig. 12.19 Arteriolar occlusion

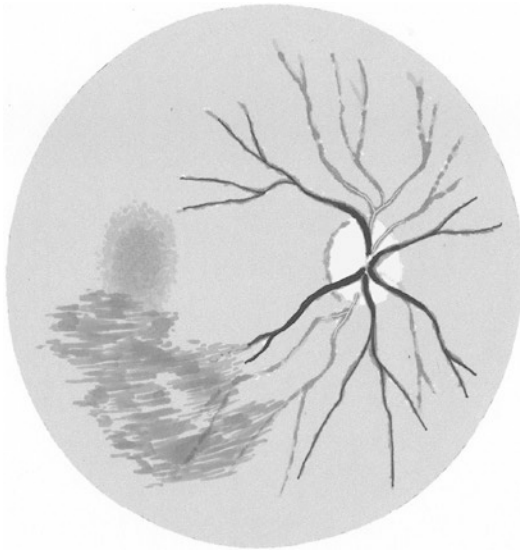

Fig. 12.20 Venule occlusion

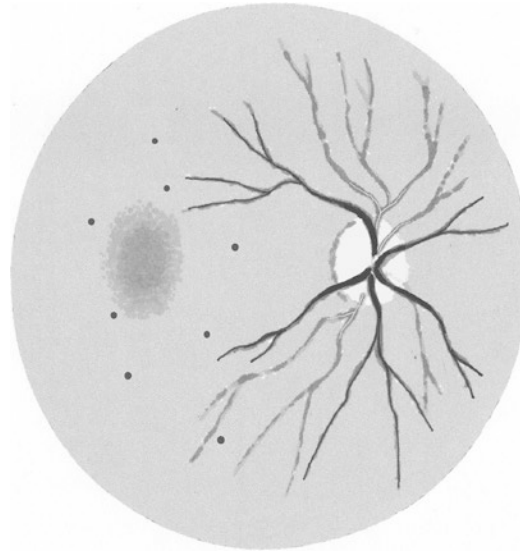

Fig. 12.21 Microaneurysms

Venous Occlusion

Thrombosis of a major venule in the retina, on the other hand, gives a ruddy appearance to the fundus along with engorgement of the vascular bed, tortuosity of the venules, and diffuse points of bleeding. In this illustration, the venular occlusion with its complications occurs in the lateral field. A disorder with hypercoagulation is a likely underlying cause (Fig. 12.20).

The venous systems of patients with hypercoagulable states tend to be dilated and tortuous in the fundus. These venules are most susceptible to occlusion.

Microaneurysms

Microaneurysms are tiny dark red beads that bulge out from capillaries and venules. They can be distinguished from the macula which is of similar size but is a much brighter red (Fig. 12.21).

Most prevalent in diabetes, microaneurysms have the potential to rupture suddenly, dissecting into the retina and into the vitreous humor. The degree of visual acuity lost when they rupture depends upon the size and location of the ensuing hemorrhage. If near the macula, the lost acuity could be devastating and permanent. This complication underscores the desirability of early detection of microaneurysm that can be obliterated with laser photocoagulation.

Cotton Wool Spots

Infarcts of the retinal nerve-fibrin layer tend to produce yellow-whitish, fluffy patches that are known descriptively as "cotton wool spots" or "soft exudates." Mostly found near the optic disc, they are the result of occlusion of precapillary arterioles, causing ischemic damage to the subtended axons. Diabetes is the most common cause although malignant hypertension, connective tissue diseases, and HIV are possible causes of multiple cotton wool spots. They have also been reported in giant cell arteritis [12] (Fig. 12.22).

Retinal Detachment

A detached retina gives a folded-up appearance. Changes in focus occur as the ophthalmoscope sweeps across the site of detachment. Retinal detachment anywhere near the macular will severely compromise vision.

Discovering a venous aneurysm or sign of retinal degeneration in the area of the fovea should lead to prompt intervention by an ophthalmologist. To restate, either condition near the macula may lead overnight to an event that can permanently destroy central vision.

Macular Degeneration

Age-related macular degeneration is the most common worldwide cause of the loss of central

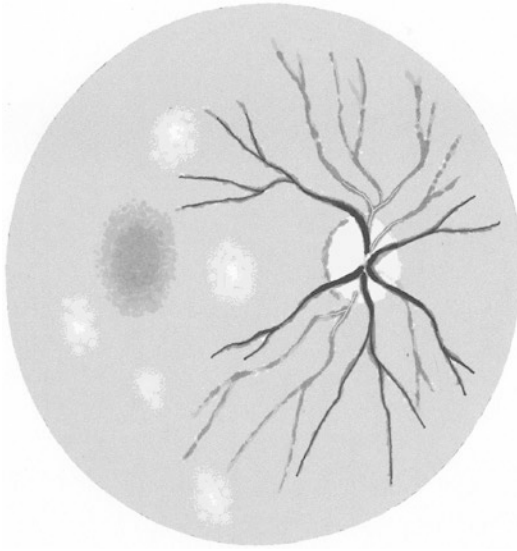

Fig. 12.22 Cotton wool spots

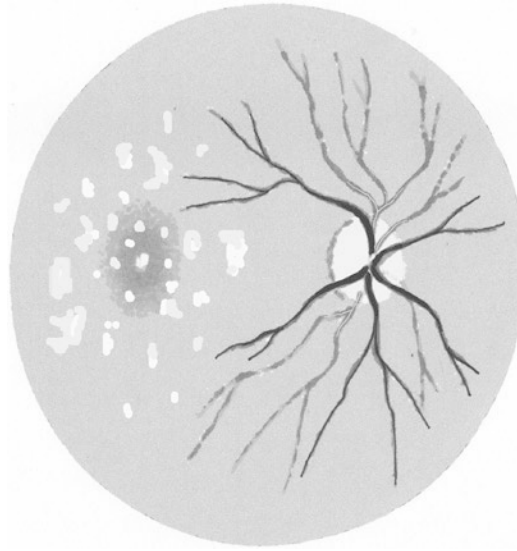

Fig. 12.23 Macular degeneration

vision. It is likely a chronic inflammatory condition in which the epithelial layer of the retina atrophies. Proteinaceous, orange-yellow products of the immune system are deposited in the retina. Pigments of the deposits are released into the light reception layer [13]. As in any retinal lesion, how close and extensive the spots impinge on the macula determines how much critical eyesight will be lost (Fig. 12.23).

Chorioretinal Atrophy

Gaps in the choricoid membrane and the retina expose the white sclera, resulting in patches of white seen in the retina. The choroid is a thin, densely vascular membrane between the sclera and retina that provides nutrition and gaseous exchange to the metabolically highly active cones and rods. In addition to a congenital defect, infections by protozoan or fungus organisms (such as *Toxoplasma gondii* or *Histoplasma capsulatum*) are usually culpable.

Streaks or irregular patterns of white from choricoid thinning can be observed in the fundus without any indication of pathology. This condition, as if a mosaic, is known as the "tessellated retina."

Vitreous Opacity

Hemorrhage into or proliferation of fibrovascular tissue into the vitreous will result in partial or diffuse haziness. The details of the retina will be obscured. Before coming to the conclusion that neovascularization or hemorrhage in the vitreous humor is present, check to make sure that the lens system has been advanced appropriately.

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Ear

13

Essentials

Hearing acuity
Pinnae
Canals
Tympanic membranes

Hearing Acuity

Testing for hearing is often omitted in the routine physical examination, yet it is an important component and one that is quickly performed. Persons with reduced hearing in one ear habitually compensate for it by directing the "good" ear toward a sound source or telephone. Thus, a person may be totally unaware of substantial hearing loss in one ear. Certainly, cochlear defects, acoustic neuromas (Schwann cell tumors), and foreign bodies in the ear canal can reduce hearing on one side even when unsuspected and without associated symptoms.

Truly astonishing is the capacity of the human ear to perceive a wide range of tones and to distinguish complex waveforms, recognizing the difference between a violin note and a tree frog at nearly the same pitch. Audible sound ranges from 20 to 20,000 Hz. The range of normal conversation falls at the low end: 85–150 Hz for men and 250–300 Hz for women. With aging, there is normally a gradual decline in sensitivity for hearing, particularly at the higher register, a process that begins during middle age.

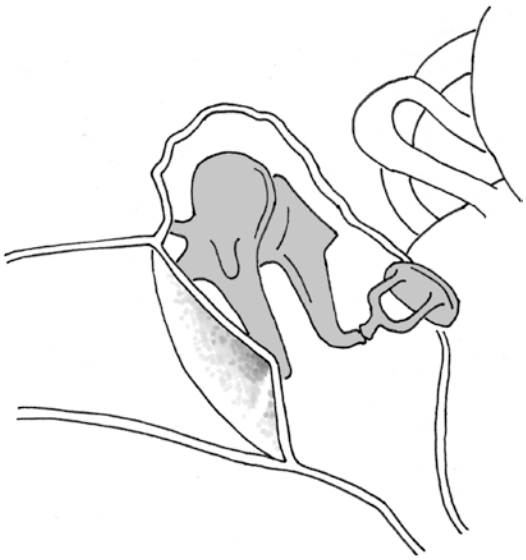

Fig. 13.1 Ossicles of the middle ear

Hz is the symbol for hertz, equal to a wave frequency of one cycle per second. For reference, the lowest note on the standard 88-key piano is 27.5 Hz; the highest note is 4186 Hz. It is the combination of simultaneous vibrations at different frequencies that give any sound its unique character.

A series of three mobile bones – the ossicles – that connect the tympanic membrane to the oval window of the middle ear greatly amplify sound by a lever action. They lie in the middle ear, a chamber connected to the ambient air by the auditory (Eustachian) tube (Fig. 13.1).

R.E. Phillips, *The Physical Exam*, https://doi.org/10.1007/978-3-319-63847-8_13

The club-shaped malleus lies against the tympanic membrane, its long "handle" causing the impression seen with the otoscope. The stapes fits against the oval window and transmits vibrations to the inner ear. The incus connects the two and contributes to the amplification.

The ear is protected against loud noises by reflex contraction of the stapedius, the smallest muscle of the body at 1 mm. The muscle dampens oscillations of the stapes. It is supplied by a branch of the facial nerve. Another muscle, the tensor tympani, attaches to the malleus and further attenuates ear-splitting sounds. Its innervation is the mandibular branch of the trigeminal nerve.

Measuring auditory acuity in the usual clinic or hospital setting can hardly be compared for accuracy with that of visual acuity. Practical limitations are ambient noise and a narrow range of available testing frequencies. Yet, a reasonably useful and rapid assessment for significant hearing loss is possible.

The best one can do is to compare perception of certain tones with one's own. Some examiners prefer to rub fingers for a sound source; others use the whispered voice. While quite acceptable, these methods are difficult to quantify and present a blurry combination of pitches. Preferred here for screening is the pure tone of a tuning fork pitched within the middle range of the spoken voice. The full range of sound can be well documented only by formal audiometry testing.

A stimulated tuning fork vibrating at 128 cps is brought in toward one ear from the lateral distance, and, when perceived, that distance is tested by comparing with the distance of the examiner's own perception of it (presuming that he or she has normal hearing acuity). The direct comparison makes up for any difference in the striking force. Certainly, it is a gross test but is quite adequate for a general physical evaluation. The 128 cps tuning fork is the one with the thick disks at each free end; it doubles handily for testing vibratory sensations in the extremities.

If hearing is normal and if there is no vertigo, tinnitus, or suggestion of imbalance or incoordination, there is usually no need to perform the more time-consuming Weber and Rinne tests. Should reduced acuity for hearing be detected, these specialized tests can help sort out whether a defect is from (1) impairment of conduction of mechanical sound, (2) conversion to and conduction of electrical stimuli, or (3) interpretation of the signals.

1. *Conductive Hearing Loss*

   Impaired transmission of sound waves from the tympanic membrane to the cochlea is considered a conduction defect. The most common cause is "otosclerosis," a stiffening of the auditory ossicles that comes with aging. In addition, impacted earwax, a perforated tympanic membrane and an effusion in the middle ear, reduces the mechanical phase of sound perception. The common cold has such a temporary effect by obstructing the auditory tube.

   In the Weber test, the tip of the tuning fork handle is placed firmly against the center of the patient's forehead. Normally, the intensity of the sound is equal on both sides. A louder sound on one side may indicate that the bone conduction is greater on that side. In effect, the vibrations are transmitted better through the skull than through the stiffened ossicular chain.

   The Rinne test consists of placing the tip of the vibrating fork firmly on the mastoid bone. The sound is then compared with that heard when the fork end is then held close to the ear. A helpful instruction is to say, "Which is louder, number 1 or number 2," timing it so that there is no confusion with the change of position. The normal response of the Rinne test is that air conduction is louder than bone conduction. If hearing is better on direct contact than it is by air, the test indicates a hearing defect of the conduction type on that side.

2. *Sensorineural Hearing Loss*

   Loss of hearing of the "sensorineural" type involves any defect from the hair cells within the cochlear membrane to the conduction pathway of the eighth cranial nerve, the auditory or vestibulocochlear nerve. Sensorineural deafness, by far the most common type reported, is coincident with aging, a gradually declining hearing loss called "presbycusis"

with an incidence approaching half of the senior population. Other causes affecting both ears are infections (especially viral infections), chronic exposure to loud noises, and certain drugs. Aspirin, quinine, and furosemide may cause transient impairment of hearing. Aminoglycoside antibiotics, such as gentamicin, and some anticancer drugs can permanently injury the sensorineural apparatus. If recent and unilateral, suspect trauma (from a skull fracture), tumor (particularly acoustic neuroma), or Ménière's disease (endolymphatic hydrops).

On the Weber test in sensorineural dysfunctions, vibrations from the forehead to the ears radiate loudest to the normal side, thus distinguishing this form of hearing loss from that of the conductive type. The Rinne test in sensorineural deafness reveals better hearing through air than through the bone.

Ménière's disease is most notorious for sudden episodes of violent vertigo, although hearing loss along with tinnitus is the most constant abnormality. The cause is edema within the inner ear. The underlying condition may be pressure-induced membrane stress within the labyrinth [1]. Of uncertain etiology, the condition is unilateral in the majority of cases.

3. *Central Hearing Loss*

Mentioned for completeness, this form of hearing loss is rare and detected not by pure tone reception but from impaired ability to assimilate sounds, particular those related to language. Disorders include vascular or mass lesions of the brain stem or of the auditory centers of the cerebral cortex.

Vertigo

Clinicians often hear the complaint of "feeling dizzy." It is here that a clear definition of the dizziness is of critical importance, separating the sensation of vertigo from the wooziness experienced during pre-syncopal episodes. The first mentioned is inner ear and neurological in origin; the second has a cardiovascular basis. In the emergency ward, getting an MRI of the head in the workup of "dizziness" is not always the appropriate first step.

Vertigo is the illusion of the surroundings moving around oneself or of oneself moving around the surroundings. The symptom from dysfunction of the labyrinthine canals is usually accompanied by nystagmus. In the great majority of cases, vertigo is related to a sudden change in head position and is referred to as "benign paroxysmal positional vertigo" [2]. The mechanism is presumed to be from the movement of calcium debris in the semicircular canals, most likely to occur after suddenly turning or getting up after being recumbent in one position for a long time. Pills for insomnia or alcohol at bedtime can induce prolonged immobile sleep that favors accumulation of debris in the position-dependent area of the semicircular canals.

Pre-syncopal symptoms, on the other hand, may be described as "light-headedness" or "giddiness," as if one were about to faint. Distinguish with near certainty the difference between vertigo and light-headedness by referring to experiences almost anyone has had. Vertigo: getting off the merry-go-round or twisted swing. Light-headedness: suddenly standing after being overly warmed by sitting or lying in the sun for a long time, causing diffuse cutaneous vasodilation. A typical early symptom of near fainting is being aware of losing color vision owing to the high metabolic demands of the retinal cones. Vision turning black announces impending syncope. These pre-syncopal sensations, arising from a temporarily insufficient cerebral blood flow, have a cardiovascular basis.

Imbalance

There is another symptom that a patient may describe as "dizziness." That is a sense of imbalance, especially when walking in poorly lighted areas. The symptom is virtually confined to the elderly who have compromised functions in any combination of coordination, proprioception, and vision.

External Ears

Certainly, the most frequent cause of inflammation of the pinnae is either infection or local allergy from earrings.

Two kinds of bumps that may appear in the pinnae are deposits of urate crystals in tophaceous gout and nodules in rheumatoid arthritis. Others of lesser consequence are sebaceous cysts and dermatitis affecting the underlying cartilage.

Canals

Cerumen

Other than small accumulations of cerumen, the ear canal should be clear. Trauma from habitual insertion of improvised instruments to remove wax is the greatest threat to its delicate surface. Cerumen, by engulfing particulates in the air, prevents them from piling up at the eardrum; it is formed in the outer portion of the canal. Indeed, cerumen pressing against the tympanic membrane indicates that it has been mechanically impacted. A cotton-tipped swab is usually involved.

Erythema

The ear canals are exceptionally sensitive. Even a slight break in the skin or an early furuncle can produce marked discomfort. It is also important to remember that complaint of an "earache" may actually be referred pain from an impacted wisdom tooth or from a malocclusion of the jaw, producing the temporal-mandibular syndrome.

Ear canal erythema alone or complicated by an excoriating dermatitis may be an allergic response, often to the nickel in earrings, to hair spray, or instilled medications. Of the latter, neomycin is particularly notorious. "Swimmer's ear" is otitis externa from bacterial invasion of the canal epithelium promoted by prolonged aquatic activity [3]. Infection in a non-traumatized ear canal is rare; *Pseudomonas* organism is most frequently the culpable organism. In one clinical practice study, the most common presentation of otitis external was a mixed infection of bacteria and fungi. Staphylococci and *Bacillus subtilis* (the hay or grass bacterium) are the most frequently isolated organisms [4]. Herpes zoster can produce a localized dermatitis in the ear canal (Ramsay Hunt syndrome); the eruption may involve only the canal although there are usually telltale vesicles in other branches of the facial nerve [5].

Hemorrhage

Blood in the ear canal after blunt head trauma points to a fracture of the skull, most especially of the parietal bone. Even a drop of blood in the ear canal following trauma to the head is a significant clue.

Bulges

Bulges along the ear canals can be caused by anything from cartilage or bony overgrowths to cysts or hemangiomas to carcinoma. Definitive imaging studies should be initiated to resolve any uncertainty.

Foreign Bodies

Foreign bodies are not infrequently found in the ear canal, particularly in children but certainly not restricted to children. An insect, particles from a towel, or the eraser from the end of a pencil are examples that may give a sense of "water in the ear." Sometimes a foreign body will be unrecognizable owing to it being encased in a waxy mass. A patient may adapt to years of hearing difficulty on that side simply by favoring the other ear for direct or telephone conversation.

Tympanic Membrane

The normal eardrum has a translucent, pearly white appearance. It forms the outer limit of the air-filled middle ear, the oval window sealing the inner aspect. The manubrium ("handle") of the malleus ("hammer") is seen, causing the tympanic

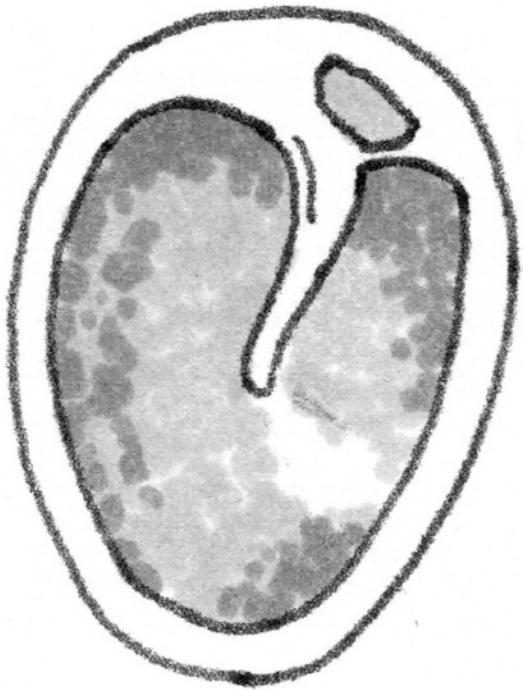

Fig. 13.2 Tympanic membrane, right ear

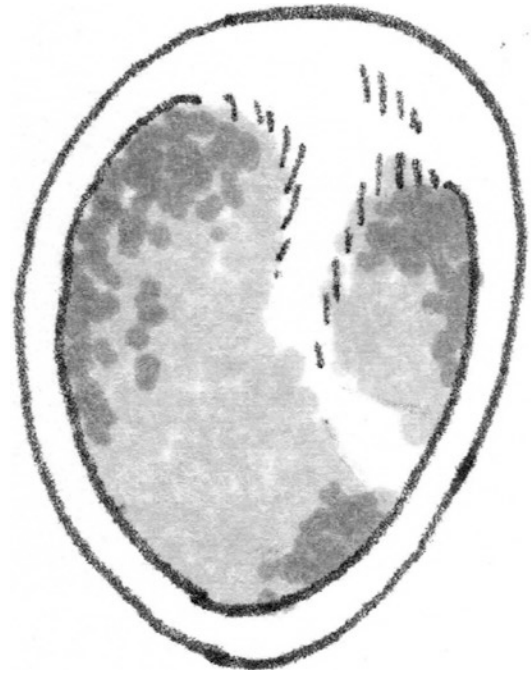

Fig. 13.3 Otitis media

membrane to bulge out. The outline of the malleus projects in the perimeter of the eardrum at about 1 o'clock and extends downward and posterior to the central portion (Fig. 13.2).

Color

The normal tympanic membrane is translucent and a pale gray. Usually, there will be a reflection of the examiner's light.

Erythema

Erythema of the eardrum along with earache does not necessarily indicate infection. It represents massive dilation of blood vessels that line the drum. Significant differences between ambient pressure and that of the middle ear cause erythema. It results from obstruction of the air vent on the auditory (Eustachian) tube. The common cold is almost always responsible by causing generalized swelling of the nasal and oropharyngeal mucus membranes and blocking exchanges between the middle ear and ambient air. The air entrapped in the middle ear is absorbed into the circulation, and the negative pressure is gradually resolved by infusion of fluid into the middle ear.

Even before erythema of the eardrum occurs, edema may cause the loss of definition of the malleus "handle." Bulging of the drum is a late finding consistent with accumulation of fluid within the inner ear caused by closure of the auditory tube (Fig. 13.3).

The red eardrum is likely to be acute otitis media, the most common reason that a pediatrician sees a "walk-in" urgent case. Earache, fever, and irritability often accompany the illness.

Yellow

An effusion in the middle ear casts a yellowish hue to the eardrum, sometimes with a bluish tint. Recent guidelines for evaluation of otitis media with effusion (OME) have been published [6]. These include determining the mobility of the eardrum with pneumatic otoscopy; it is impaired in otitis media with effusion.

The effusion can be serous or purulent. When hemorrhagic, it darkens the eardrum. A blood

level may be evident. The initiating problem is usually persistent inflammation from acute otitis media or from barotrauma from sudden and drastic changes in ambient pressure. Scuba divers sustain a high incidence of barotrauma to the middle ear. Reduced hearing from these problems is of the conductive type.

White Lumps

A cholesteatoma is white to gray mass visible as if it were pushing from behind the tympanic membrane or often just above it (in the "attic"). Cyst-like, its size cannot be predicted from the otoscopic view. Although a benign growth, a cholesteatoma tends to be invasive and to erode neighboring structures such at the ossicles and the labyrinth, affecting hearing and balance. When infected, it can erode into the ear canal and release a foul-smelling yet painless discharge. Discovering what appears to be a cholesteatoma merits prompt further evaluation.

Auditory Tube Dysfunction

To assess the patency of the auditory (or Eustachian) tube from retro-pharynx to the middle ear, have the patient perform Valsalva's maneuver. This test is recommended in patients who complain of earache or fullness in the ear or – as often expressed – "water in the ear." These symptoms most often stem from obstruction of the auditory tube. Bulging of the drum observed during Valsalva's maneuver indicates a patent auditory tube; it does not move when the auditory tube is occluded. For optimal viewing, it is important that the patient perform this maneuver while keeping the head steady.

Sometimes, when performing a diagnostic Valsalva's maneuver, a patient who has ear discomfort will feel a popping on that side on, followed by a clearing of the symptom. This result is particularly common among those who have an upper respiratory infection or who have recently been Scuba diving or on an airplane flight. It is one of those rarities in medicine when an intervention meant for diagnosis turns out to also be therapeutic. Divers and pilots of high-performance aircraft frequently apply Valsalva's maneuver as they descend rapidly into increasing ambient pressure. The rationale behind this technique is to force air from the pharynx through the auditory tube and into the middle ear, thereby equalizing pressure. The oropharyngeal opening of this tube is floppy; that is, it allows air from the middle ear to pass freely out of the tube but is slow to let air enter.

To restate, failure of the tympanic membrane to move on Valsalva's maneuver indicates a blocked auditory tube. An alternate explanation, perforation of the eardrum, should be obvious on otoscopy.

Perforation

Perforation of the tympanic membrane usually occurs at the outer margin, particularly the inferior aspect. Some loss of hearing can be expected, the degree depending upon the location and size of the perforation as well as any ancillary damage to the ear. Valsalva's maneuver will not cause the tympanic membrane to bulge if there is a perforation (Fig. 13.4).

Chronic or recurring infections are the most common causes of a perforated eardrum. Otherwise, trauma from explosion, slapping, or

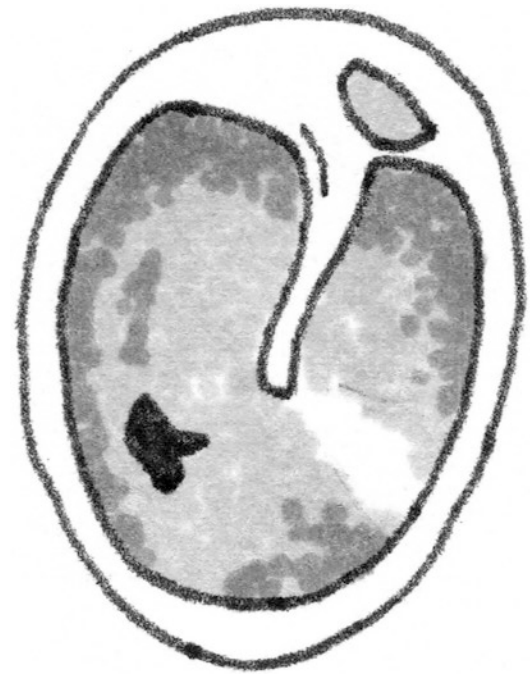

Fig. 13.4 Perforation of the eardrum

probing the canal with a sharp object must be considered. More than half those struck by lightning will incur a ruptured eardrum, either from the shock of compression or from explosive heat-expanded air. The injury can cause disarticulation of the ossicular chain [7]. Stunned survivors may be left with only blood in the ear canal and deafness of the conduction type.

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Nose

14

Essentials
Shape
[Smell]
Airway
Septum

The nose gets little attention in the "routine" medical examination. Nevertheless, there are some meaningful observations that can be quickly accomplished on this remarkable organ.

What is the nose for? Any schoolchild will answer "to smell." The answer is, of course, correct. Indeed, a good nose can detect millions of distinct odors. While the tongue distinguishes only a few basic tastes, it is smell that adds the infinite variety and subtleties to our perception of flavor. What makes a "good" smell is what we associate with pleasure: an ocean breeze, mom's apple pie, and the inside of a new car. It is a sensitive smoke alarm. A "bad" smell is one that has – perhaps long ago – caused us displeasure, remembered or not.

Yet the nose does far more than smell; it serves as a multipurpose "air conditioner" for the lung. This appliance consists of the external nose and, inside, a large chamber that extends to the back of the throat. The lateral walls contain the turbinates that greatly increase both the surface area and the air-to-membrane contact by causing turbulence of moving air (Fig. 14.1).

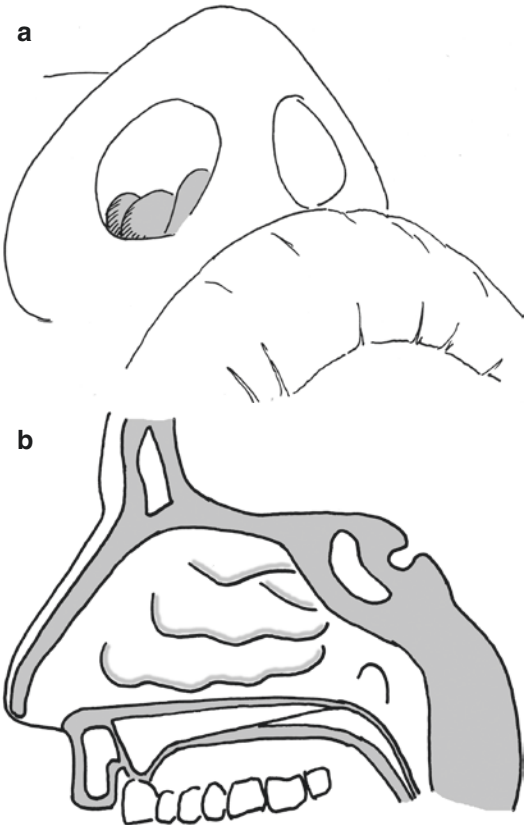

Fig.14.1 (**a**, **b**) Nasal turbinates

Openings in the chamber lead to tubes that connect with air pockets in the sinuses (the ostia) and with the middle ear (auditory or Eustachian tube). They allow the pockets to remain at or near atmospheric pressure.

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Aerosol

With each breath, the nose processes inspired air in three important ways:

Air Filter

A tangle of coarse hair just inside the nostrils traps large particles, such as those suspended in smoke. Farther on in, the air washes along the highly vascular nasal membranes. The ciliated epithelial cells lie on a membrane that presents the dense and spongy layer of tiny blood vessels; these bring the circulating blood into close contact with the air.

Specialized cells within the nasal membranes secrete a constant flow of mucus that is quite thin and watery (*serous glands*). Others, stimulated by inflammation, produce more viscous mucus (*goblet cells*). Small aerosolized particles become mired within the secretions. Cilia – as many as 50–200 – crowd the exposed surface of a single cell on the mucosal lining. These provide a continuous waving motion that propels the layer of mucus outward, just as a wide conveyor belt would move a blanket; it is referred to as "the mucociliary escalator."

In addition, the vascular membrane delivers white blood cells that are called into a defensive action against invading microorganisms. These neutralize pathogens while absorbing and carrying away many of the products of inflammation.

Humidifier

By the time the air inspired by the nose reaches the windpipe, it is virtually 100% humidified. The high moisture content of air that reaches the lung is critical for promoting the exchange of gases. Humidification is so efficient that drying of the respiratory tract is seldom a problem regardless of the moisture content of ambient air or from the heavy breathing of prolonged exertion.

Temperature Control

When cold air passes through the nose, it warms rapidly as it comes into contact with the mucous membrane. On reaching the major airways within the chest, the air comes to within a few degrees of body temperature, even in frigid weather. Warm air contains much more water vapor than cold air and thus is able to maintain moisturizing the airways more effectively. The lungs receive filtered air that has been warmed to body temperature and is fully saturated with water.

These functions are accomplished 15–20 times a minute. Three ridges along the lateral nasal chamber, the turbinates, increase the surface area of the vascular membrane to react with passing air. The name is apt since the contour induces turbulence on the passing air, thereby augmenting the air-to-nasal mucosal exposure. Airborne particles do not make these sharp turns as readily as does air in which they are suspended; they brush against the membranes and adhere to the sticky surface. In addition, the turbinates greatly enlarge the surface area to which passing air is exposed. It is the middle turbinate, incidentally, that is mostly visible on the otoscopic examination.

How the nose adapts to serve as an air processer is clearly demonstrated in nature. The wolf that hunts by running in pursuit of large prey for hours and even days has a long snout. It provides an extensive exposure of nasal mucosa. The cat that has practically no external nose depends upon short bursts of energy for running down prey; it does not need such a broad air-to-nasal membrane contact.

Smell

Should the sense of smell be tested in a routine physical examination? Emphatically no! There are some pressing indications, however, for including the sense of smell, noted below. Chemical messages from vaporized food and drink are received by the olfactory nerve endings that are spread out along the cribriform plate. The signals are then conveyed to the olfactory center of the cerebral cortex that lies just above the palate. Here, the subtleties of flavor are sorted out while in communication with cortical centers for memory and emotion.

Hyposmia/Anosmia

Smell should be evaluated when a patient complains of the "loss of taste" other than that coming with the common cold. Handy and familiar items for testing are coffee, pepper, liquid soap, and alcohol wipe. Admittedly, such testing is perfunctory considering the seemingly infinite number of smells that the normal nose can detect. Lack of recognition of these basic stimuli, however, documents a major disruption of the olfactory system. The defect can be anywhere from the receptors of the olfactory nerve to the cerebral cortex and be caused by infection, degenerative or demyelinating diseases, neoplasm, or injury.

Another indication for testing smell is a recent blunt trauma to the face. Abrupt jarring of the brain across the cribriform plate can shear off any number of the millions of olfactory nerve endings. A subsequent clear fluid discharge from the nose after a head injury suggests rhinorrhea from cerebrospinal fluid. This complication is described below under section "Color."

Upper respiratory infections typically cause a temporary loss of smell, although the antibiotics prescribed to "treat" them may actually be responsible [1]. Rarely, a drug-induced loss of smell is permanent. There is also some reduction of smell sensitivity with aging. Chronic diseases – from Parkinsonism and allergic rhinitis to HIV – may be contributing conditions. In a clinic dedicated to disorders of taste and smell, the most frequent complaints of hyposmia followed bouts of influenza [2].

The inveterate tobacco smoker loses much of the sense of smell. The physical examination could be an opportunity to point out this deficit to such a patient. The demonstration would certain add hard evidence to bring home the customary advice to "stop smoking."

Appearance

Shape/Size

The skeleton of the nose consists of projections of the ethmoid bone from which the cartilage extends farther out. In the skin, precapillary blood vessels dilate readily with a flush in response to spicy foods, hot beverages, alcohol, and emotional reactions. Sebaceous glands are abundant and mostly concentrated near the tip.

Noses come in an infinite variety of shapes and sizes. Racial differences in flatness or pointedness of the nose have no known functional meaning, but they may have something to do with evolutionary adaptations to a cold or hot climate.

A collapsed bridge from obvious trauma (the "boxer's nose") should not present a diagnostic challenge. When it is acute and unexplained by injury, however, the clinician must consider the possibility of a cartilage-destroying disease. Most serious is granulomatosis with polyangiitis (GWP, formerly Wegener's granulomatosis). This is an ANCA-related autoimmune disease that causes small- and medium-sized blood vessels in predisposed sites to rupture or occlude.

The great majority of cases of granulomatosis with polyangiitis have a respiratory component that is found in the upper respiratory tract in virtually all cases [3]. Typical are intermittent or chronic sinusitis and rhinitis with a foul-smelling mucous discharge, excessive crusting of the nose, and epistaxis. Spontaneous collapse of the intranasal cartilage with a "saddle-nose" deformity should bring a high level of suspicion of GWP and lead to a look for ancillary complications. Prominent among these are fever, scleritis, uveitis, and pulmonary symptoms. Scarring of the tracheal (subglottic stenosis) from disease of cartilage can produce cough and hemoptysis or exertional dyspnea and stridor [4].

Rosacea

Rosacea is a persistent flush in the nose that tends to spread laterally to the cheeks and eyelids. Sebaceous glands are hyperactive, leading to tiny pustules. These lesions differ from typical "teenage" acne by not having comedos. Telangiectasias and thickening of the skin complicate the picture in more advanced cases.

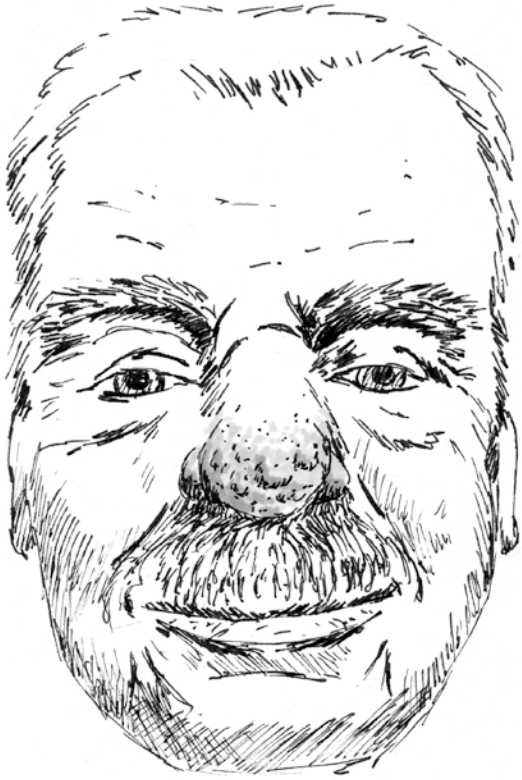

Fig. 14.2 Rhinophyma

Rhinophyma

The bulbous disfigured nose appears to be an exaggerated form of rosacea with fibrous hyperplasia of sebaceous glands. The enlargement can be enough to interfere with breathing (Fig. 14.2).

Historically, rhinophyma has been attributed to excessive alcohol consumption. While it is undeniably true that alcohol can induce a nasal flush, there is no evidence that this opinion is more than a myth [5, 6].

Airway

Our natural aversion to looking up peoples' noses – even when nasal symptoms are present – may lead to a regrettable oversight. Yet, valuable information can be gathered even with a quick look. While inspecting the vestibule with a nasal applicator tip on the otoscope, ask the patient to breathe through his or her mouth. This tactic will prevent the lens of the scope from fogging up as typically occurs with nasal breathing.

Color

The clinician becomes familiar with the pink color of normal nasal mucosa only by repeated practice. This skill is helpful in evaluating the causes of rhinitis.

Rhinitis

Rhinitis from a common cold produces erythema of the nasal mucosa. Allergic rhinitis tends to cause pallor of these membranes.

Color of the mucus discharge in rhinitis receives much attention. There is a common belief that it turns from white yellow to green when bacteria have superseded the viral infection. It is used as a sign to begin antibiotic therapy. Actually, the green discoloration of nasal discharge, occurring in the late stage of a common cold, is from enzymes that are in sacrificed neutrophils in shredded mucosa. The change occurring as the cold develops does not mean that a bacterial infection is complicating the viral illness.

Clear fluid dripping from the nose after a head injury suggests a cerebrospinal fluid origin. A fracture at the base of the skull may have torn the dura, resulting in a persistent leakage. The discharge may be unilateral.

Sicca

Nasal mucosa that is dry suggests excessive application of topical vasoconstrictor drugs used as decongestants. Rarely, the cause is one of the sicca syndromes in which dryness will also be noted in the eyes and the oropharynx.

Polyp

A polyp hanging down from the upper nasal vestibule may be seen partially blocking the

nares. Nasal polyps are soft and non-tender; they are not precancerous. Although they are usually asymptomatic, one that is easily visible probably causes some degree of nasal congestion. When large, they are a source of a persistently stuffed nose.

Chronic rhinosinusitis and nasal polyps are frequently associated. There is also a strong correlation between the incidences of nasal polyps and bronchial asthma [7]. It is recommended that intranasal inspection be part of every examination of patients with chronic lung diseases. About half of the patients with cystic fibrosis have nasal polyposis beginning in childhood [8]. A substantial number of patients with nasal polyps are intolerant to aspirin [9] (Fig. 14.3).

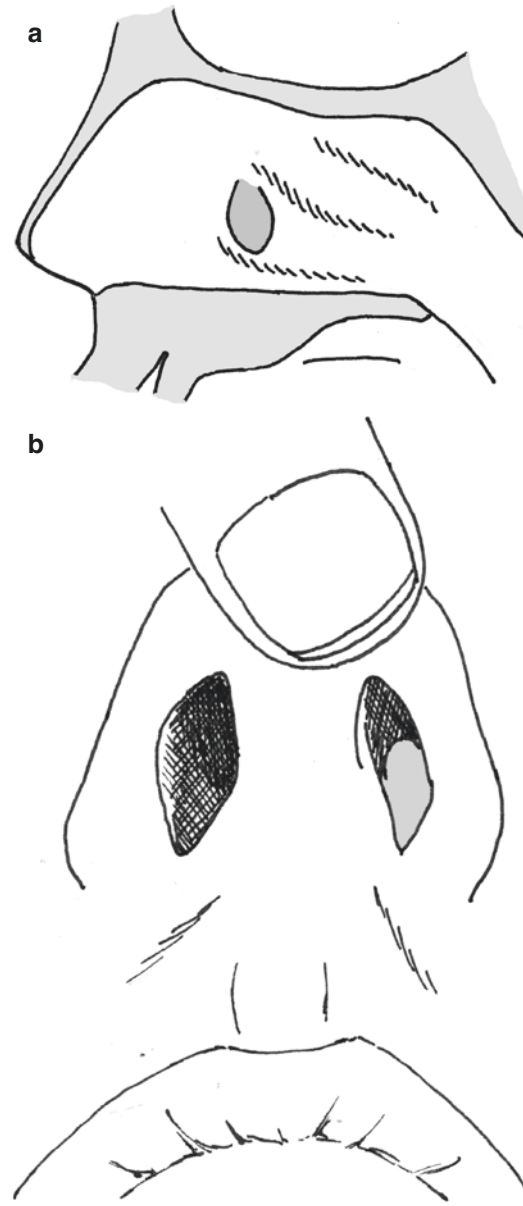

Fig. 14.3 (**a**) Polyps: sagittal. (**b**) Polyps: frontal

Septal Deviation/Perforation

Deviation of the nasal septum is best detected by transillumination. The technique simply involves looking into one nostril with the examining light directed into the other nostril. A perforated septum is easily spotted in this way. A minor degree of asymmetry, often seen, is quite typical normally (Fig. 14.4).

Perforation of the septum can occur with any cartilage-wasting condition, including granulomatosis with polyangiitis, leprosy, and spectrum of connective tissue diseases. Overuse of nasal drops and sprays that induce vasoconstriction pose a hazard leading to chronic rhinitis, including perforation of the nasal septum. The complication is referred to as "rhinitis medicamentosa." Habitual trauma from nose-picking (rhinotillexis) is an additional etiology.

Cocaine has been shown to produce extensive destruction of cartilage in the nasal cavity by intensive vasoconstriction and necrotizing inflammation [10].

Congestion of Sinus and Middle Ear

The chambers of the facial sinuses as well as the middle ear require free movement of air within the nasal vestibule. Congestion of the nasal mucosa from the common cold tends to block the ostia and auditory canal, resulting in interruption of the mucociliary escalator and leading to acute sinusitis or otitis media. So frequent is some degree of sinusitis (especially of the maxillary sinus) during viral upper respiratory infections that the term "rhinosinusitis" is more appropriate that simple "rhinitis."

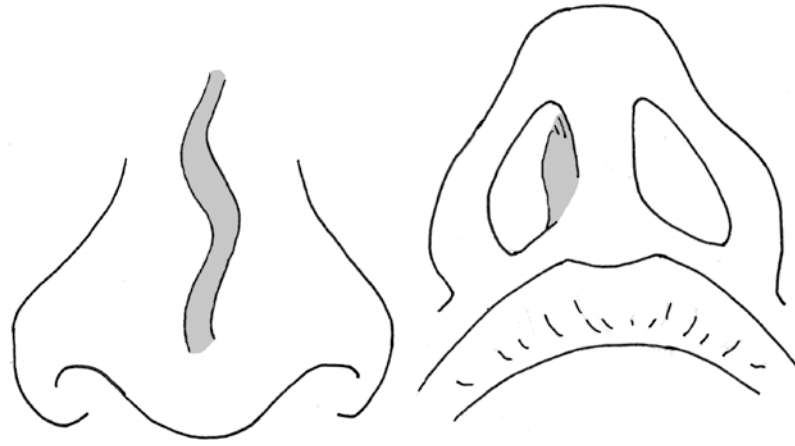

Fig. 14.4 Septal deviation

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Mouth

15

The oropharynx, of course, is a complex structure having several anatomical components with highly integrated functions. Yet, despite the complexity, they can be evaluated rapidly and with reasonable thoroughness by a well-organized approach.

Lips

The dense vascularity of the lips provides a sensitive indicator of vasomotor tone and volume and the oxygenation of blood.

Edema

Swelling of the lips is a frequent early symptom of an allergic reaction known as "angioedema." On first appearing, it is not possible to predict how extensive the process will develop. Certainly, sudden edema of the lips could be an early warning sign of an anaphylactic reaction, in which case angioedema would be expected to spread to the throat within minutes [1]. A change in voice or difficulty in swallowing and/or breathing indicates a full-blown emergency. Despite the myriad benign causes of swollen lips, the prudent clinician will first consider the possibility of anaphylaxis when it is of recent onset.

Vesicles

The "cold sore" or "fever blister" around the lips is typical of an infection with herpes simplex virus (HSV). It evolves from a painful cluster of vesicles on an erythematous patch. The vesicles excoriate within a few days, forming a crusted ulcer before healing completely. Herpes labialis may be associated with fever and myalgia.

Cheilitis

Cheilitis is most familiar as the dry, wrinkled, and cracked lips resulting from extensive sun and wind exposure. Inflammation along the vermilion border with crusted erosions can be painful. Other causes of chapped lips include habitual lip-licking, contact or atopic dermatitis, nutritional deficiencies, and infection. All these may lead to fissures, scales, and crusts. Poorly fitted complete or partial dentures with improper vertical dimensions also cause cheilitis. "Flutist's cheilitis" calls attention to a contact hypersensitivity to metal.

Fairly common among the elderly – and particularly the edentulous – is overlapping of the skin at the corners of the mouth. Called "angular stomatitis," the friction of surfaces in a warm and moist environment promotes intertrigo and infection (bacterial or fungal) from chronic maceration.

R.E. Phillips, *The Physical Exam*, https://doi.org/10.1007/978-3-319-63847-8_15

Chancre

Chancres from primary syphilis can appear on the lips [2]. They are typically a round reddish ulcer with a raised indurated border. That they are painless separates them from the somewhat similarly appearing but benign canker sore or herpetic eruption. Their rapid appearance distinguishes them from a slowly growing carcinoma. The clinician must observe meticulous hygiene, including gloves, when palpating a chancre, as it will be swarming with living spirochetes.

Two important telltale signs of systemic diseases may present in the lips: telangiectasias and hyperpigmentation.

Telangiectasia

Multiple telangiectasias scattered across the lips can be the visible component of similar extensive lesions in the nose, gastrointestinal tract, lung, liver, and brain [3]. These pencil point, red spots may be particularly numerous on the gingival surface. These are mini-arteriovenous malformations in the syndrome called "hereditary hemorrhagic telangiectasia." It is also known as the "Osler-Weber-Rendu syndrome." Nearly all patients with this familiar condition have spontaneous episodes of epistaxis before the age of 21 [4]. Intestinal bleeding is another very common complication.

Hyperpigmentation

The natural pigmentation of lips varies enormously among different races. Color may range from light-pink to deep-purple. Hyperpigmented splotches that extend from the lips to the gingiva and to the buccal mucosa may be a clue to the syndrome of "familial polyposis." Also referred to as the "Peutz-Jeghers syndrome," the characteristic small, brown maculae may be the earliest manifestations. The syndrome carries the burden of multiple polyps in the stomach and intestine. These present a substantial risk of becoming cancerous, particularly of the breast and gastrointestinal tract [5].

Mucosa

This section addresses those lesions that are most likely to appear on the mucosa of the buccal surface of the cheek and on the floor and the roof of the mouth. The clinician must keep in mind that white to gray lesions that can occur anywhere in the mouth are often troublesome diagnostically for the nonspecialist. They include leukoplakia, lichen planus, candidiasis, chancres, canker sores, and squamous cell carcinoma; each type of lesion can have a highly variable appearance. Oral lesions of different types can coexist. In practice, confidence in distinguishing persistent oral lesions by the specialist often comes down to tissue analysis. The concern, of course, is separating benign from more serious abnormalities or the potential to become malignant.

Aphthous Stomatitis

Perhaps the most common oral lesion, the canker sore is an ulcer that appears over a day or 2. Notoriously painful, it goes away in 7–10 days. Its round and shallow white cavity bordered by a wide, erythematous halo is easily recognized. A canker sore, typically less than 1 cm in width, can crop up virtually anywhere in the mouth as single aphtha but sometimes appears in multiples. Because of the recurrent nature of canker sores, a patient is likely to be familiar with their coming and going.

The cause of aphthous stomatitis remains uncertain, although multiple etiologies have been proposed. As a rule, if an ulcer presumed to be aphthous lasts longer than 2 weeks, another diagnosis should be considered.

Leukoplakia

Leukoplakia (in Greek, a "white plate") is a hyperkeratotic patch that is firmly adherent. Typically, a patch develops at the site of chronic irritation from, for example, a denture. It will heal within 2 weeks after elimination of the irritant. Persistent leukoplakia is at risk of transforming

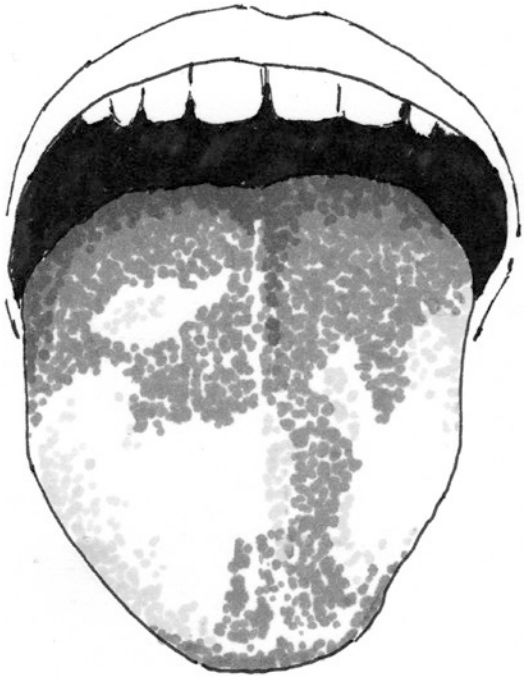

Fig. 15.1 Leukoplakia

into carcinoma. An erythematous component of the plaque greatly increases possibility of malignancy. This potential is particular high when combined with heavy tobacco and alcohol use. Transition to a basal cell carcinoma is much more likely if the leukoplakic lesion is on the undersurface of the tongue or on the floor of the mouth [6] (Fig. 15.1).

Lichen Planus

Lichen planus is an autoimmune disease of the skin and mucous membranes, usually found on the buccal surface of the mouth. As the name implies, the typical lesion reminds one of the mossy patches that grow scattered on rocks.

Its "lacy" appearance is most revealing, but lichen planus can cause swelling, atrophy, plaques, and ulcerative lesions. It may produce no symptoms, although at times it can be painful. It may be localized or extensive. Lichen planus usually appears in persons of middle age or older. Women are twice as likely to develop it than men. There is an increased susceptibility to

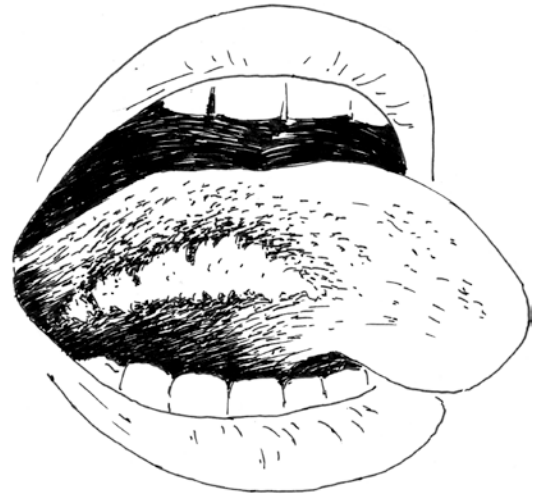

Fig. 15.2 Lichen planus

lichen planus in systemic diseases, especially those involving the liver. Particularly in HIV, it has a propensity to occur along the side of the tongue [7] (Fig. 15.2).

Mycotic Stomatitis

Oral thrush is a fungal infection from *Candida albicans*. It "paints" the mucous membranes with a thin, white, and patchy coat. At the beginning, the lesions of thrush may be spotty, but they gradually coalesce. In contrast with leukoplakia and lichen planus, the coat can be rubbed off, leaving behind a raw, reddened undersurface. Oral thrush is typically painless, but it can become sore and cause painful swallowing at an advanced stage, suggesting an esophageal component.

Candida albicans resides normally and unobtrusively within the mouth. It becomes invasive, however, when the normal flora is altered. Such occurs after a course of antibiotics, by far the most common cause of oral thrush. Uncontrolled diabetes is a predisposing condition. There is also a strong association of candidiasis glossitis with compromised immunologic defenses, such as prolonged corticosteroid treatment and HIV/AIDS. There is a high incidence of thrush among smokers.

The source of candidiasis may also be mechanical from an ill-fitted dental appliance or

by a sharp-edged tooth. Poorly cleaned dental appliances are a strong contributing factor to *Candida* infection.

Tongue

The tongue is a reflection of general health. In an earlier era, physicians relied heavily on the appearance of the tongue as their barometer of health and the key to diagnosing illnesses. The tongue and its coating, if any, told them much about disease by its color, texture, and odor. The lengths to which fanciful conclusions were made based on the tongue's appearance tell much about the lore of long-ago medical practice.

An interesting question for a game of trivia is: what is the only muscle of the body that does not have both a point of origin and a point of insertion? The answer, of course, is the tongue.

Size

Enlargement

Angioedema of the tongue can be an early warning sign of a serious allergic reaction. It will probably occur with swelling of the lips. In anaphylaxis, the swollen tongue can very quickly progress to edema of the throat and larynx.

Macroglossia

An expected feature of acromegaly is an enlarged tongue. It is also characteristic of primary amyloidosis, that is, the replacement of muscle tissue with an amorphous amyloid substance. The other preeminent site of amyloid deposition in this condition is the heart. In myxedema thickening of the tongue results from infiltration of a mucin-like substance. Common to all three systemic diseases is garbling of speech from the oversized tongue.

Movement

Weakness

Normally, the tongue will protrude midline. This action is wholly dependent upon the hypoglossal nerve which has a purely motor function. The tongue has bilateral innervation from the cerebral cortex. Therefore, injury or mass pressure that compromises the hypoglossal nerve or its root unilaterally results in relatively mild weakness of the tongue. Dysarthria from a lesion in the cortex or brain stem will be characterized by slurring of words and difficulty in sounding "la-la-la." A bilateral lesion of the corticobulbar tract causes speech to be spastic and labored.

Injury to the hypoglossal nucleus or in the nerve projecting from it causes a weakness in the tongue of the lower motor neuron type. The tongue on protrusion deviates to toward the weaker side. For detection of a more subtle weakness, ask the patient to press the tongue into each cheek and compare sides with external resistance. Eventually, atrophy will occur on the affected side of the peripheral hypoglossal nerve injury.

Taste is not affected by solitary lesions of the hypoglossal nerve. Loss of anterior taste will occur, however, if peripheral hypoglossal dysfunction is accompanied by injury to the afferent branches of the facial nerve.

Fasciculations

Sporadic fine twitches are common in the normal, resting tongue. Sustained, wormlike quivering, on the other hand, occurs from lower motor neuron disease. The finding is suspect for degenerative neurological disease, particular amyotrophic lateral sclerosis.

Color

Erythema

Reddened areas at the edges of the tongue are usually caused by local irritation from dentures. Tongue piercings not only cause mechanical irritation; they also provide an entryway for microorganisms in addition to being a potential allergen to the implanted ornament. Furthermore, keloid formation, chipped or fractured teeth, and interference with speech or swallowing are reported consequences of tongue jewelry [8].

Glossitis

Glossitis, characterized by a red tongue that may or may not be coated, painful, swollen, and textured, has many causes, including infections by bacteria, fungi, and viruses. The best known of the intensely reddened tongue along with enlarged papillae is the "strawberry tongue" of scarlet fever. Additional examples are Kawasaki disease, a vasculitis of childhood [9], and the toxic shock syndrome from an exotoxin released by *Staphylococcus* [10].

Median rhomboid glossitis is a fairly common discoloration on the dorsum of the normal tongue. It has an ovoid (egg-shaped) outline and within it the papillae have not developed. The area varies in color from light-pink to dark-red. It is thought to be of developmental origin and is wholly asymptomatic.

Moisture

Sicca

Excessive dryness of the tongue, of course, suggests dehydration. A more reliable place to look for dehydration, however, is at the junction of the labial and gingival mucosa.

Mouth breathing can cause a dry tongue as well as dry buccal mucosa. Habitual mouth breathers may have bright red and slightly swollen labial gingiva around the upper front teeth because of constant cycles of wetting and drying.

The loss of the sense of thirst with advanced aging is probably a greatly underestimated cause of dehydration. A dry mouth is commonplace in the nursing home population where patients have no obvious cause of fluid loss. It likely reflects asymptomatic dehydration in those who have an inadequate fluid intake.

Along with dry eyes, a dry tongue is a prominent feature of sicca disease (Sjögren's syndrome). The condition sometimes fits within the spectrum of rheumatic diseases.

Texture

The texture of the dorsal surface of the tongue is made up of thousands of tiny, mushroom-shaped papillae, each papilla containing about 100 neuronal receptors for taste. The ventral surface, which is smooth, can be quickly visualized by asking the patient to place the tip of the tongue on the roof of the mouth. It is here in the sublingual fossa where neoplastic lesions can develop while long remaining asymptomatic.

Smooth Tongue

A smooth tongue top represents the loss of functioning papillae. Historically well-known examples of such vitamin deficiencies were beriberi (thiamine) and pellagra (pyridoxine). Smooth tongue (atrophic glossitis) from severe lack of nutritional iron, vitamin B12, and folic acid remains a clinical concern in vulnerable populations. Indeed, a smooth tongue is found on about half the patients with newly diagnosed pernicious anemia, almost all of them elderly [11]. Astonishing recoveries have been witnessed following nutritional replacement in patients with multiple, debilitating features of vitamin B12 deficiency [12].

Geographic Tongue

A patchy area of erythema separated from smooth areas by a whitish border is known as "geographic" tongue because of its map-like appearance. The condition causes no symptoms other than the patient's worry and perhaps some burning from spicy foods. The sharp outline of the "map" appears to migrate as one area heals and new area of inflammation begins. These features have earned it the name "benign migratory glossitis."

Hairy Leukoplakia

Shaggy, white projections along the side of the tongue are known as "hairy leukoplakia." They form from activation of Epstein-Barr viruses (EBV) when natural immunity is compromised. The finding is highly suggestive of HIV/AIDS [13].

Ulcer of the Tongue

Other than the painful and easily recognized canker sore, an ulcer of the tongue is worrisome. Prompt steps toward a definitive diagnosis are indicated. Causal infections range from syphilis to tuberculosis. A neoplasm must also be considered.

Oral Cancer

Of course, the possibility of cancer must be considered in any mass or ulceration around the tongue. The most likely sites for oral cancer are the lateral, posterior third of the tongue and the floor of the mouth. These lesions are generally solitary or asymmetrical; their physical appearance on and around the tongue is too variable for meaningful description here. The wary clinician will recognize something on the tongue that should not be there and will initiate prompt diagnostic steps.

Risk factors for oral cancer are important considerations. Most incriminating is the use of chewing tobacco. Tobacco smoking, excessive use of alcohol, and infection with human papillomavirus (HPV) are well-known provocateurs. At the same time, it must be recognized that oral cancer occurs in a substantial percentage of the population without any known predisposing factors.

Taste

The taste buds are carried on about ten thousand fungiform papillae that spread out across the dorsal surface of the tongue. Each contains taste-specific receptors for salt, sweet, sour, and bitter. A fifth basic taste now recognized is called umami (Japanese for "pleasant taste"); it is a savory flavor found in meat broths, seaweed, fermented products, tomatoes, and soy sauce.

The popularized map showing areas on the tongue that defined where various tastes are sensed has been proven incorrect. Instead, all five tastes can be received anywhere on the tongue. The receptor nerves, however, do have their dominant territories. The anterior tongue is supplied by the chorda tympani branch of the facial nerve. Supplying the posterior tongue are the branches of the glossopharyngeal and vagus nerves. Activated receptors for taste also stimulate secretions of the salivary glands. Touch-sensitive receptors in other papillae are efferent neurons of the trigeminal nerve mandibular branch, and these contribute to the sensation of texture of food.

Loss of Taste

The ordinary physical examination does not include testing for taste. In fact, most patients complaining of a loss of taste are usually found to have a loss of smell, described in Chap. 14.

To check for taste, simply place a drop from a strong solution of table sugar or salt on the tip of the tongue. When there is an isolated loss of the ability to taste these substances, damage to the peripheral, sensory branch (chorda tympani) of the facial nerve can be assumed. If there is, in addition, unilateral paresis or paralysis within the facial nerve distribution, the lesion is more proximal, in or above the mastoid area where the chorda tympani branch joins the facial nerve trunk. An acute onset is consistent with Bell's palsy.

Teeth/Gingiva

A systematic scanning of the teeth and gums into which they are anchored provides a quick but useful picture of significant abnormalities. The general clinician, no one expects, will have the expertise of the dental specialist, but some fundamentals of observations can be rapidly addressed.

Disfigurement

Notice any jagged edges of a tooth and whether or not these are causing irritation of the adjacent buccal mucosa or the side of the tongue. The biting edges of opposing teeth may show worn notches from habitually holding objects – nails, bobby pin, splicing tools, or cord – at work or as hobby. Archeologists have a special interest in the grooves on teeth; these reflect the wear from fibers held there in the process of making primitive tools and clothing.

Caries

Yellow to brownish spots anywhere on a tooth indicate that, possibly, some erosion of the enamel has occurred. As the breach deepens, it reaches

the dentin and turns dark-brown or black. At this stage, pieces of the crown may have broken off. A red, swollen, and tender area at the apex of the root indicates that an abscess has formed.

Plaque is a white-to-yellow film secreted by the salivary glands. It tends to accumulate in gaps and grooves (called "plaque traps") beginning at the intersection of the tooth and gum. This layer harbors colonies of diverse bacteria that have a critical role in the development of gingivitis and caries.

Tartar consists of calcium-containing concretions of plaque. It builds up around the base of the tooth, especially along the lingual surfaces of the lower front teeth and on the buccal surfaces of the maxillary molars. It is what the dental hygienist scrapes off. Tartar may be white, yellow, or brown depending upon exposures. In tobacco smokers, it can be black.

Gingivitis

Gingivitis is inflammation of the gums, identified by tender and swollen, bright red margins visible around the base of a tooth and contrasting sharply with the pink surrounding it. There is a tendency for the fragile tissue to bleed from chewing hard foods or after brushing. Coupled with these changes is one cause of halitosis, originating from dense colonies of gram-negative bacteria.

As gingivitis develops into a more advanced stage, recession of the gum from the base of the tooth occurs, exposing the roots. Indeed, it was the "long in the tooth" observation that told the wary horse trader that a prospective purchase was actually pretty well along in years.

Erosion of the bone of the tooth socket is a further complication of gingivitis, a condition denoted as "periodontitis." Infection is superimposed. An involved tooth becomes loose. Without dental intervention, its loss is inevitable.

At this point, it is obvious to an older clinician rather than one just beginning a medical career that the state of contemporary dentition has markedly improved from that of the past. In fairly recent decades, edentulous patients were common, and those who were not usually had many missing teeth. The inability to chew properly surely compromised nutrition and must be considered a major factor in statistics of population longevity. With what we know from the emerging scientific advances of dental hygiene – regular brushing and flossing to remove bacteria-nurturing food between teeth along with fluoridation – new generations can enjoy the blessing of a full set of healthy teeth.

On a historical note, George Washington lost his first tooth at the age of 21. From then on, he lost about one tooth a year. By the time he became president, at the age of 57, he had one tooth left. Portraits do not depict the hollowed cheeks. The famous false teeth (and they were not wooden) were used more for cosmetics as they were not much help in chewing. A scar on the left cheek was from surgery to drain an abscessed tooth.

Hyperplasia

Some drugs are known to induce fibro-epithelial overgrowth of the gingiva between teeth and encroach on them vertically. The first recognized of these drugs was phenytoin (Dilantin®) [14] although many other anticonvulsives, calcium channel agonists, and immunosuppressant drugs have been implicated. These include phenobarbital, nifedipine [15], amlodipine [16], and cyclosporine [17]. The relationship between drug and gingival hyperplasia may involve a cofactor such as accumulated plaque. It has been postulated that these drugs may concentrate where plaque is plentiful.

In addition to drug induction of gingival hyperplasia, there may be other causes such as pregnancy, puberty, mouth breathing, and poor dental hygiene. Blood dyscrasias and neoplasms have also been implicated in susceptibility.

Palate

On the normal palate are tiny pink vascular tufts lying within the cribriform plate.

The hard palate is an extension of the ethmoid bone, forming the roof of mouth. It is perforated with nerve ending for smell.

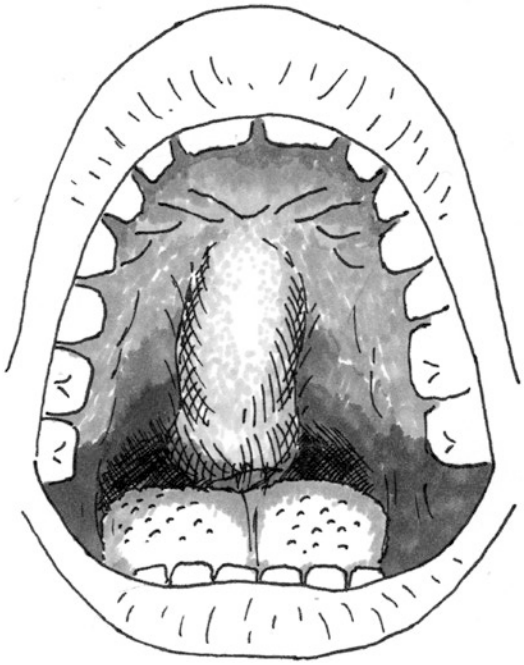

Fig. 15.3 Exostosis

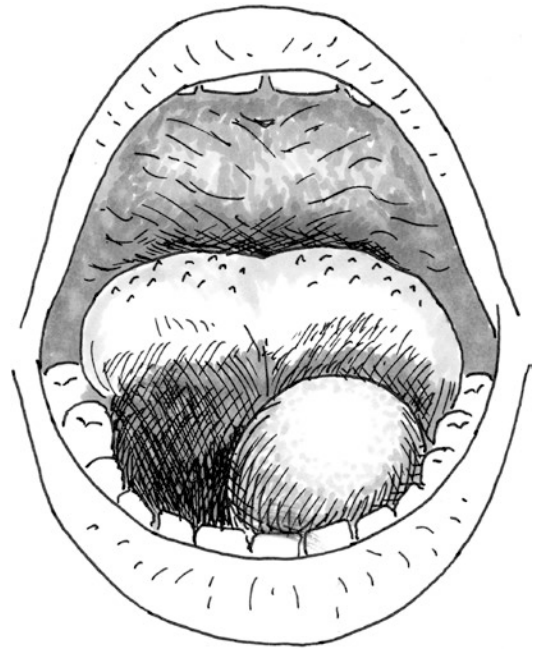

Fig. 15.4 Ranula

Petechiae

The normal vascular tufts present in the hard palate must be differentiated from petechiae which are about the same size but are dark-red in color. Petechiae are commonly seen here in the early stages of infectious mononucleosis. Once thought pathognomonic for this infection, further experience has found them in various forms of vasculitis, including lupus erythematosus.

Exostosis

A completely benign and painless bony bulge from the hard palate is an exostosis. It is referred to as a maxillary "torus" (Latin for a "round swelling"). Mandibular tori typically appear bilaterally and occur on the lingual side of the premolars (Fig. 15.3).

Sublingual Fossa

The floor of the mouth is quickly inspected by having the patient place the tip of the tongue against the palate. Most patients can cooperate enough to permit observation of the sublingual fossa without needing a tongue blade to control the tongue.

Cystic Lesion

Cystic lesions lying in the sublingual floor are called "ranula." The name translates from Latin as "little frog" (perhaps because of the resemblance to the distended throat tissue when a frog croaks). It is a mucocele derived from a salivary gland. Ranulas are rounded, soft, fluctuant, and translucent with a bluish tint. They are benign but can be large enough to interfere with swallowing (Fig. 15.4).

Clear mucoceles can also form in the lips. They are usually caused by trauma to the minor salivary glands located there.

Pharynx

Observing the posterior oropharynx without a tongue blade can be facilitated by using this three-point method:

1. The tongue is relaxed (not protruded), and the head is hyperextended.
2. Base of the tongue is flattened with vocalization of the "a" sound as in *cat*. This sound is much more effective for visualizing the pharynx than the traditional "ah" of *car*.

Pharyngitis

Acute pharyngitis can manifest any degree of visible pathology, from barely erythematous to huge, ulcerating areas of exudates. There is no certain way to tell apart bacterial, fungal, and viral etiologies (proven by many studies) although there are some distinctive clues. The presence of coryza (runny nose, nasal discharge, and sneezes) strongly suggests a viral upper respiratory infection and which, in the vast majority of infections, is the cause of acute pharyngitis. Indeed, one cannot confidently diagnose acute pharyngitis from a common cold in the absence of nasal symptoms. High fever and cervical lymphadenopathy, on the other hand, tend to favor a bacterial cause.

The constellation of findings typical of pharyngitis from infectious mononucleosis includes palatal petechiae, extensive cervical lymphadenopathy (especially in the posterior region), and splenomegaly. Thickened patches of adherent exudates that may extend forward onto the tongue and buccal mucosa are more typical of a fungal infection.

Pharyngitis is one of the earliest manifestations of significant leukopenia. The symptom or the signs of pharyngitis should be in mind on any patient taking a drug that may lower the white blood cell count. Propylthiouracil taken to treat hyperthyroidism is such an example [18]. The causal agranulocytosis can develop precipitously even after prolonged therapy.

Peritonsillar Abscess

Suspect a peritonsillar abscess if the acutepharyngeal pathology is predominantly or exclusively unilateral. It can affect the voice and obstruct the upper respiratory tract. Here, a bacterial infection is most likely causal. Extension of the abscess can lead to regional cellulitis. Formerly called "quinsy" (from Greek, "to choke"), it was most likely the recurring infection that finally did in the indomitable George Washington.

Neurologically, the pharyngeal functions of both the glossopharyngeal and the vagus nerves are assumed normal if swallowing is unimpeded and there is no pooling of secretions. The "gag reflex" needs to be done only when symptoms or signs of dysphagia are present. Failure of the tonsillar arch to rise on contact stimulation of the oropharynx with a tongue blade, coupled with deviation of the uvula to the opposite side, confirms a defect of these cranial nerves.

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The One-Minute Cranial Nerve Examination

16

Similar in intent to the "one-minute physical examination," this screening discipline is meant to provide reasonable reassurance that the patient has no major cranial nerve deficit. The survey, based upon reading aloud, is quickly conducted. If the patient without neurological symptoms demonstrates normal reception of print, can interpret the symbols into words, and is able to express them in speech, the clinician has acquired substantial evidence that cranial nerve function is healthy. Any abnormality, of course, dictates that a comprehensive evaluation of the cranial nerves is warranted. Testing by having the patient read aloud does not include functions of the trochlear, abducens, olfactory, and accessory nerves.

The "one-minute cranial nerve examination" is appropriate at this time, now that the cranial nerve functions have been covered in the preceding chapters. For expedience, however, the clinician could accomplish this survey earlier as he or she begins to examine the eye. Detailed guidelines for testing have been provided in Chap. 12.

Simply put, by giving the patient instructions to read aloud, by observing the eyes during several seconds of near vision, and by listening to the voice, an astonishing amount of information about cranial nerve function is sampled. Certainly, reading aloud in the physical examination is a colossal leap beyond the traditional recognition of letters on an eye chart.

If the "one-minute physical examination" is an example of determining the health of a tree by looking at the details of a leaf, the "one-minute cranial nerve examination" may be thought of as akin to a home appliance. Each of its many components would not perform normally if any of its parts in a complex pathway were disabled. If the "on" indicator on the toaster lights up, you have proof beyond question that the municipal power plant – be it scores of mile away – is functioning. Furthermore, the wire that carries the current from power plant to outlet is intact. Furthermore, the little *on* bulb works. The crucial test of the sensor mechanism is the toaster's response to a setting between *light* and *dark*. If the toast comes out the way it should, one can conclude that all elements in a complex system are in working order.

Components of cranial nerve function required for reading aloud are described briefly here.

Cranial Nerves

II. Optic Nerve

During the act of reading, single letter images are picked up and carried to the brain. Perception of these individual symbols proves that the peripheral photon receptors (the 0.1 mm fovea) and the central receptors are intact, as are the conduits to them. In other words, the optical physics are intact, as is the delivery of each image to the brain. The clinician has now determined that binocular acuity for near vision is competent as well as acuity of each eye determined separately.

R.E. Phillips, *The Physical Exam*, https://doi.org/10.1007/978-3-319-63847-8_16

The primary visual cortex, area 17 of occipital lobe, receives the raw images combined from each retina. Here at the primary visual cortex, the images of individual symbols (in this case letters) are received. They are then relayed to language receptive centers in various areas of the brain where the letters are reassembled and interpreted into words. Of all the intricate tasks that humans learn over a lifetime, this integration is the most complex and the longest in developing. Certainly, a major activity of the brain is devoted to the visual cortex and the interpretation of these images.

III. Oculomotor Nerve

Both eyes have been engaged in near vision, demonstrating that ocular convergence has occurred. Thus, the medial rectus muscles were activated. Constriction of the pupils, also a function of the oculomotor nerve, provides improved resolution from optical aberration.

V. Trigeminal Nerve

The nasociliary branch of the trigeminal nerve supplies the ciliary muscles that govern the curvature of the lens. Near vision depends on increased tension of these muscles to increase the curvature of the lens, thereby decreasing the focal length.

Speaking words aloud, of course, involves additional sensory input from the face. Who has not experienced slurred speech after receiving a local anesthetic into a trigeminal branch by a dentist?

VII. Facial Nerve

The maxillary and mandibular branches of the facial nerve enervate the muscles of the cheek and jaw. They are critical in the formation of verbal expression.

VIII. Auditory Nerve

The patient has heard the instructions "Read aloud" in a low voice and responded accordingly. Admittedly, this component of testing has low sensitivity but may be useful in some assessments. Nystagmus during reading reveals a dysfunctional vestibular component.

IX. Glossopharyngeal Nerve

Paralysis of the glossopharyngeal nerve weakens the soft palate. The result is speech that has a distinctive nasal quality.

X. Vagus Nerve

The complex function of swallowing is controlled by the vagus nerve. When compromised, secretions that are audible tend to accumulate in the pharyngeal space.

XII. Hypoglossal Nerve

The muscular component of the tongue is activated solely by the hypoglossal nerve. Any neurologic defect in this muscle is readily evident on attempting to speak.

In summary, most cranial nerves function normally in a patient who:

1. Reads small print with each eye separately and together
2. Expresses the words read in a normal tone
3. Enunciates word clearly
4. Has no audible secretions in the throat

In other words, there is no dysarthria, no dysphonia, and no dysphagia. Dysarthria indicates compromised muscle and sensory functions of the face and tongue for speech. Dysphonia – an abnormal quality of voice – is present with paralysis of the muscle in the soft palate. Dysphagia is

difficulty in swallowing that causes pooling of secretions in the oropharynx.

The skill of reading aloud requires such a high level of neuromuscular ability that any appreciable impairment is readily detected. Examples are mild degrees of intoxication from alcohol or narcotic drugs when proficiency of reading is noticeably compromised. In addition, memory can be grossly assessed simply by asking the patient to roughly describe what was just read. "Something about being made in Michigan" is quite enough to indicate understanding and expression of what was read on a facial tissue box.

If normal, this information can be quickly and accurately recorded as "reads Nl = R/L/B" (denoting normal reading in right/left/binocular). Adding "without dysarthria, dysphonia, and dysphagia" substantiates a wealth of additional observations.

Cranial nerve functions not tested on reading aloud are smell, lateral and rotary eye movement, head turning, and shrugging the shoulders. The first mentioned, the olfactory nerve, is seldom tested in the routine physical examination. Indications for testing smell, however, are covered in Chap. 14. Significant weaknesses of muscles innervated by cranial nerves IV and VI (trochlear and abducens, respectively) are highly unlikely in the absence of even transient diplopia. Certainly, a patient with limitation of neck and shoulder movement, requiring a functional cranial nerve XI, the accessory nerve, is likely to have symptoms or have "failed" the palmar drift test described in Chap. 9.

The "one-minute cranial nerve examination" (that can actually be accomplished in 15 s) is meant as a survey in the general physical examination. Any question of the ability to read aloud dictates that each component of this complex function be tested separately and with meticulous care.

Part II

Patient Sitting with Legs on Table at 90° from Examiner

Chest

17

Position #2 Patient sitting at a right angle to the examiner.

The patient is asked to raise his or her legs while turning to sit lengthwise on the bed or examining table. This change in position is made with minimum instructions and consumption of time. Oriented at right angles to the patient, the clinician has easy access to observe, palpate, and listen to the posterior chest as well as the precordium.

Essentials
Shape
Breathing ease, rate, pattern, and phases
Percussion
Breath sounds

Examine the back from the nape of the neck to hips for moles, sebaceous cysts, and other skin lesions. Keep in mind, the back is the area that the patient never sees.

Shape

This side view is optimal for observing the dynamics of breathing as well as configuration of the chest. The kyphosis of typical advanced osteoporosis is most evident in profile; it is described in Chap. 25.

Emphysema

In emphysema, the "barrel chest" ascribed to an increase in the fore-aft dimension has been questioned. The impression has been attributed instead to some degree of kyphosis and an asthenic abdomen [1]. Noted in advanced emphysema is the chest rising and falling as a fixed shell rather than an expansible arrangement of ribs and muscles.

Pectus Carinatum

Pectus carinatum ("pigeon chest" or "keeled breast") is an anterior protrusion of the chest, simulating a keel. In mild degrees, the condition is asymptomatic. Moderate to severe degrees, however, can impair respiratory movement and reduce lung compliance [2]. Associated is a high incidence of mitral valve prolapse [3]. Patients with significant pectus carinatum commonly have scoliosis and inherited disorders of the connective tissue (e.g., Marfan syndrome and Ehlers-Danlos syndrome). The cosmetic aspect of pectus carinatum, regardless of the severity, can impose a heavy burden on self-esteem (Fig. 17.1).

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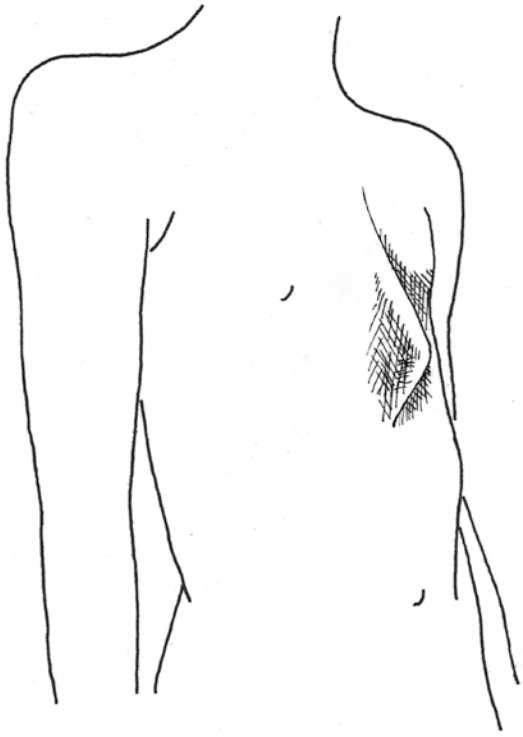

Fig. 17.1 Chest: pectus carinatum

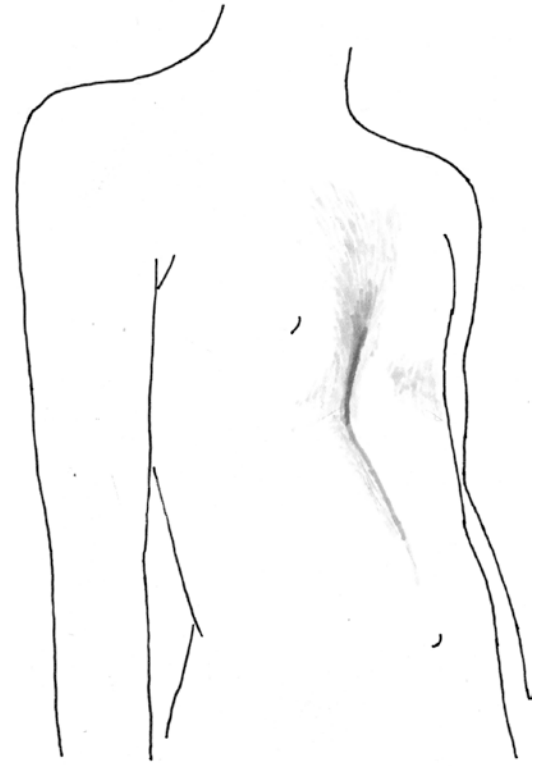

Fig. 17.2 Chest: pectus excavatum

Pectus Excavatum

The sunken sternum in pectus excavatum ("funnel chest") is often found with flaring of the lower rib cage. Patients with pectus excavatum who do not experience respiratory symptoms may have near normal lung capacities, as demonstrated in adolescents [4]. The inward displacement of the anterior chest, however, may be of sufficient degree to shift the heart and lungs, compromising cardiac filling during ventricular diastole. Breathing, especially during exertion, will be seriously impaired (Fig. 17.2).

Pectus excavatum has been dramatically depicted by the seventeenth-century artist Jusepe de Ribera [5]. Although it is far more common than pectus carinatum, the two deformities of the chest share many of the coexisting problems: mitral valve, connective tissue, and skeletal anomalies.

Breathing

On physical examination, the dynamics of respiration can be sorted into several components.

Ease of Breathing

A cardinal observation in pulmonary assessment is the effort of breathing at rest. A clue to breathlessness is the tendency to speak in phrases rather than full sentences. This phenomenon can be sampled in people with normal respiratory function immediately after they have walked up a couple flights of stairs at a rapid pace.

Enlistment of the accessory muscle of breathing at rest indicates respiratory insufficiency from virtually all causes. Typical of air-trapping disorders, such as emphysema and asthma, is

pursing the lips during exhalation and nose breathing in inhalation. The benefit of pursed lips is to increase airway resistance, preventing the collapse of small and collapsible airways and easing the work of moving air. The technique is useful in rehabilitation of dyspneic patients [6].

Rate of Breathing

The rate of breathing is a highly sensitive barometer of respiratory function. Even so, the minute rate is often overlooked in the patient assessment, partly because of the time required to measure it (minimally, 30 s with multiplying by 2). In hospitals, staff personnel routinely take periodic respiratory rates, but the recordings may not be routinely checked by the clinician.

The respiratory rate of the normal, resting adult is typically 16–18 times a minute. Variations in body size, adiposity, ambient temperature, and ease of mind expand the range of normal to 14–20 breaths a minute in adults and up to 25 a minute in sub-teenagers. Even slight deviations either way may represent a problem of pulmonary, cardiac, metabolism, or hematological origin.

Determinates of respiratory rate are numerous. They are noted here in outline.

1. Increased respiratory rate
   (a) Fever (particularly with lung infection)
   (b) Airway restriction (oropharynx to bronchioles)
   (c) Reduced lung expansion (pneumothorax or large intrathoracic mass)
   (d) Reduced chest wall expansion (scleroderma, muscular weakness, rheumatoid arthritis)
   (e) Significant anemia
   (f) Compromised ambient air such as reduced oxygen (including high altitude), increased carbon dioxide, or toxic aerosols
   (g) Sympathomimetic medications (including vasoconstrictors such as cocaine)
   (h) Metabolic disease (hyperthyroidism and acidosis from diabetic ketoacidosis or uremia)
   (i) Anxiety
   (j) Cardiac insufficiency
2. Decreased respiratory rate
   Disorders affecting the central nervous system are preeminent causes:
   (a) Drugs, notably the opiates
   (b) Diffuse brain disease (encephalitis)
   (c) Brain stem vascular event (stroke)
   (d) Peripheral nervous system diseases (botulism)
   (e) Muscle diseases (muscular dystrophy)
   (f) Reduced metabolism (hypothyroidism)
3. Periodic respiratory rate
   Referred to as Cheyne-Stokes respirations, the varying rates of breathing consist of periods of rapid and deep breathing with transition of periods of apnea or shallow, slow breathing. This periodicity occurs in a coma or near coma from cerebrovascular events, hyponatremia, encephalopathy, head trauma, tumor of the brain, and opiate toxicity. Periodic breathing has also been documented in congestive heart failure [7].

Phases of Breathing

Evaluation of respiratory phases is distinct from that of respiratory rate. The duration of inspiration, normally, is about twice that of exhalation in adults and closer to equal time in children.

Increased Inspiration

Effort and prolongation of inspiration are likely in obstructive conditions in the larger airways. Chronic bronchitis with thickened mucus or a foreign body partially blocking an airway should be considered. The "whoop" in pertussis is inspiratory stridor. When the obstruction involves the larynx, the speaking voice is altered.

Increased Exhalation

Bronchospasm prolongs exhalation, as is typical of asthma. In emphysema, increased airway resistance from bronchitis is often a cofeature that together – comprising the clinical syndrome

of chronic obstructive lung disease (COPD) – lengthen the expiratory phase.

Movement of Breathing

A reduction in the movement of the chest occurs when the chest wall is stiffened or otherwise physically restricted.

Tension Pneumothorax

Respiratory distress of acute onset coupled with restricted movement of breathing is highly suggestive of a tension pneumothorax, truly an urgent condition. Here, the skill and confidence of identifying deviation of the trachea by palpation and hyperresonance on percussion on the hyper-aerated side are of critical value.

Emphysema

Hyperinflation of the chest, emphysema being a common example, decreases movement of the chest wall and diaphragm. The reduced excursions superimpose a burden on the compromised surface area of effective alveoli.

Obesity

The sheer weight of the morbidly adipose trunk significantly increases the work of breathing and contributes to restrictive and functional impairment of respiration. Hypoventilation in the very obese, along with abnormal expiratory flow rate and reduced vital capacity, has been demonstrated [8]. Many patients with body weight indices of 30 kg/m^2 or more have signs of cor pulmonale: increased venous pressure, dependent edema, hepatomegaly, and polycythemia from right-sided myocardial insufficiency [9].

Chronic hypoxia and hypercapnia in severe obesity are the products of insufficient respiratory exchange. Somnolence and nocturnal obstructive sleep apnea are common. The chemoreceptors for reduced blood oxygen and increased carbon dioxide appear to be desensitized [10].

The clinical picture of the obesity hypoventilation syndrome comes up in fiction of Charles Dickens. Writing in a serialized novel, Dickens describes Joe, a servant "fat and red-faced boy in a state of somnolence"… who "consumes great quantities of food and constantly falls asleep in any situation at any time of day" [11]. The term "Pickwickian syndrome" was first mentioned in an article on the "obesity hypoventilation syndrome" that appeared in the *American Journal of Medicine* in 1956 [12].

Rheumatoid Arthritis

In pulmonary function studies on non-smokers with rheumatoid arthritis, there is evidence of both restrictive and obstructive lung disease [13]. Most patients had some degree of dyspnea, cough, wheezing, or sputum. Pulmonary findings were in direct correlation with severity of the arthritis. Most patients had a significant decrease in the one-second forced expiratory volume (FEV_1) when compared with sex- and age-matched subjects [14]. It is presumed that impeded chest movement is an integral part of the pulmonary complications of generalized joint disease.

Ankylosing Spondylitis

Mainly a fibrotic disease of the vertebral column ("bamboo spine"), ankylosing spondylitis can seriously affect the mobility of the chest wall. Indeed, restrictive pulmonary function from stiffness of the rib cage impairs pulmonary function [15]. Fibrosis and ossification causes a more rigid fusion between ribs and vertebrae.

Percussion

Percussion of the chest is, admittedly, a rather crude technique, hardly worth the time in most encounters in patients without respiratory symptoms and without evidence of pulmonary or cardiac disorders. On the other hand, percussing the posterior chest at the bases can reveal an elevated diaphragm or a pleural effusion. The procedure is performed rapidly with a fair degree of reliability. Because these conditions can be present and perhaps unsuspected, it is good practice – while the patient is sitting – to incorporate percussion at the lung bases in every comprehensive examination. There is little point in routine percussion higher in the chest.

Trying to outdo the old-time clinicians or the chest x-ray by determining a suspected small effusion in the costophrenic angle or a pulmonary mass or infiltrate by percussion is not time efficiently spent. The same argument is offered regarding the use of tactile or vocal fremitus, still taught in medical schools. Certainly, the pulmonary specialist may rightly take exception to this viewpoint.

Normal and symmetrical resonance can be rapidly ascertained. Only the posterior, lower chest need to be percussed in the routine examination and with the patient sitting. It is at the base where a pleural effusion will settle and where an elevated diaphragm will be evident. Normally, the right diaphragm is slightly higher than the left (Fig. 17.3).

Loud percussion is not necessary. The best technique mimics that used by the orchestra timpanist, using one hand very firmly against the chest (to tighten the "drum skin"), firm enough to feel resistance of the patient against the push. The stroking is performed with the hand loose at the wrist (simulating the flicking touch of the percussion mallet). Resonant tones at the posterior bases that are symmetrical are good evidence that no large pleural effusion or paralyzed diaphragm is present. Placing one ear close to the area percussed is helpful, especially in areas where the examination competes with ambient noise.

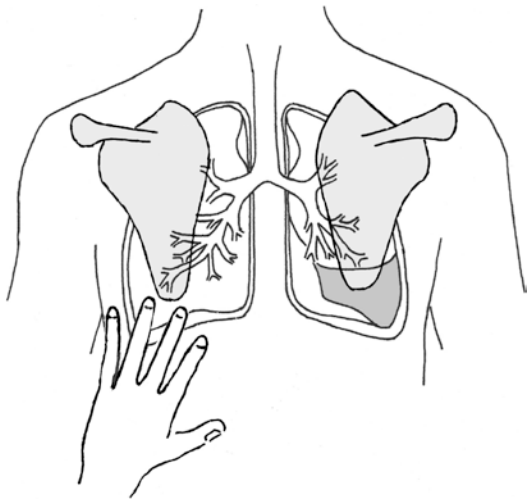

Fig. 17.3 Pleural effusion

Abnormal percussion tones at the bases, of course, dictate more extensive demarcations. Continue percussing upward until a dull-resonant border is detected. The cause may be an effusion or a high hemidiaphragm from paralysis or a large abdominal mass. Consistent direction in side-to-side comparisons is recommended.

Breath Sounds

Auscultation

Sites

With the patient sitting with one side toward the clinician, there is easy access for moving the stethoscope across the back and the lateral and anterior chest bilaterally. The stethoscope should be placed directly on the skin, not over the gown, to avoid extraneous sounds. This is when the open-back gown is convenient.

Routinely, listen to at least one full breath sound in each of six sites on the posterior chest from upper to lowermost areas. Then listen over the site in the lateral chest on both sides. The stethoscope can also be easily moved to the anterior chest on the near side; it is a bit of a stretch to reach across to listen to the anterior chest on the far side. Thus, breath sounds are heard throughout from a single vantage point and with a progression that is particularly convenient when the patient is a woman.

Compare the sounds on each side at the same level before proceeding to another level. It is helpful that the patient exaggerates the depth of normal breathing somewhat although deep breathing is generally not necessary (and can cause light-headedness). As advised throughout the physical examination, a consistent direction with side-to-side comparisons at each point is recommended (Fig. 17.4).

When a pulmonary problem is suspected, also listen also high in the axilla. The axillary area is one where overlying muscle and adipose tissue are minimal and where sounds are remarkably audible. Auscultation here is particularly useful when tuberculosis is considered

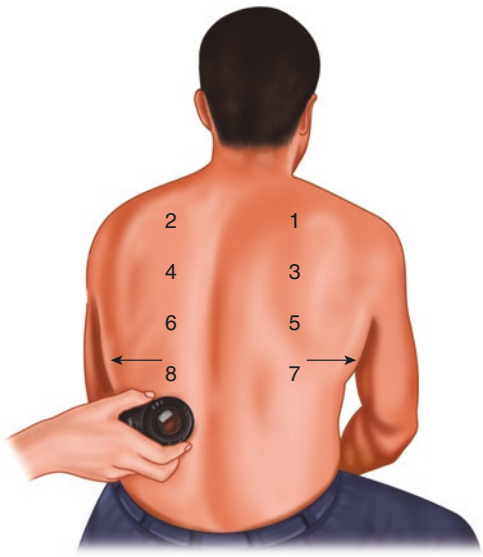

Fig. 17.4 Sequential back examination

in adults, an area of higher oxygen-to-blood ratio favoring infiltration from *Mycobacterium*. Listening over the axillae completes the "Ten Points of Auscultation".

Sound Qualities

It is critical that the nomenclature for breath sounds be consistent. The various qualities are defined as:

Vesicular A normal, soft, and clear sound of relatively low pitch.

Bronchial A normal, higher pitched more harsh sound, heard over larger airways, normally over the upper sternum and the axilla. The sound is abnormal when heard over the posterior or lower anterior chest.

Rales Crackling sounds that are high-pitched and fine, as if listening to rubbing hair close to the ear. These are similar to the sounds of effervescent liquids or perhaps to the "Velcro® sizzle." They reveal fluid in the smaller airways and are typical of congestive heart failure and pneumonia. Rales can also be somewhat lower pitched and coarse, particularly when the smaller airways are congested with mucus.

Rhonchi Low-pitched, coarse, and rattling sounds, suggesting mucus plugs in the larger airways. The sounds tend to change with each breath and more so after coughing. Indeed, it is helpful to have the patient cough forcibly to detect changes in sounds. Etymologically, the word "rhonchi" comes from ancient Greek, "snoring."

Wheeze "Musical" sounds that are high-pitched. Wheezing during exhalation indicates narrowing of the smaller airways. They are particularly characteristic of bronchospasm in asthma and bronchitis.

Wheezing during inspiration reveals an obstruction in the upper airways, anywhere from oropharynx and larynx to trachea and bronchi. A foreign body, fibrous scarring, or a neoplasm may be responsible. That inspiratory wheezing is a medical emergency is understood.

Stridor A harsher sound than the wheeze. Stridor occurs during both exhalation and inhalation and indicates, usually, laryngeal obstruction. In addition to a large foreign body (e.g., a cherry pit), pertussis and tumor may be responsible.

Tubular A solid mass between the stethoscope and the lung produces a sound similar to that normally heard directly over the trachea. Such a sound over the lung parenchyma suggests that a neoplasm is responsible.

Friction rub Scratching sound of inflamed membranes moving against each other, namely, the parietal and pleural membranes. The intensity of the fiction rub may vary as the patient assumes different positions such as leaning forward or to the side or lying down.

Pain

The physical examination of a patient with chest pain is fraught with misadventure regarding etiology and implication. Certainly coronary arterial disease should be foremost in mind, but it is not always the cause of chest pain. Some guidelines

are presented to help navigate the challenge of diagnosis with reasonable confidence. Here, the sequence begins externally and proceeds inward.

Skin

A cluster of vesicular lesions is typical of herpes zoster. The dermatome affected may or may not be painful or tender. In the pre-eruption stage of herpes zoster, patients will have localized discomfort (often described as a burning sensation); a light sweep across the area with a cotton ball or facial tissue elicits increased sensitivity when compared with the contralateral area. To this point, more than one patient with chest pain has ended up in the coronary care unit only to develop a vesicular chest rash in the following day or two. A more detailed description of herpes zoster lesions and pre-eruption hypersensitivity is provided under Chap. 20.

Chest Wall

Costochondritis

Inflammation of cartilage can occur at the joints of ribs and sternum, causing pain on deep breathing and coughing. Tenderness on firm manual pressure over the painful area may duplicate and intensify the chest pain. Most commonly, the area is where cartilage joins ribs to the sternum, termed "costochondritis" (or "Tietze syndrome"); it can usually be traced to trauma from strenuous effort, by stretching or on vigorous coughing [16].

Rib Fracture

Fractures of ribs can occur from injuries thought minor. Pain is pleuritic in nature and is aggravated by certain positions. A powerful cough can generate enough pressure to fracture a rib. The patient with advanced osteoporosis is particularly susceptible. Subcutaneous crepitus in the chest wall after fracture of a rib indicates that the lung has been puncture. Hemothorax and pneumothorax are possible concurrent complications.

Metastatic Neoplasm

The clinician should also consider a metastatic bone lesion in a highly localized area of persistent pain and tenderness along a rib or clavicle. The metastasis may or may not be palpable. It is most likely that the primary tumor is in the lung, the breast, or the prostate gland.

Pleural Membrane

Pain that increases on deep breathing suggests inflammation of the parietal and/or pleural membranes of the lung, by definition "pleurisy." A friction rub may be heard by listening over or close to the painful site. Deep inspiration or changes in chest position is sometimes required to provide an audible rub.

Pericardium

Patients with a pericarditis often experience pain when they are supine or leaning forward. The clinician should listen to the sound of a possible friction rub if the patient can duplicate the position in which it causes pain. Ancillary signs of pericarditis with effusion or constriction are distention of veins and a reduction in the pulse pressure (the difference between systolic and diastolic blood pressure). These signs are exaggerated by the patient taking a deep breath. Pain from acute infectious pericarditis is often accompanied by fever.

Notwithstanding these descriptions, inflammation of both pulmonary and pericardial membrane can be a complication of an acute coronary event. Implicated as causal is a transmural myocardial infarction. Cardiac tamponade can result from either an acute myocardial event or from out of therapeutic range anticoagulation therapy.

Mediastinum

Pain located in the mediastinum is ill-defined. Telltale physical signs are most likely indirect and related to venous obstruction, particularly of

the superior vena cava. Marked neck vein distention that is increased with deep inspiration is an expected finding.

Most cases of acute mediastinitis follow cardiovascular surgery, although rupture of the esophagus (spontaneously or after esophagoscopy), blunt trauma or transbronchial needle aspiration procedure or extension to the oropharynx or the lung may be causal. Chronic mediastinitis strongly implicates fibrous granulomatosis, particularly from *Histoplasma*, tuberculosis, or radiation [17].

Aorta

In acute dissection of the thoracic aorta, patients describe a migrating tear or stabling quality of pain. Dissection in the ascending portion is likely to produce pain over the anterior chest. If it occurs in the descending section, pain tends to radiate to the back and in this way simulates the pain of acute cholecystitis. Signs of pericarditis complicating dissection of the aorta indicate rupture into the pericardial space.

Cataclysmic symptoms and signs are characteristic of aortic dissection. These can range from shock, weakness of a limb, regurgitant flow of the aortic valve, and acute cardiac tamponade. Hypertension as a strong risk factor for thoracic aortic dissection has been well documented [18].

Coronary Arteries

The preeminent worry with chest pain of recent onset is that of acute obstruction disease of one or more coronary arteries from a critical stenosis by atherosclerotic plaque or by disintegration of an atheromatous plaque with secondary thrombosis. Referred pain to the neck, jaw, arms, or hands often complements the symptomatic description.

When nausea, sweating, weakness, lightheadedness, and dyspnea accompany the onset of chest pain, decompensation of the myocardium from ischemia can be presumed. Resulting are weakened peripheral pulses and reduced blood pressure. These signs and symptoms may be transient. If they occur at the onset of the chest pain, acute coronary arterial disease is highly suspect.

Pulmonary Veins

Pulmonary Embolism

A sizeable pulmonary embolism is likely to produce sudden chest pain that tends to be pleuritic in quality with varying degrees of severity. Direct clinical signs of pulmonary embolism are few and these have indeterminate reliability. Revealing but non-specific is an increase in the rate of breathing with or without the sensation of dyspnea. More telling diagnostically, perhaps, is the presence of thrombophlebitis in a limb that has given rise to an embolism. It must be remembered that thrombophlebitis can occur in the deep and the superficial veins of the upper extremities as well as in the legs [19].

A large released venous thrombus may occlude the pulmonary vein at the entry into the lung, thus reducing blood flow to both right and left lung. This "saddle" embolism will cause immediate severe respiratory distress and can cause syncope. Venous thrombi are friable collections of fibrin that tend to break up in the chest and scatter, often to both lungs. Once so dissipated, the patient may experience some degree of symptomatic relief after an initial episode of severe dyspnea. Interpretation by imaging may implicate multiple pulmonary emboli that could actually come from a single, fragmented thrombus.

Mention is made of a pulmonary embolism that has a situational inference. A person with acute chest pain minutes to hours after scuba diving suggests the possibility of nitrogen bubble emboli, commonly known as the "bends" [20]. Associated symptoms are respiratory distress, joint pain, and neurological deficits. Air embolism can complicate a central line catheter access. Liposuctioning is a source of fat emboli.

Subdiaphragm

A diagnostic conundrum of chest pain presents itself when the cause is somewhere below the

diaphragm. Inflammatory disorders of the liver, pancreas, stomach, and esophagus must be considered. Physical signs of these conditions are described in Chap. 20.

Breasts

To examine the female breast with confidence requires nothing short of a comprehensive approach. There is always the possibility that the breast examination performed as a "screening" procedure provides little more than unwarranted reassurance or consternation. Guidelines for the comprehensive female breast examination have been clearly defined and are beyond the scope of this writing. Certainly, obvious abnormalities or symptoms referable to the breast warrant further investigation. These include nipple rash, retraction or discharge, palpable masses, tenderness, or asymmetry of the breasts of recent onset. The breast is a fairly common place for a paradoxical necrotizing thrombosis to occur as a side effect of anticoagulation therapy.

In women, diagnostically unexplained lymph nodes that are palpable in the axilla should arouse suspicion of a neoplastic disease. Beyond such obvious conditions, the full breast examination of women for early detection of cancer should be planned for a separate session, perhaps coupled with a complete screening pelvic examination, imaging, and cytology, not as part of a general physical examination.

Gynecomastia

Causes of excessive breast tissue in males are many. Most commonly, it is a temporary condition appearing in boys (especially obese boys) around puberty when the yin and yang of androgenic and estrogenic hormones are vying for dominance. The enlarged breasts may be tender and have a diffuse, nodular texture of glandular tissue [21].

Gynecomastia of adolescence is not the quarter-size, rubbery buds that appear during puberty around the nipple-areola for about a year. Obesity can produce prominent breasts in males, but these have an adipose rather than a glandular texture; they are referred to as "pseudo-gynecomastia."

Gynecomastia in an adult male mostly originates from a non-endocrine gland source and is instead the product of an excessive estrogen-like substance. A deficiency of androgen activity is usually associated. Chronic liver or renal diseases are well-known causes. Also implicated are adrenal disease and hypogonadism, including tumors of the adrenal gland or testicles. Most incriminated for drug-induced gynecomastia are the antiviral drugs and nucleoside reverse transcriptase inhibitors used in treatment of HIV/AIDs [22]. Spironolactone, steroids, alcohol, cimetidine, heroin, marijuana, and methamphetamine are potential causes [23]. Paradoxically it would seem there is a high incidence of gynecomastia in athletes taking large doses of anabolic steroids [24].

Cancer of the liver and lung can produce estrogen-like hormones and gynecomastia at any age. It is when gynecomastia is unilateral that there is a high suspicion of cancer in boys and men. Evidence of a neoplastic origin is rocklike hard induration and discharge or bleeding from the nipple, although none of these clues may be present. The possibility of a breast tumor is all the more likely in the presence of a testicular mass of unknown etiology.

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Heart

18

At this point in the physical examination, evaluation of cardiac function has already been performed. The clinician has assessed color of extremities, peripheral temperature and moisture, quality of the arterial pulsation, signs of venous distention, blood pressure, and respiration. These parameters reflect what the heart does, not what it sounds like. Again as a reminder, to evaluate the health of a tree, first examine the leaf (periphery). Focus is now directed to the trunk (heart).

Essentials
Palpation
Auscultation
Rate
Rhythm
Output

Palpation

To start, palpate the point of maximal impulse (PMI) of the apical. It is the most reliable physical sign for accessing left ventricular enlargement [1]. The impulse may be more easily detected with the patient leaning forward. Whether caused by hypertrophy (from hypertension or endurance exercise training) or by dilation (in myocardial failure), the PMI is displaced

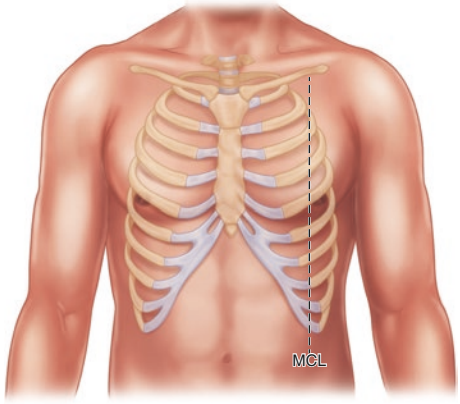

Fig. 18.1 Midclavicular line

to the left of the midclavicular line. For reliability, this line is centered half way between the sternoclavicular and the acromioclavicular joints, *not* at the position of the nipple (Fig. 18.1).

Hyperkinetic Apical Impulse

An exaggerated impulse at the cardiac apex often results from an atrial septal defect [2], the most common congenital heart abnormality in adults. The impulse may be strongest in the left lower parasternal area directly over the right ventricle. Most likely, a fixed split second heart sound and a mid-systolic murmur will accompany this finding.

R.E. Phillips, *The Physical Exam*, https://doi.org/10.1007/978-3-319-63847-8_18

When palpated, a high cardiac output from any cause gives a heaving quality. Causes include fever, hyperthyroidism, and volume overload in mitral regurgitation. A thin chest or cardiac displacement from pectus excavatum may also produce a strong apical impulse.

Thrill

The palm placed flat over the precordium may pick up the turbulence of a major cardiac lesion. The duration of a thrill is much longer than the ordinary maximum impulse and it may be continuous. While an easily palpable thrill is diagnostically non-specific, it does indicate a significant abnormality, whether a septal defect – atrial or ventricular – or regurgitation or stenosis of a valve.

Auscultation

Interpreting heart sounds has traditionally been a premiere focus of the clinician's training. Certainly, the stethoscope has assumed the iconic and ubiquitous symbol of the modern clinician. Ultrasound technology, on the other hand, provides an amazingly higher degree of reliable details on cardiac structure and performance. Even so, in the everyday practice of medicine, the present-day reliance on cardiac ultrasonography in no way diminishes the importance of careful auscultation by the well-trained ear with a stethoscope.

Listening to breath sounds before listening to the heart has its advantage: the mild hyperventilation during deeper breathing is followed by a short period of reduced depth and frequency of breathing. Respirations overlay heart sounds to a lesser degree.

Cardiac auscultation over four points (right and left upper sternum, left lower sternum, and left apical area) is generally adequate when there is neither murmur nor history of heart disease or cardiac symptoms. Should a murmur be detected or if there is suspicion of a cardiac problem, further evaluation is indicated using various positional and respiratory maneuvers. A third or fourth heart sounds, if detected, may be as important in both diagnosis and prognosis as are split sounds. Each component will be covered in brief.

The first and second heart sounds, both relatively high-pitched, are best heard using the diaphragm of the stethoscope pressed firmly against the chest. For optimal listening to the lower pitched and softer third and fourth heart sounds, the bell is recommended; it should be held very lightly against the skin. To hear these "extra" heart sounds, a quiet setting is required. They are often best heard over the cardiac apex with the patient lying or leaning on his or her left side.

All heart sounds may be faint in myocardial insufficiency and pericardial effusion. They tend to be distant also among the very sedentary as well as in the extremely obese and the emphysematous chest.

First Heart Sound (S1)

The first heart sound (S1) in the "lub-dub" sequence is from sudden redirection of blood flow after closure of the mitral and tricuspid valves that vibrates the whole myocardial mass [3]. Tension on the chordae tendineae may contribute tonal quality. The sound becomes more intense in the presence of high cardiac output (anemia, thyrotoxicosis, late pregnancy) and after strenuous exercise. The first heart sound is normally much shorter than the second. At fast heart rates, however, these sounds tend toward equal duration and take on a "tic-toc" pattern.

Systolic Murmur

Systolic murmurs are very common. Separating the "innocent" from the "pathological" is not always easy or reliable. In general, a systolic murmur is more likely to represent a significant cardiac abnormality if:

1. It is loud.
2. It continues through most of systole.
3. There is a precordial thrill.
4. It does not elicit a vigorous arterial upstroke on palpation of a peripheral artery [3].

Second Heart Sound (S2)

Closure of the aortic and pulmonary valves initiates the second heart sound (S2). Closing of the aortic valve is best heard just to the right of the upper sternum, while that of the pulmonary valve is best heard just to the left.

The sound of aortic valve closure becomes more intense in the presence of systemic hypertension. In pulmonary hypertension, closure of the pulmonary valve is louder. The difference can be detected only when S2 is audibly split. Stiff or stenotic semilunar valves will reduce the intensity of the second heart sound.

Usually, the aortic valve closes an instant before the pulmonary valve closes, rendering the sounds almost superimposed. Even slight asynchronous closing of the semilunar valves will result in the splitting of the second heart sound. This split sound can be used to diagnostic advantage by implementing exaggerated respiratory maneuvers. There are three possible changes and interpretations:

1. *Physiological*
   The splitting becomes more separated during full inspiration. This response is completely normal, caused by delayed closure of the pulmonary valve in the more plethoric lung. On full exhalation, splitting is shorter or disappears altogether.
2. *Paradoxical*
   When split S2 sounds come closer together in full inspiration and separate more widely with forced exhalation, an abnormal cardiac event is identified. A preeminent cause is slow emptying of the left ventricle and a reduced ejection fraction. Other possible causes include left bundle branch block, severe aortic stenosis, and reduced myocardial contractility.
3. *Fixed*
   If neither inspiratory nor expiratory maneuvers produce a perceptible change in the interval of a split second heart sound, the cause is most likely an atrial septal defect. Other possible causes, however, are a right bundle branch block or stenosis of the pulmonary valve.

Third Heart Sound (S3)

A third heart sound is commonly heard in youthful subjects and after vigorous exercise. The presence of S3 in middle age and beyond is, however, abnormal. It indicates rapid ventricular filling associated with high cardiac output or congestive heart failure. It also occurs after severe regurgitate flow of the mitral and tricuspid valves.

S3 is best heard with the patient lying in the left lateral position. The cadence developed with the third heart sound is often taught as the "Ken-*tu*-cky" gallop, the "cky" representing S3 that occurs shortly after S2. The sound is low-pitched and much softer than the preceding sounds of the cardiac cycle.

Fourth Heart Sound (S4)

An S4 represents low compliance of the ventricular myocardium. It occurs in hypertrophy of the left ventricle and from stiffness of the chamber wall after myocardial injury. Coming late in diastole, it develops a rhythm verbalized as the "*Tenn*-es-see" gallop. The fourth heart sound is definitely abnormal.

Valves

It is likely that the subject of greatest focus in the basic physical examination course concerns the cardiac sounds and, more specifically, on the normal and abnormal sounds produced by individual cardiac valves. Because of this emphasis, descriptions of the various valve defects will not be covered here – with one exception, explained next.

It is with great admiration of the pre-ultrasound cardiologists that the origin of nearly all the abnormal cardiac sounds had been worked out before the anatomical dynamics could be directly visualized in motion by ultrasound. The most notable exception is the redundant (or floppy) mitral valve. The sound it projected was long called the "innocent murmur" or it was attributed to the murmur of childhood or pregnancy.

Worried parents often heard "He'll grow out of it." Here, the dominant abnormality lay more in the movement of the valve than in its anatomy. It is this exception of mitral valve prolapse that is described here in some detail.

Mitral Valve Prolapse

The two flaps of the mitral valve butt together during contraction of the left ventricle, shutting off blood flow to the left atrium. The valves are prevented from prolapsing into the atrium by chordae tendineae that are attached to the edges of each cusp and to the tip of the papillary muscles (Fig. 18.2).

Prolapse of the mitral valve is a common condition that causes no clinical issue throughout life in the large majority of those with it. Indeed, it is only when mitral regurgitation is present that symptoms are likely to develop [4], and significant impairment of cardiac function can result [5]. Symptoms attributed to the syndrome ranged from dyspnea, palpitations, and chest pain to anxiety attacks, dizziness, and chronic fatigue.

The pathology of mitral valve prolapse consists of (1) enlargement of either or both of the two valve leaflets in the mitral ring and/or (2) excessive length of the chordae tendineae that normally limit the movement of the valve cusps. Echocardiography reveals thickened valves and flailing of the leaflets. These changes are found in somewhat less than 1% of the general population [6]. They are more common in inherited diseases of connective tissue such as Marfan's syndrome, Ehlers-Danlos syndrome, and osteogenesis imperfecta and in polycystic kidney disease. Rupture of a chorda tendinea (the "parachute lines") will also result in prolapse of a leaflet of the mitral valve (Fig. 18.3).

With mitral valve leaflets too large and floppy or overstretched chordae tendineae, there is incomplete closure during ventricular systole. The cusps of the leaflets may bulge toward or well into the left atrium, resulting in marked regurgitation of the mitral valve.

When is mitral valve prolapse not "innocent"? A small minority of people with a floppy mitral valve endure lifelong, recurring, or sustained supraventricular arrhythmias (particularly atrial fibrillation). Worsening dyspnea and palpitations are well-documented complications associated with severe mitral regurgitation [7]. The chordae tendineae, which share in the myxomatous structure of the valves, are prone to rupture, causing flailing leaflets and marked regurgitation. Rarely, the tip of a papillary muscle (where chordae tendineae are anchored) is evulsed, a complication of acute myocardial infarction.

The typical blowing murmur of mitral valve prolapse occurs late in systole. The murmur of regurgitated flow is highly variable in intensity

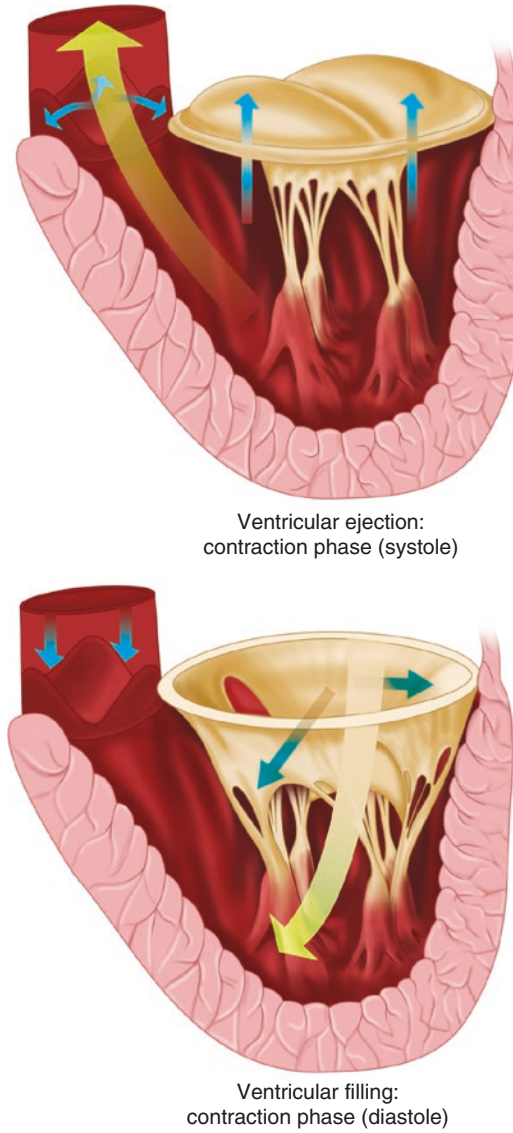

Fig. 18.2 Mitral valve, normal (Modified from Phillips [28])

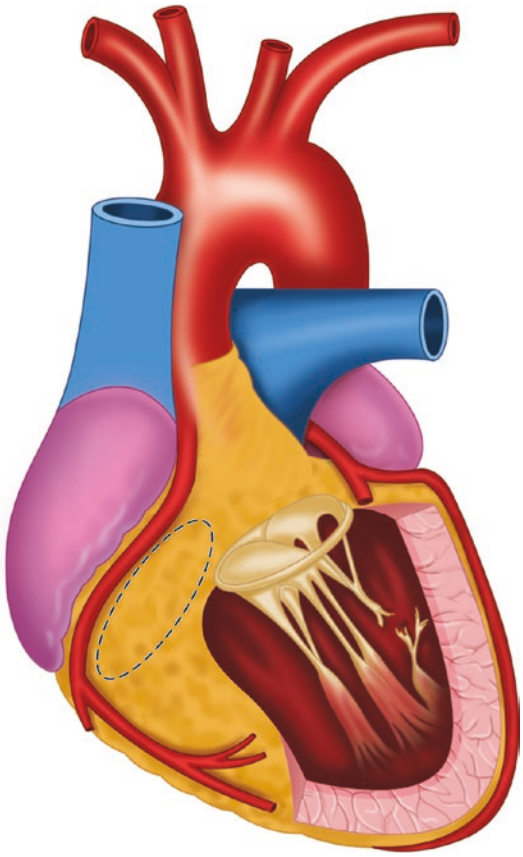

Fig. 18.3 Mitral valve prolapse (Modified from Phillips [28])

and quality. Indeed, evidence of mitral valve prolapse on auscultation can vary immensely according to activity, position, and other factors.

An identifying feature of mitral valve prolapse is a high-pitched click that occurs between the first and second heart sound. It is caused by the abrupt tension placed on the prolapsed valve and the elongated chordae near the end of left ventricular contraction. Rarely, the click is heard without the murmur in the asymptomatic person. In mild cases, the murmur and click may even come and go.

To best elicit the typical late systolic murmur and the click of mitral valve prolapse, listen at the cardiac apex during maneuvers that suddenly and transiently reduce left ventricular volume. Sudden standing from a squatting position or Valsalva's maneuver will, in this way, intensify the systolic murmur of the regurgitate flow and shift the timing of the click to a little later in systole.

Position-Related Symptoms

If a patient has dyspnea or pain in a certain position, the examiner should duplicate that position to listen. A pericardial rub may be heard only with the patient bending forward or lying on one side, coinciding with the position that is most symptomatic. Establishing the link between a symptom-producing position and an objective finding is a critical diagnostic step. The atrial myxoma, described next, is particularly noteworthy.

Myxoma

A rare but never-to-be-missed cardiac problem is the atrial myxoma. They are usually in the left atrium, although right atrial myxomas do occur [8]. Myxomas in a ventricle have also been reported [9]. The possibility should cross the clinician's mind in any patient who develops acute dyspnea or light-headed in a certain position, such as leaning forward. Most myxomas are pedunculated on a stalk and are therefore mobile. In certain body positions, they can severely impair blood flow between the cardiac chambers. There is no murmur typical of a myxoma but any abnormal heart sound produced by it will vary according to the body position. Diagnosis depends upon a strong suspicion and an echocardiogram.

Because a myxoma is a gelatinous tumor, it is friable and prone to breaking up. The result is a shower of emboli to many areas of the body. Necrotizing fasciitis of a limb from a compartment syndrome complicates massive embolization. These potential consequences emphasize the importance of suspecting a myxoma from the patient's symptoms and their reproduction during the physical examination.

Intra-atrial Thrombus

A large thrombus in an atrium may be mobile. By producing symptoms that are position-related, it can simulate a pedunculated myxoma. Of course, disintegration of the clot with embolization is a constant threat.

Rhythm

The cardiac rhythm is better accessed by auscultation than by palpation. Listen for rhythm dynamics at the point of the most distinct heart sounds.

Normal Sinus Rhythm

The sinus node does not depolarize at constant intervals. Under control of the autonomic nervous system, the rate slows and speeds slightly during inspiration and exhalation. Respirations produce changes in volume and pressure in the right atrium, the seat of the sinus node. Variation of rate during normal, quiet respirations, however, is usually too slight for ordinary detection. "Sinus arrhythmia" is an exaggeration of this phasic pattern; it is most common in the youthful and in endurance-trained athletes.

The exquisite sensitivity of the sinus node to changes in respiration, body position, and circulatory demands is a marker of normal autonomic control. A rapid method for confirming normal sinus rhythm is to exaggerate this rate change by having the patient inhale rapidly and deeply. The heart rate will immediately speed up for a few beats, followed by slowing to below the previous resting rate. Continued breath holding will result in a gradual increase in rate. These dynamics that are characteristic of normal sinus rhythm have been described in Chap. 8.

When there is no change of sinus rate in response to a deep inspiration or to Valsalva's maneuver, it can be concluded that an autonomic neuropathy is present, as has been shown in diabetic patients [10]. Heart rate variability from these stimuli can be blunted in diabetic children, demonstrating that autonomic neuropathy occurs early in the illness [11]. Diabetes can impose the burden of such neuropathies even in childhood and adolescence when manifestations may be orthostatic hypotension, intolerance to exertion, gastrointestinal disturbances, urinary incontinence, and desensitized awareness of hypoglycemia [12]. This lack of response correlates with the prevalence and severity of complications such as retinopathy and nephropathy.

Listening for the phasic changes in heart rate in response to deep inhalation and Valsalva's maneuver provides an easily applied tool for assessing visceral autonomic function. The information gained from documenting evidence of neuropathy as it affects the sinus rhythm adds diagnostic support for neuropathy in other systems. For example, in a diabetic patient with acute abdominal distention, the absence of sinus node responsiveness to a respiratory maneuver provides indirect evidence that neuropathic gastroparesis must be considered as causal.

Premature Heart Beats

A premature contraction, by definition, occurs sooner after the previous beat than would be expected. Ventricular filling is reduced during the abbreviated diastolic period, and consequently the heart sound is diminished compared to the previous normal beat. This difference will likely be too slight to detect with a stethoscope. The "compensatory pause" that follows a ventricular premature beat provides a longer duration for filling. In this way, the augmented ventricular volume increases the sound of the post-premature contraction but also increases the intensity of the peripheral pulse. Usually, ventricular premature contractions can be identified by the augmented character of the beat that follows the premature one.

Rate

Normally, the criteria generally accepted for the sinus rhythm in the resting adult is a rate between 60 and 90 beats per minute. These limits do not take into account differences in age, body size, position, body or ambient temperature, anxiety, and exercise training for endurance.

Sinus Node Dysfunction

Sinus node dysfunction is more prevalent in the elderly and in persons with hypertension, obesity, diabetes, and chronic heart or kidney diseases [13]. Encompassed within the popular designa-

tion of "sick sinus syndrome" are sinus bradycardia, periods of sinus arrest, and sinoatrial block. Periods of light-headedness and limitations of exertion are common features of the syndrome.

Postural Orthostatic Tachycardia Syndrome ("POTS")

Chronic orthostatic intolerance with persistent sinus tachycardia while the blood pressure remains normal is labeled the "postural orthostatic tachycardia syndrome." The syndrome occurs predominantly in young women who otherwise appear healthy. Typically the heart rate rises excessively on standing to 120 or more beats per minute. There may be no significant reduction of blood pressure. Light-headedness, palpitations, nausea, and fatigue are frequent complaints [14].

It has been postulated that the common cause is reduced stroke volume [15] of which there may be one or more contributing physiological deficits. Autonomic neuropathy, hypovolemia, adrenergic overdrive, autoimmune reaction, and decondition have been suggested [16]. International experts on this newly recognized condition have recently published a consensus statement expanding on this constellation of problems [17].

Increased Heart Rate

Sinus tachycardia is a normal response to a demand for increased cardiac output. The point is emphasized that sinus tachycardia is not an arrhythmia. It is a natural response to the physiological call for an increase in cardiac output. The stimulant may be physical exertion, emotional stimuli, elevated body or ambient temperature, reduced blood volume, and generalized or visceral vasodilation.

It is when the sinus node fails to accelerate in support of increased physical activity that limits normal activities such as walking and climbing stairs. Medications – particularly those taken for cardiovascular disorders – can have an appreciable effect on blunting appropriate acceleration of the sinus node. In this way, the elderly are particularly sensitive. Indication for the problem can be quickly assessed by having the patient walk down the corridor and back and simply checking for a change in heart rate.

The clinician must direct attention to the circumstances when the heart does not speed up appropriately. If a patient with influenza has a temperature of 102 °F and a heart rate of 80 bpm, the complication of myocarditis should be suspected. A child who has sustained a minor injury after falling from a tricycle but who has a resting heart rate of 120 may have a ruptured spleen. If the problem is inordinate fatigue or shortness of breath on climbing a flight of stairs, detecting that the heart rate failed to accelerate identifies at least one underlying cause. This possibility emphasizes the importance of examining patients where feasible in the situation that induces symptoms. It also brings up the traditional problem of treating hypertension and heart failure with drugs when the doses of medications are prescribed in accordance with their effects at rest. An example is the use of a beta-adrenergic blocking agent to lower blood pressure; it may prevent adequate cardiac acceleration during physical effort. For the same reason, the drug will shave off the peak performance level of an athlete at maximum effort [18].

Cardiac acceleration occurs with any sustained increase in physical exertion, taking about 3 min to gradually speed up then level off into the "steady state." The time is somewhat less in persons trained in the activity. This pattern is incremental for each increased level of exertion. Normally, when exertion stops, heart rate slows and blood pressure falls within 3 min to the pre-exercise level. Finding tachycardia and elevated blood pressure cannot be explained simply by the long walk from the parking lot to the clinic 10 min before.

Decreased Heart Rate

Sinus bradycardia, usually between 50 and 60 beats per minute, is common among normal teenagers and young adults. It is characteristic of persons at any age who habitually train for long-endurance events such as running, rowing, and biking. This form of exercise is termed

"isotonic," referring to maintaining a low to moderate degree of exertion in which the active muscle masses undergo large distances of contraction and relaxation with little change in power. In contrast, training for strength such as weight lifting does not lower the resting heart rate. Weight lifting is called "isometric" exercise because the heavy exertion involves little change in distance of muscular action. This old terminology, admittedly, is a stretch. The concepts behind it, however, define the differences in cardiovascular responses, and these have an important bearing on daily activities.

Systemic diseases must also be considered in bradycardic syndromes. Foremost is the possibility of hypothyroidism. Other myocardial tissue replacement syndromes causing bradycardia are primary amyloidosis and sarcoidosis. In addition, some infections have a predilection for affecting the sinus node and atrioventricular node. Lyme disease is notorious for producing bradycardia, usually by causing sinus node disease or by interfering with atrioventricular conduction [19]. "Relative" bradycardia (i.e., an inappropriately slow heart rate in response to high fever) is sometimes seen with myocarditis from typhoid fever [20] and Chagas' disease [21].

For patients taking a beta-adrenergic blocking agent for any reason, the optimal dose is generally achieved when the heart rate is maintained at about 50–60 beats per minute. In fact, heart rate is our most reliable gauge of determining the ideal dose. This gauge is particularly useful considering the great range of sensitivity of adrenergic blocking drugs between individuals. Some limitation of heavy, sustained exertion can be expected with such a dose.

Vasovagal Reaction

The sudden withdrawal of vasoconstricting sympathetic tone may explain the hemodynamics of pre-syncope and fainting following strong and prolonged emotional stimuli in otherwise healthy persons. Known as the ill-defined "vasovagal reaction," there is a sudden transition from tachycardia to bradycardia that portends symptoms of light-headedness. This sequence is reproducible in head-up tilt table testing, producing pooling of blood in the lower body that is continued until the subject is symptomatic. It is speculated that the high metabolic activity required to long maintain a heightened adrenergic state becomes dissipated, leaving dominant a strong parasympathetic overtone including vagal stimulation and bradycardia. The frequently associated pre-syncopal symptoms of yawning and paresthesia around the mouth and fingertips suggest that hyperventilation is a contributing feature.

Cardiac Output

Cardiac output is determined by heart rate and stroke volume. The interrelationship of these two factors is the deciding factor in meeting the ever-changing vicissitudes of demand for systemic blood flow. Cardiac output is one of the three functions that determined the circulation.

The normal resting adult has a cardiac output of about 5200 ml a minute. At 80 beats a minute, the stroke volume is 70 ml. There is, however, a wide spectrum owing to differences in body size, physical condition, blood volume, and activities.

High Cardiac Output

By this definition, the normal heart responds to the abnormally large peripheral demand. The common denominator may be increased metabolism, requiring increased rates of cellular gas exchanges and the dissipation of heat as in hyperthyroidism or fever. Severe anemia requires the heart to work harder to make up for reduced oxygen-carrying capacity. An arteriovenous fistula of any appreciable magnitude places a heavy workload on the heart. These shunts may be large but occult, usually occurring at sites of old trauma or surgery. The extensive micro-arteriovenous fistulas of advanced Paget's disease of bone (osteogenesis imperfecta) increase cardiac output by increasing stroke volume while decreasing peripheral vascular resistance [22].

Syndromes with high cardiac output may exhibit high, normal, or low systolic blood

pressures, depending upon the degree and mechanisms of compensation. Vasodilation will characteristically result in a low diastolic blood pressure. With sustained exposure to high heat, for example, the diastolic pressure may approach zero even while the systolic pressure is maintained within the normal range. In this instance, the peripheral pulse can be expected to be very rapid and bounding.

The normal heart can sustain a high cardiac output up to twice the normal capacity. Demonstrating this extraordinary capacity of the heart are field workers who tend crops in very hot climates, even when burdened by superimposed circulatory issues such as chronic anemia from malaria or dehydration, perhaps from chronic diarrheal diseases. College-aged sedentary men increase their cardiac output about four times on strenuous physical effort for short periods [23]. Cardiac output of endurance-trained athletes can double that.

Low Cardiac Output

Low output represents inadequate cardiac performance of any component, whether pericardial, myocardial, or defects of valves. Hypotension is expected and, in the extreme, cardiogenic shock, the result of failure of the heart to provide sustained circulatory support to the bulk of the cell mass.

In addition to failure of the central pump, "relative" low cardiac output can be the inability to sustain the demands of the peripheral circulation. Asynchrony of these interactions between central and periphery functions is the clinical feature of orthostatic hypotension.

Orthostatic Hypotension

Standing immediately increases distal blood pooling by 1/2–1 L in the dependent area compared with the supine position. To adjust, the heart rate and stroke volume increase immediately. Peripheral vasoconstriction occurs more slowly. During this orthostatic adjustment, systolic and diastolic pressures tend to fall but normally return to usual levels within a minute.

When orthostatic adjustments are blunted, postural symptoms frequently occur. Common is the light-headedness on first arising after sleep. Particularly prone to this symptom are persons on antihypertension medications, some of which may retard cardiac acceleration or vasoconstriction in dependent areas on first standing. With diuretic therapy to reduce blood volume, any or all three determinants of the circulation are affected.

Symptoms from orthostatic hypotension will occur when venous return cannot be augmented by periodic contractions of the leg muscles. Examples are a soldier standing at attention or a patient on a high-angle tilt table for many minutes. Observed events that lead to fainting from a sustained, immobile position is the sudden transition from a compensatory tachycardia to severe bradycardia.

Postural hypotension is exaggerated in a hot environment. Almost everyone has experience the brief period of light-headedness on first standing after lying or sitting in direct sunshine for a long time. Vasoconstriction in position-dependent areas is slow to offset the sudden redistribution of blood.

Neuropathy

In addition, reduced tolerance for upright activity can represent an autonomic neuropathy. Cardiac acceleration and peripheral vasoconstriction to positional changes are compromised. Such may be common in chronic alcohol abuse and in diabetes. A rare and highly disabling dysfunction of autonomic regulation is "multiple systems atrophy" (formerly known as the Shy-Drager syndrome) in which orthostatic hypotension is a major feature. It is a neurodegenerative disease affecting mainly the brain stem [24]. In addition to postural intolerance are anhidrosis, disturbances of gastrointestinal motility, and urinary retention.

In Parkinson's disease, postural hypotension is common; bradykinesis and rigidity of the limb support this diagnosis [25]. A recent finding on these patients reveals a substantial deficit in memory and executive functions in those who exhibit some degree of orthostatic hypotension when studied with tilt table positions [26]. The

finding emphasizes the need to use drugs with cardiovascular effects (or side effects) with due regard to their action during upright activity.

Cardiac Tamponade

Cardiac tamponade should be suspected when heart sounds are diminished, cervical neck veins are engorged in the semi-recumbent position, blood pressure is at a hypotensive level, and there is a paradoxical pulse [27]. The diagnosis of acute pericarditis is further defined if pleuritic chest pain in certain positions and a pericardial friction rub are present. Tamponade may be from a pericardial effusion (including hemorrhagic) or fibrosis with thickening. Chapter 19 provides additional description of these conditions.

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Part III

Patient Lying in 30°, Semi-supine Position

Neck: Semi-supine

19

Position #3 The patient lies back into a semi-recumbent position. The head of the bed or examining table is elevated to 30°. This second evaluation of the neck focuses on the major blood vessels. The position is also advantageous for examining the axilla.

Essentials
Carotid pulsations
Jugular vein filling
Axilla

The large cervical blood vessels closely reflect the dynamics of cardiac inflow and outflow. The signs can be subtle, however, and their interpretation – so finely honed by doctors of the pre-imaging day – is now less in demand. Even so, being adept at identifying some basic features of arteries and veins in the neck is essential for the modern clinician.

Carotid Arteries

Palpating each carotid artery provides a qualitative sense of cardiac dynamics as well as arterial compliance. The preferred place to palpate is over the bifurcation located just proximal to the mandibular angle. The carotid body is located in this area so that it is important to apply gentle pressure to avoid overstimulating the vagal nerve (Fig. 19.1).

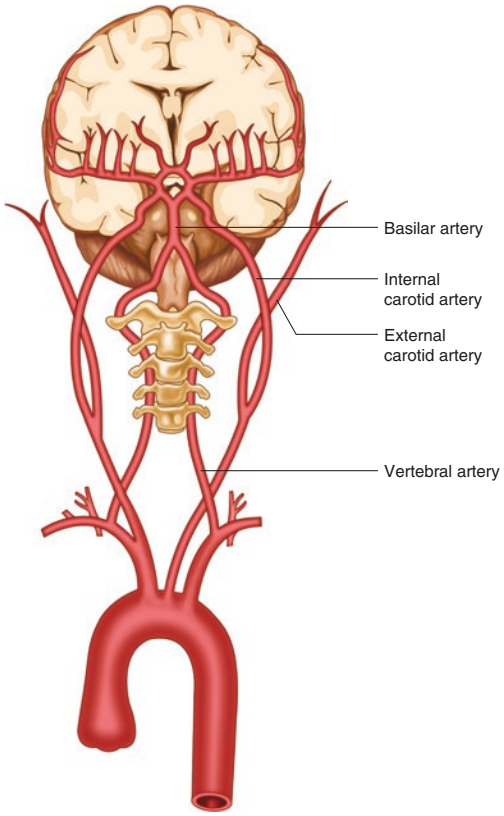

Fig. 19.1 Carotid artery (Modified from Phillips [21])

For the examination of the carotid arteries, it is advantageous that the patient to be in a semi-recumbent rather than a sitting position. In the rare instance of a supersensitive vagal nerve reflex, even a light touch from palpation may

R.E. Phillips, *The Physical Exam*, https://doi.org/10.1007/978-3-319-63847-8_19

induce symptomatic bradycardia from a drastically slowed down sinus rate or inhibited atrioventricular conduction. Such hypersensitivity in otherwise asymptomatic elderly people can produce brief periods of asystole or a precipitous fall in blood pressure [1].

If a friable atherosclerotic plaque is present, kneading the carotid artery could release emboli and set off a cerebrovascular event [2]. While such complications are quite uncommon, they are real [3]. The value of palpating the carotid arteries is questionable in the relatively youthful patient with no symptoms and no known cardiac risk factors.

Hyperkinetic Pulse

An increase in stroke volume produces a bounding carotid pulse. The finding is expected in states of increased left ventricular stroke volume. Fever, a high level of anxiety, and vigorous exercise are common causes. Cardiac abnormalities – reflux of mitral and aortic valves and a large intraventricular septal defect – can also produce an exaggerated arterial pulsation. Reduced diastolic pressure accentuates pulsatile force, which is like that occurring in high ambient heat and in aortic valve insufficiency. In the extreme form, aortic regurgitation gives the classic "water hammer" pulse [4]. Peripheral conditions associated with rapid arterial runoff, such as a major arteriovenous fistula or patent ductus arteriosus, also increase stroke volume.

Athletes trained for endurance may have a bounding carotid arterial pulsation that reflects an increased stroke volume at rest. In order to maintain cardiac output, it is the mechanism that compensates for the marked resting bradycardia associated with endurance training.

A strong pulsation at or around the arterial bifurcation can actually be from a kink in a highly tortuous artery, usually that of an elongated internal carotid artery [5]. The hyperkinetic pulse can also be from an aneurysm [6]. There appears to be no link of tortuous cervical arteries to atheromatous disease. Sorting out these causes of an accentuated cervical pulsation is beyond the capacity of a physical examination and relies on imaging studies.

Hypokinetic Pulses

A delayed upstroke of the carotid artery and a diminished peak indicates some form of compromise in stroke volume. The most common cause is left ventricular failure. Hypertrophic obstructive cardiomyopathy, severe mitral stenosis, and pericardial constriction or tamponade may also be responsible. Systemic causes are severely reduced circulatory volume (anemia, blood loss, or visceral vascular "pooling"). Weak cervical pulsations from these conditions are, of course, bilateral.

The finding of a reduced carotid pulsation only on one side most likely indicates arterial stenosis. In the vast majority of cases, atherosclerosis is the cause [7]. Transient ischemic attacks and stroke – focal or non-focal – are most often from emboli arising from friable atheromatous plaques near the bifurcation. Severe arterial obstruction from thrombi and low-flow stenosis are far less common (Fig. 19.2).

There is a much greater incident of carotid artery stenosis in patients taking cholesterol- lowing medications [8]. Also sharply increasing the possibility of obstruction here in stable ambulatory patients is the presence of hypertension, peripheral arterial disease of the lower limbs, and coronary artery stenosis. An older age at the time of onset of symptomatic coronary arterial disease increases the likelihood of stenosis of a coronary artery. There is, in fact, a high incidence at 65 years old and older even without identified risk factors.

Fibromuscular dysplasia may rarely be responsible for carotid arterial stenosis [9]. This etiology is demonstrated by angiography as a "string of pearls" in the internal carotid artery.

Bruits

Blood flow may be turbulent enough to be audible. Listening for a bruit at or near the bifurcation

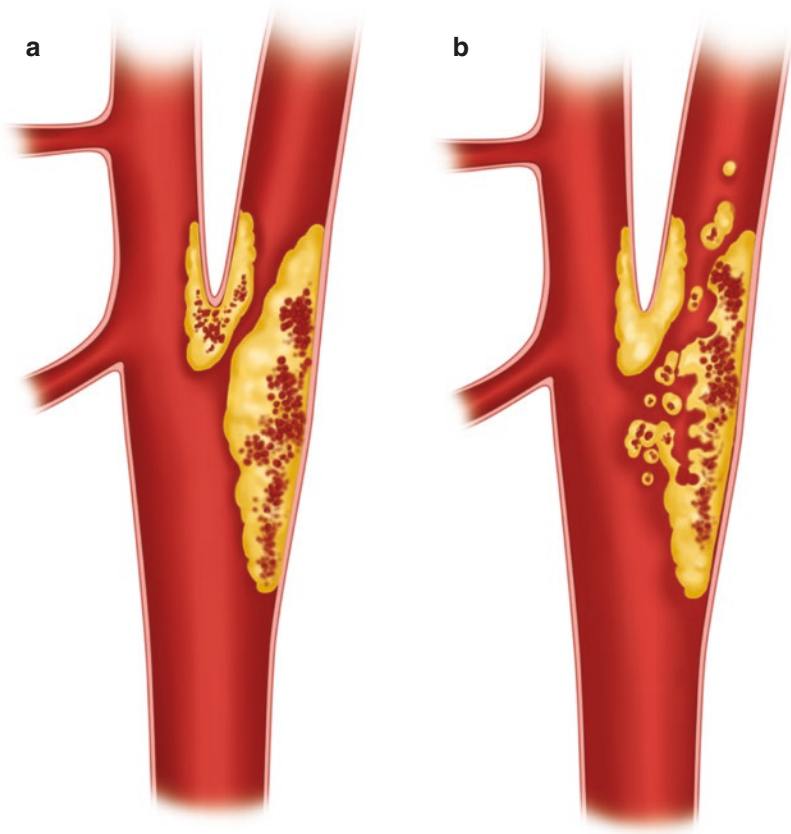

Fig. 19.2 (**a**) Carotid artery: atheroma (Modified from Phillips [21]). (**b**) Carotid artery: atheroma rupture (Modified from Phillips [21])

of the common carotid artery should be performed if a unilateral reduction of pulsation is detected. It is also prudent to check for a cervical bruit whenever there is a suspicion by symptoms or sign of a cerebral vascular event or substantial risk factors for atherosclerosis. An arterial bruit is present over the majority of carotid arteries with significant occlusion. At the same time, it must be emphasized that the absence of a bruit here does not rule out any degree of arterial stenosis.

Placing the bell of the stethoscope under light pressure in the supraclavicular fossa – which is close to the bifurcation of the common jugular vein – is most effective place to listen for neck bruits. A bruit may be loudest directly over the obstructed artery, but sometimes the sound is heard only more distally. Bruits are primarily systolic but sometimes extend into the diastole [10]. With advancing obstruction, a bruit will become higher pitched, louder, and more prolonged. Most bruits originate in the internal carotid artery. Those confined to the external carotid artery signal no potential danger. Atheroma in either branch, however, forewarns of the possibility of obstructive lesions in major arteries elsewhere.

The murmur of aortic stenosis is usually transmitted to the neck. Careful auscultation over the cardiac apex and extended to the neck will sort out the murmur from a bruit. Differentiation of an arterial bruit from a venous hum, often normal in children, is simply done by obstructing flow in the jugular vein by slight, direct compression.

A bruit over the common carotid artery area is most likely from a stenotic atheroma that can

extend well up into the internal carotid artery. In one large study, less than half of those referred with a cervical bruit had significant stenosis [11]. The minority of cervical bruit comes from turbulence and stenosis in the external carotid artery where an atheromatous plaque does not pose the danger of causing a stroke. High blood flow conditions such as hyperthyroidism and anemia can produce a cervical bruit as can an arteriovenous fistula, including one constructed in the upper arm for periodic hemodialysis. Rarely, a bruit can be heard when there is no vascular anomaly demonstrable by imaging studies.

The absence of a cervical bruit does not rule out the possibility of carotid arterial stenosis. None may be detected when the degree of obstruction is high [12].

Arterial Impulse Patterns

Failure of the left ventricle, when severe, may result in peripheral pulses that alternate in force from beat to beat. In a normal sinus rhythm, this phenomenon is called "pulsus alternans." Certain arrhythmias, for example, ventricular bigeminy, can produce the same alternating pattern.

Normally, deep inspiration results in a momentary reduction in the upstroke of the artery. This reduction is greatly exaggerated when cardiac expansion is limited because of constrictive pericarditis or cardiac tamponade. A similar phenomenon may occur in obstruction of the superior vena cava and in obstructive pulmonary disease. It is referred to as the "paradoxical pulse." Paradoxical refers to obliteration of palpable peripheral pulses on deep inspiration when cardiac tones remain audible. This phenomenon is defined as a fall of 10 mm/Hg or more in systolic pressure on full inhalation [13]. Obviously, detecting this change with reliability requires careful measurements of blood pressure.

Irregular cardiac rhythms will cause a beat-to-beat difference in both the force of the carotid impulse and the intensity of a bruit heard over it. Atrial fibrillation is a common example.

Jugular Veins

When the body is horizontal, pressure in the cervical veins directly represents that within the right atrium. At 30 degrees of upper body elevation, the proximal portion of the external jugular vein just above the clavicle may be normally distended. At higher degrees of trunk elevation, the force of gravity will normally drain the cervical veins completely (Fig. 19.3).

The level of the venous blood meniscus in the neck will fluctuate with each heartbeat, rising on right atrial contraction. The level falls with each inspiration and sharply so with a deep breath. Observed carefully, the neck veins can serve as a reliable site to assess right-sided cardiac filling pressure. Turning the head slightly away from the veins examined is helpful for observation. The dynamics of venous filling and emptying reflects the interrelated functions of blood volume, myocardial contractility, and competence of the tricuspid valve.

The master clinicians of a previous era learned to sort out five distinct components of the venous pulsatile waveform. Various imaging procedures now available have made such detailed observations superfluous, however admirable. Nevertheless, certain basic observations of veins in the neck should, however, be an integral part of every comprehensive physical examination.

Observe the external jugular veins for distention. On the surface, the external jugular vein is more visible than the deeper internal jugular. Normally, what distention may be present

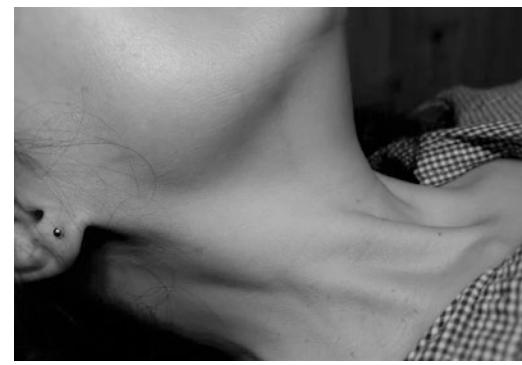

Fig. 19.3 Jugular vein distention: normal

proximally will immediately disappear when the patient takes a deep breath. Exhalation, even from speaking, increases right atrial filling pressure and causes the jugular veins to distend slightly. Exaggeration of this normal phenomenon occurs with strong exhalation against closed pressure, as in Valsalva's maneuver. Distention is particularly obvious when the soprano in an opera hits that high note.

Curiously, the cervical veins have valves, the anti-gravitational system that is essential in the legs [14]. The purpose of valves in the jugular veins is not known, although they possibly protect the brain from intermittent high pressures such as that developed on coughing.

Venous Distention

If the veins are distended above the lower third of the neck a 30° from recumbency, it is assumed that there is an increase in right atrial pressure. Alternatively, there could be some restriction of venous blood entering the atrium as in the "vena cava syndrome."

Ventricular insufficiency (either right- or left-sided myocardial failure) is the most likely explanation when compression of the congested liver will cause the meniscus to rise even higher. This maneuver, incidentally, is called the "hepato-jugular reflux," not reflex. As distention extends into the mid-neck and higher, it is the engorged internal jugular vein that is most conspicuous. The traditional task of measuring the height of the level above that sternal notch is now seldom performed in the general clinical setting (Fig. 19.4).

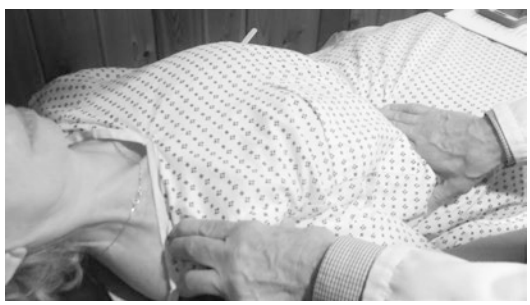

Fig. 19.4 Hepato-jugular reflux

Superior Vena Cava Syndrome

The clinician must be wary of diffusely engorged neck veins as a sign of obstruction of the superior vena cava. Suggestive findings are swelling and plethora of the face and neck. Cutaneous veins over the face and upper body may be distended. Symptoms of dyspnea, headache, and chest or head congestion are generally exaggerated on lying down. When the voice is affected and disorientation, suggesting cerebral edema, is present, the condition dictates urgent management.

Causes of the superior vena cava syndrome are many, but most cases are related to extraluminal compression. The cause is usually an intrathoracic neoplasm of which cancer of the lung is most likely [15]. Additional causes are mediastinitis from fibrosing infections or granuloma-forming conditions such as tuberculosis or sarcoidosis, an aneurysm of the arch of the aorta or constrictive pericarditis. Thrombosis within the subclavian-jugular vein systems occurs in hypercoagulable states as well as by induction from indwelling catheters and the electrode wires of an electronic cardiac pacemaker. Street drugs injected into the jugular veins introduce not only thrombogenesis but also the possibility of mediastinal sepsis. Superior vena cava syndrome from thrombosis has been reported as a presenting complication of bronchogenic carcinoma [16].

Pericardial Restriction

When venous flow into the right atrium is impeded, the jugular veins will not collapse on deep inspiration but rather become more distended. That is, the veins fill higher up on the neck when the semi-recumbent patient takes a deep breath. This phenomenon is a classic sign of constrictive pericarditis and tense pericardial effusion (tamponade). The diagnosis is further secured if a paradoxical pulse is also present; that is, the amplitude of the peripheral pulse is severely obtunded. Sustained pressure over the liver will briefly increase volume in the vena cava and thereby increase distention of neck veins.

Myocardial Failure

Distention of the cervical veins may also occur when right atrial pressure is elevated by myocardial insufficiency. Other causes range from right ventricular insufficiency and tricuspid stenosis to pulmonary embolism. When severe, failure of the left ventricle can also be expressed by venous distention in the neck.

Axillae

Palpating the axilla for lymph nodes in the supine position allows for convenient and rapid access. With the free hand, raise the patient's flexed arm, and then with the examining hand reach high into the apex of the axilla. Using gentle pressure of the fingertips, bring the hand slowly down against the thoracic wall. For patients who are especially ticklish by having someone poking into their armpits, the hand-on-hand technique of palpation, described in Chap. 20, may prove helpful.

Because significant axillar adenopathy may be the first evidence of a neoplastic disease, it is worthwhile performing in every comprehensive examination. It is not necessary to poke one's hand directly into a sweating axilla or to take the time to glove up. Simply use the edge of the patient's gown or bedsheet as a barrier. Palpable lymph nodes will not be masked.

Lymphadenopathy

Enlarged lymph nodes are generally felt as spindle-shaped masses that roll beneath the fingertips. As with other palpable lymph nodes, the texture, tenderness, and mobility of those in the axilla should be defined. Unilateral axillary adenopathy suggests an infection in the hand or arm, pulmonary tuberculosis, or metastasis from the breast. Bilateral lymphadenopathy may result from infection or neoplasm; that is, causes can range from infectious mononucleosis to Hodgkin's lymphoma. In these conditions, it is highly likely that palpable lymph nodes will also be present in the neck.

Acanthosis Nigricans

Mention is made here of a skin condition, acanthosis nigricans, that has a predilection for the axilla although it can appear virtually anywhere, particularly on folds of the skin. It is seen as a darkened patch of skin with a soft but rough texture resembling velvet (the Greek word *akantha* means "thorny"). Papillomatous lesions may be scattered throughout. While generally benign, the condition can be associated with insulin resistance and is known as a surveillance sign for diabetes [17]. An association with the metabolic syndrome has been reported [18]. Children and adolescents who are obese and who have hyperinsulinemia are particularly subject to developing acanthosis nigricans [19]. There is also a more remote association with internal malignancy, particularly adenocarcinoma in the gastrointestinal tract [20] (Fig. 19.5).

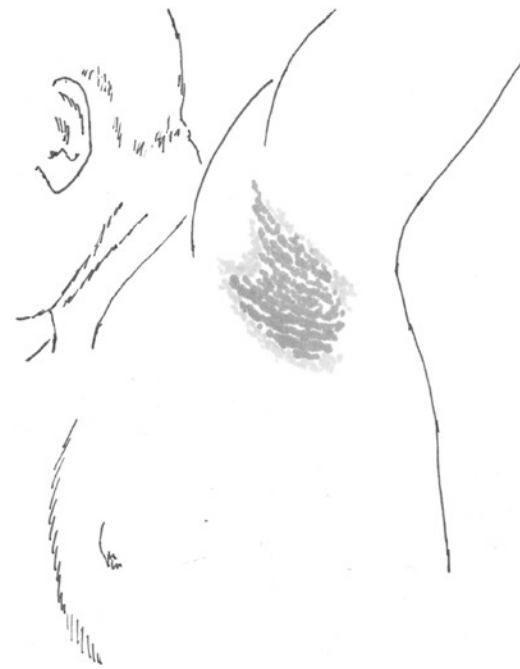

Fig. 19.5 Acanthosis

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Abdomen

20

Imaging has so vastly improved the reliability of identifying abnormalities within the abdominal organs that the modern clinician has come to rely heavily on this advanced technology. The pre-imaging assessment, nevertheless, is still required, since the highly challenging skills of the physical examination determine the significance, urgency, and subsequent workup of an abdominal complaint. Indeed, the "hands-on" examination of the abdomen pertains to both the "acute abdomen" and those conditions with chronic or intermittent symptoms on deciding the next step. This chapter offers an approach to the examination of the abdomen multiple organ systems that may help the clinician recognize and assess pathologies in the abdominal wall and within the abdomen whether they are symptomatic or not.

Essentials
Observation
[Auscultation]
[Percussion]
Palpation

Abdominal Wall

The normal abdomen is traditionally described as "scaphoid" (meaning bowl-like) and has come to imply "normal." It is a term for an ectomorph model, hardly descriptive in a modern population that has had a substantial increase in girth.

Symmetry

Symmetry of the abdomen is usually confirmed by the midline position of the umbilicus. If there is lateral deviation of more than 1 cm from a line between the xiphoid process to mid-symphysis pubis, asymmetry must be explained. If not a scar from surgery (including liposuction) or trauma, suspect that a massive liver or spleen is responsible. Sometimes, however, the intestinal bulk from severe stool retention can present as a "phantom tumor" with distortion of the topography. Rarely, the visible outline of a gas-distended portion of bowel will shift the umbilicus.

Size

Evaluation of an enlarged abdomen usually comes down to differentiating obesity from ascites. Far less likely than either of these – but still a possibility – is either a giant pseudocyst of the pancreas or an ovarian fibroma.

Obesity

The definition of "obesity" should still be objective, relying generally on a Body Mass Index

R.E. Phillips, *The Physical Exam*, https://doi.org/10.1007/978-3-319-63847-8_20

(BMI). The BMI is derived from a chart based upon the relationship between weight and height. The normal index is between 18 and 25. Above 30 represents obesity with the morbidly obese above 40. There are certain characteristics that are important to recognize when examining the very corpulent patient.

When the patient with marked obesity is supine, the abdominal wall remains domed while the adipose layer can be detected extending beyond the flank into the back. The omental layer may overlap, as well, into the inguinal space. A large pannus can obscure the suprapubic region and must be retracted to expose the abdominal wall. Only then can the lower abdomen be properly evaluated, allowing for percussion or palpation of a full bladder.

Ascites

In the adult, physical signs of ascites cannot be appreciated unless there is at least an accumulated liter of intraperitoneal fluid. Thus, it is only when the ascites-producing process is fairly well advanced that it can be detected.

Free fluid in the peritoneal cavity gravitates to the flanks when the patient is supine, causing a bulge, but it does not extend beyond. This finding separates ascites from obesity.

If the volume of intraperitoneal fluid is great enough to cause tense ascites, the abdominal dome may be preserved and the navel protruded. The symmetrical distribution of ascites disappears when the patient turns to lie on one side, making the down side more prominent. Performing "shifting dullness" will confirm the presence of ascites; the procedure is described later in this section under "Percussion."

Ascites from increased hemodynamic pressure implicates a disease of the heart or lungs: congestive heart failure, pulmonary hypertension, or pulmonary valve stenosis. In contrast, ascites caused by portal hypertension from, for example, hepatic cirrhosis is mainly due to the resultant hypoalbuminemia.

Eliciting a fluid wave is perhaps the most reliable method for detecting ascitic fluid, but it needs to be done only if its presence is uncertain. Technically, a tap on one flank of the supine patient is felt as a shock wave from a hand against the opposite flank. Essential in this test is the placement of firm pressure on the mid-abdomen by a third hand, the patient's or an assistant's.

Movement

The wall of the abdomen normally moves with respiration and is easily visible in the supine patient. In peritonitis, such movement may be absent. The lack of movement is sometimes localized to the right lower quadrant in appendicitis and to the epigastrium in acute pancreatitis.

Discoloration

Ecchymosis

Bruising in the abdominal wall is cause for concern. A spontaneous bruise around the umbilicus indicates intraperitoneal bleeding (Cullen's sign). Bleeding into the retroperitoneal space produces ecchymoses that settle into the abdominal wall at the flank (Grey Turner's sign). Historically, these signs have pointed to ectopic pregnancy and acute pancreatitis, respectively [1]. Experience has shown, however, that many sources of bleeding can appear on the abdominal wall [2].

Obvious causes of abdominal wall bruising are physical injury and injected medications, most particularly from heparin. Ecchymoses can occur in hypocoagulable states. They are certainly more common in the anticoagulated patient even when coughing [3], and abdominal exercise [4] can induce bleeding into the abdominal wall. Bruising here has been reported after procedures: percutaneous liver biopsy [5], paracentesis [6], coronary angiography [7], and percutaneous coronary intervention [8].

The most notable inflammatory condition that causes ecchymoses in the flanks is acute pancreatitis in which there is a strong hemorrhagic component. Amebic abscess has been reported to produce periumbilical bruising [9]. Malignant

diseases of the pancreas, ovary, liver, and kidney can produce hemorrhagic ascites and abdominal wall bruising. Other causes are advanced portal hypertension, strangulation of bowel, ruptured ectopic pregnancy, splenic rupture, and perforated duodenal ulcer The most famous case of an ecchymosis occurred in the only known survivor of a meteorite strike, hit in the abdomen [10].

Venous Pattern

Visible veins in the upper abdominal wall will normally drain toward the head. Veins in the lower wall drain toward the feet. Tracking these distributions has utility in identifying certain major vascular disorders.

Distended Veins

In obstructive diseases of the superior vena cava, the veins in the upper wall of the abdomen become particularly prominent. Those in the lower wall are most conspicuous with obstruction of the inferior vena cava. The veins have extensive collateral connections so that filling from the direction opposite to the usual refilling direction occurs. Manually emptying an engorged abdominal wall vein with compression and observing its direction of refilling can help determine if the obstruction is in the superior or inferior vena cava (Fig. 20.1).

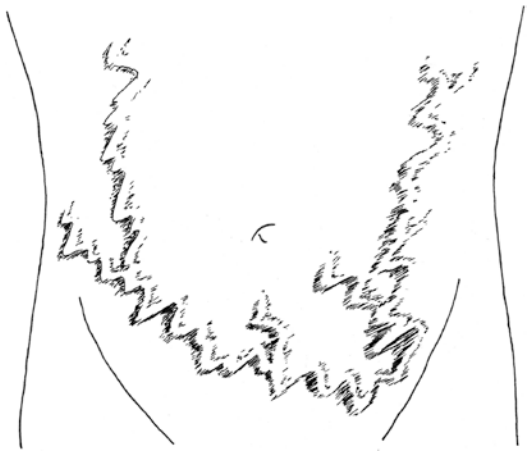

Fig. 20.1 Abdominal veins

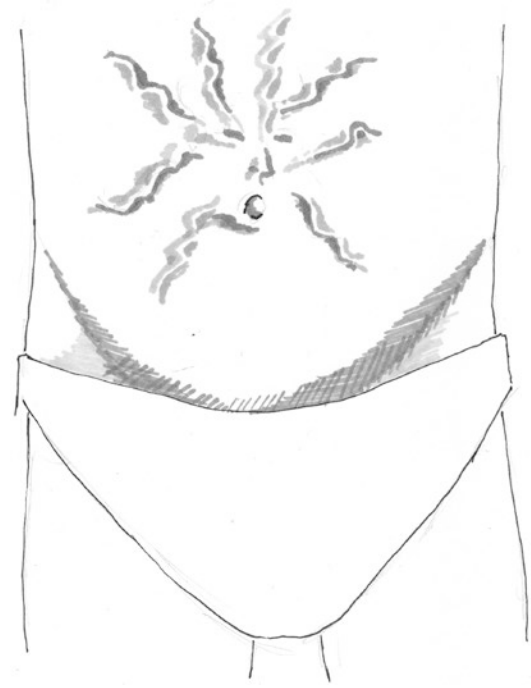

Fig. 20.2 Caput medusa

Portal Hypertension

In portal hypertension, veins tend to be prominent around the umbilicus. When in the advanced stages, the collateral venous circulation in the abdominal wall may be massive. The term *caput medusae* is restricted to veins that radiate from the umbilicus, not just pass by it. The name comes from ancient Greek mythology, referring to the evil goddess who had snakes rather than hair radiating from her head (Fig. 20.2).

Coarctation

Coarctation of the aorta leads to the development of pronounced veins over the entire abdominal wall while at the same time the extra-aortic arterial circulation develops to support the lower body. This is a rare but serious condition that can be overlooked through much of a lifetime. Patients learn to cope with coarctation by avoiding exertion that involves the legs; they can experience distressing complications during the challenges of illness, pregnancy and surgery.

Among the telltale signs of coarctation of the aorta are robust arterial pulses in the warm upper

extremities in contrast to feeble ones in the cool lower extremities. Treatment resistant hypertension is an expected feature. Low blood pressures and weak pules taken at the ankle are in sharp contrast to those in the arm. The subject is described in Chap. 8 Hypertension.

Scars

Any abdominal scars from surgery or trauma should be noted. These include the short and multiple scars from laparoscopic procedures. A curvilinear scar across the lower abdomen is likely to be from a Caesarean section (called a "Pfannenstiel's scar"). The presence of any scar substantially increases the possibility of an intra-abdominal condition being caused by adhesions of bowel. Volvulus, intussusception, and an internal intestinal hernia are examples of such complications.

Striae

Stretch marks are commonly found on the abdominal wall and are caused by disruption of elastic fibers. At first they are erythematous streaks, but over time they become a whitish or silvery scar. They are most likely to follow pregnancy, a major loss of weight, or tense ascites. In the over-stretched skin of pregnancy, the normal elastic fiber network is greatly disrupted and subsequent repairing fibers are highly disorganized [11].

Striae produced by steroid therapy are generally wider and more widespread. A purplish hue suggests hyperadrenalism, owing to a loss of the structural integrity and thinning of subcutaneous tissues [12]. It is the androgen activity of the overactive adrenal gland that causes striae. Thus, purple striae are present either from excessive ACTH stimulation (Cushing's syndrome) or by spontaneous adrenal hyperplasia from a functioning adenoma. Glucocorticoid therapy with, say, prednisone (which lacks androgenic activity) does not cause striae to become purple.

Bowel Sounds

Listening for bowel sounds in a patient without gastrointestinal symptoms or other abdominal complaints is so unlikely to provide useful information, there seems little productivity in taking the time. On the other hand, when either symptoms or suspicions of intraperitoneal disease are present, the frequency and character of bowel sounds may yield helpful clues to both the diagnosis and its urgency. Furthermore, listening for an abdominal bruit may be productive when considering causes of hypertension.

Since palpation may affect bowel sound activity, auscultation is performed first. Normally, bowel sounds are heard every 4–10 s but can be very infrequent, especially between meals. The clinician needs to listen only in one area, such as the mid-abdomen, as bowel sounds are carried widely. The clinician should be aware that the reliability of bowel sounds for boosting confidence in a presumptive diagnosis is poor, as will be noted in the descriptions to come. More important than bowel sounds for determining gut motility is the passing of flatus and stool. These functions are especially pertinent to detect restoration of bowel activity during the postoperative period.

Increased Bowel Sounds

Frequent high-pitched "tinkles" suggest at least partial obstructive disease of the small bowel. The absence of tinkles, however, does not mean that bowel obstruction is not present. Silent periods between tinkles can last for several minutes.

Prolonged "rushes" of bowel sounds at a frequent rate usually point to intestinal obstruction. The obstructed site could be anywhere along the way. Alternately, diarrhea from all causes could be responsible.

Decreased Bowel Sounds

Paralytic ileus has many causes. Before concluding that the bowel is atonic, however, the clinician must listen for bowel sounds for a full 3 min. On the other hand, the absence of audible bowel sounds does not necessarily mean that intestinal activity is absent.

A reduction or absence of bowel sounds can be the result of advanced obstructive disease anywhere along the intestinal tract. Also, uncomplicated diarrheal syndromes can inhibit peristalsis as can ischemia of the bowel and anticholinergic drugs (those with atropine-like activity, taken frequently for an "upset stomach"). Gastroparesis (as a manifestation of autonomic neuropathy in diabetes) is also associated with the "silent abdomen." The detection of autonomic neuropathies upon the physical examination has been described in Chap. 18.

Postoperative ileus is an expected event after both open and laparoscopic surgery. It is generally associated with abdominal distention and tenderness beyond the location of the incision. Ileus may stem from manipulation of the bowel mesentery, use of carbon dioxide gas in laparoscopy, bowel resection, or fresh anastomosis. Bowel contractility returns within minutes to hours after intra-abdominal surgery but coordinated peristalsis among serial bowel segments can take much longer. The passing of flatus and production of a bowel movement provides a much higher degree of confidence in intestinal recovery than bowel sounds.

The bowel without active peristalsis allows gas to accumulate and promotes colonic distension. Patients may complain of mid-epigastric and left-sided pain from distension of the transverse colon. The give-away sign of an air-distended bowel is almost always a tympanic response to percussion.

Bruit

A systolic bruit can sometimes be heard in the epigastrium of normal people. This phenomenon is more commonly detected in the asthenic person, in those with high fever or with marked anemia. Caution must be taken to avoid mistaking the bruit with a murmur radiating from the heart or from the pregnant uterus.

Stenosis of a renal artery as the cause of hypertension is the main reason to listen for an abdominal bruit. If present, it is most likely to be heard in the epigastrium or in the flank. In the elderly, atherosclerosis is the cause of virtually all cases of hypertension-producing renal artery stenosis. In women middle aged or younger, it is most likely due to fibromuscular dysplasia. A saccular aneurysm of a renal artery has been reported in which an abdominal bruit was present but not hypertension [13].

Admittedly, abdominal bruits from renal artery stenosis occur in a minority of cases [14]. Most of the bruits heard in the abdomen are, in fact, caused by stenosis of the celiac artery. These are heard mainly in the epigastrium.

Dissection of the splenic artery or pancreatic cancer can also produce an abdominal bruit. A bruit limited to the periumbilical area suggests stenosis of a mesenteric artery [15].

Arteriovenous malformations and fistulas produce bruits that might be heard anywhere over the abdomen. An assiduous listening for such may be productive in a patient with an unexplained high cardiac output syndrome.

Despite the diagnostic uncertainty of an abdominal bruit, a prudent clinician will listen for a bruit in newly discovered or in treatment-resistant hypertension. Detection can be an early and valuable clue to the possibility of secondary hypertension from renal artery stenosis that would, unrecognized, prove difficult to treat and as well as dangerous [16].

Hum

A continuous humming sound may be present over the abdomen when the collateral venous circulation is massively developed. In portal hypertension, a hum is usually heard best around the umbilicus.

Friction Rub

If a bruit has some characteristics of a friction rub (a scraping sound, particularly if to-and-fro with respirations), the more likely cause is cancer of the liver, gall bladder, pancreas, or disseminated carcinoma. Alternatively, inflammation from granulomatous diseases, such as tuberculosis and sarcoidosis, should also be considered.

Palpation: Abdominal Wall

The abdominal wall is most easily palpated when the patient is warm and relaxed, the knees bent, soles resting on the bed or table, and the arms

lying at the side, not overhead. Of course, it is imperative that the examiner's hands be warm.

Many of the classic signs in the abdominal examination were described and named a century or so before. Modern technologies have found enough exceptions for many of the classic diagnostic descriptions that the eponyms given to them have become less meaningful. Furthermore, they are not easy to keep straight. These historical names are omitted or deemphasized in this text. A few are so entrenched in our vocabulary as to deserve continued recognition.

To palpate the abdomen of the anxious patient, it is helpful to have the patient first take several deep breaths, then continue with normal breathing but now through the mouth. Distracting conversation can add a measure of relaxation. Of course, cold hands of the examiner do not help.

The method preferred for palpating the abdomen at each site is to begin with a gentle laying on of the full hand with slowly increasing pressure. Poking is unpleasant for the patient. It is not any more informative than gentle pressure. Furthermore, it does not save appreciable time.

Some patients are so ticklish that they strongly resist any attempt to palpate the abdomen. It is helpful to ask the morbidly shy or anxious person to place one hand on top of the examining hand as it moves to an area of tenderness or to a perceived mass. The patient could even be given instruction on where to place the palpating hand. This method gives the patient a reassuring sense of control and greatly reduces resistance.

To begin palpating the abdomen, place one palm with fingers spread gently over the central belly for a several seconds. This touching over a sensitive area gives the patient some warning of what is to come, reducing anxiety, and voluntary guarding. With light hand pressure, jiggle the abdominal wall. Any pain or discomfort elicited from jiggling indicates that there is some intra-abdominal irritation and may even point to where pathology is located.

In very young children, some clinicians routinely begin their physical examinations at the abdomen. Children seem curious, non-threatened, and much less likely to cry or resist with a soft, warm hand gently laid on the abdomen than when the examination begins by someone trying to look at the ears or throat or to listen for breath and cardiac sounds with instruments.

Texture

Reliable conclusions derived from different textures of the abdominal wall require well-seasoned experience. The markedly obese abdomen may have a feel of kneading dough, but it bounces back immediately on release of pressure. This reaction is in contradistinction to the less pliable, doughy texture of myxedema. Edema associated with ascites maintains a pitting quality after release of digital pressure. In dehydration, the skin of the abdominal wall is easily "tented" by pinching; it remains so for an excessively long time. Tenting, however, can also be observed in the abdominal wall of advanced age, having lost appreciable elastic fibers.

A grainy texture of the abdominal wall is common where there has been a significant loss of weight. Inflammatory disease of peritoneum (such as disseminated tuberculosis) or diffuse neoplastic disease can produce either a feeling of granulation in the abdominal wall or – more distinctively – firm subcutaneous lumps. Metastases to the abdominal wall, most frequently from malignant melanomas, produce lumpy, hard nodules. Tumors here that are associated with neurofibromatosis are characteristically soft.

Tension

Every clinician is familiar with "voluntary guarding" – the sudden tightening of the abdominal wall when touched. It may be voluntary, but guarding could also be reflexic. This reaction can generally be overcome by beginning the abdominal palpation with continuous light pressure from the extended palm over mid-abdomen, as described above.

The ability to recognize rigidity of the abdominal wall – global or regional – is one of the most important skills of the clinician. The value of a well-practiced hand on the abdomen is beyond

the capacity of any imaging technology. Peritonitis is suspected if a light jiggle of the abdominal wall causes discomfort. Rigidity, other than reflex guarding, is the cardinal finding in peritonitis; it may be global or affect a limited region of the abdomen. The impression is strengthened if there is also tenderness associated with percussion and guarding. There is no need to elicit rebound tenderness, adding to discomfort without adding information gained by a more gentle touch. This subject is expanded below in the section on "Tenderness."

The absence of rigidity does not exclude peritonitis. Tension of the abdominal wall in response to an infectious or neoplastic process or to an acute ischemic event may not occur in the debilitated, the frail elderly, and those taking analgesics or mood-altering drugs. Neuropathies could lessen the contractile response of abdominal wall muscles in response to irritation in the peritoneum.

Sensations

The abdominal wall is sometimes overlooked in the evaluation of abdominal pain [17]. The source may be within the wall or in the sensory nerves that are distributed to it.

Hypersensitivity

Suspect hypersensitivity when the patient describes discomfort that appears to be within the abdominal wall itself. The sensation may be described as burning, raw, scraping grating, or another unusual experience of pain. Unilateral hypersensitivity can quickly be ascertained or excluded by drawing a facial tissue or cotton piece across it and comparing sides.

A sense of burning or itching of recent onset may reflect *Herpes zoster* in the pre-eruption stage. (Zoster, Gk: "girdle," the usual site of *H. zoster* recognized in antiquity.) The hypersensitivity will be unilateral, since the involved cutaneous nerves do not cross midline. This characteristic helps distinguish shingles from other vesical-producing eruptions such as plant dermatitis (e.g., poison ivy or poisonous parsnips). An exception to unilaterality is the patient with an immune deficiency condition in whom the distribution can be over multiple dermatomes.

Remember that shingles, in the early stages before the eruption stage, is often associated with malaise and sometimes slight fever. Certainly, the unsuspecting clinician can be surprised when a patient with a tentative diagnosis of cholecystitis, appendicitis, or pancreatitis develops within a day or two a vesicular rash in the area of discomfort.

Pseudohernia

Diabetes, particularly, may produce neuropathic symptoms of pain and hypersensitivity of the abdominal wall. In addition to sensory neuropathy, there may be a motor component in which the abdominal wall weakens. This resultant bulge has led to the impression of an intra-abdominal origin, called a "pseudohernia" [18].

Palpation: Abdominal Organs

Many clinicians prefer to palpate the abdomen by moving in a clockwise sequence. It is a structured method and one widely taught in medical school. In practice, when examining a patient with abdominal pain, some tend to palpate that area last. Other clinicians, perhaps to save time, begin at the site where it hurts. The approach adhered to here is to palpate the abdomen routinely in nine areas in a consistent pattern, always moving in the same direction (as already emphasized in other organ system descriptions) and proceeding from upper to lower abdomen. Of course, the sequence shown here is performed in mirror image by those who prefer the "left-to-right" approach in other regions (Fig. 20.3).

This sequence fosters comprehensiveness and accuracy in communication and recording. It certainly boosts remembering details. One can go back to areas of tenderness with some accuracy for further evaluation when indicated. Palpation of the liver and spleen, requiring an inspiratory maneuver, is expediently deferred until the global nine-step palpation has been completed.

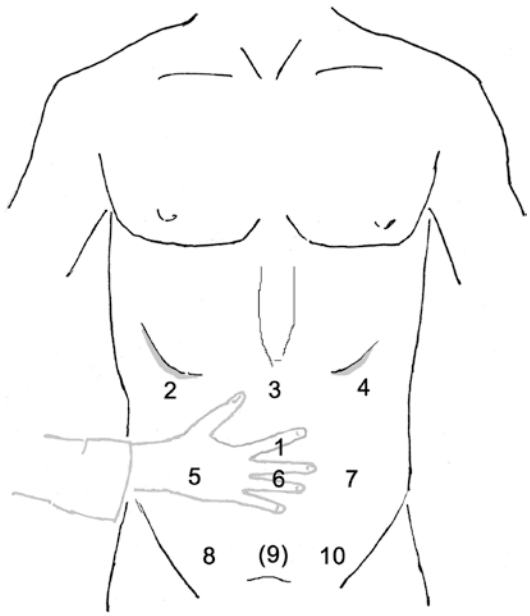

Fig. 20.3 Abdominal examination sequence

Tenderness

Tenderness usually reflects an inflammatory condition in the organ just beneath the point of palpation. This expectation can be very misleading at times, however, as every wary clinician can respect. Whatever the cause of tenderness and severity of discomfort in the abdomen, however minimal, it must be taken seriously. This caution is redoubled in the presence of fever or gastrointestinal symptoms such as vomiting and diarrhea. Only until there has been gathered convincing evidence of an innocuous etiology can further evaluation be deferred.

When the abdomen is so sensitive that a patient objects to even gentle palpation, light percussion is likely to be much more easily tolerated. This technique proves very useful in localizing areas of intra-abdominal pathology while causing minimal discomfort. The area of tenderness can be gently circumscribed.

Eliciting rebound tenderness to evaluated abdominal pain is controversial. Some experts feel that it is an unnecessary exaggeration of pain, not justifying the information gained. Yet, others hold that it can be helpful in identifying peritoneal irritation and, gently performed, need not be unduly distressful. Omitting rebound tenderness could certainly be justified when abdominal pain is intense or when involuntary rigidity has already been detected. Clearly, it is superfluous if the diagnosis has already been established. It must also be remembered that the absence of pain on rebound testing does not exclude the possibility of peritonitis.

Signs of peritonitis may be localized, as in an incarcerated bowel, diverticulitis or cholecystitis, and appendicitis. Diffuse peritonitis brings up a myriad of possibilities, most important of which is the perforated viscera. An important reminder is to consider that the very elderly often have lessened peritoneal reactivity and, for example, may present with intestinal perforation and have no physical signs pointing to peritonitis.

Upper Abdomen

Begin with the upper right, central, and left areas in that order. Tenderness from liver or gall bladder inflammation is usually felt on palpation on the right. Gastritis, pancreatitis, and distal esophagitis most likely cause tenderness in the center (epigastrium). Tenderness on the left upper quadrant should bring to mind hypersplenism, an acute enlargement, or an infarction of the spleen or distention in the splenic flexure of the large bowel.

Middle Abdomen

Palpate across the middle abdomen in the same sequence, starting at the right flank. A normal kidney is seldom palpated. If one is palpable, think of a large renal cyst, a polycystic kidney, or tumor. If both are palpable, the diagnosis is most likely polycystic kidneys; here the presence of hypertension is a given. Hydronephrosis from lower urinary obstruction must be extreme before kidneys are palpable in the adult.

The umbilicus is centered in the middle section. The bifurcation of the abdominal aorta lies beneath, not more than a centimeter north or south of the navel [19]. The aortic pulsation can

be felt here in the normal, relatively thin person. The umbilicus is displaced more caudally if there is a thick pad of subcutaneous fat [20].

Checking for an abnormal abdominal aorta is especially important on the routine examination of persons 50 years or older. An aortic pulsation felt below the umbilicus is suspicious for an aneurysm. Of course, the more obese the abdomen, the less sensitive is this evaluation. Even a large abdominal aneurysm can be undetectable by palpation in the very obese. Tenderness of a palpable pulsation below the umbilicus together with symptoms and signs of acute hypotension is suspect for dissection of an aorta aneurysm.

Lower Abdomen

Identifying the origin of pain in the lower abdomen is especially challenging when palpation elicits extensive regional tenderness. A firm bulge in an inguinal area, particularly one that is tender and/or in a patient with acute abdominal symptoms, may be an incarcerated hernia. Such, when small, can easily be mistaken for a lymph node (as has been committed by this author). Appendicitis is considered separately later in this section.

Tenderness in the lower abdomen is generally from inflammation or swelling in the colon or gynecological organs. When tenderness is central, distention of the urinary bladder or uterus must be assumed as most likely and further determined by percussion.

Percussion

There is little to gain by taking the time to percuss the abdomen of an alert patient without abdominal symptoms and without suspicion of intra-abdominal disease. Percussion, however, can be extremely revealing in special cases when performed skillfully. In a patient with severe abdominal pain, its usefulness as an alternative to palpation has been described above. For an explanation of percussion techniques, see Chap. 17.

Hyperresonance

Tympanic sounds elicited by percussion of the abdomen indicate a dilated bowel, most likely either from intestinal obstruction or paralytic ileus. The former will more likely emit hyperactive and high-pitched bowel sounds. With absent peristalsis, the abdomen remains silent. An alternate explanation of hyperresonance is free gas from a perforated viscus where peritonitis is an expected complication. More rarely, bloating of the abdomen with hyperresonance is merely the result of habitual air-swallowing (aerophagia).

Hyporesonance

Dullness in response to percussion of the abdomen may be due to an enlarged liver, spleen, or uterus or to a large mass. Ascites of detectable magnitude causes dullness in both flanks in the supine patient. In tense ascites, the dullness to percussion extends up to the central abdomen.

"Shifting dullness" differentiates ascites from massive obesity and from an abdominal mass or a massively enlarged organ. The procedure requires establishing a line of demarcation between normal resonance and dullness on one side of the supine patient, then finding a new line of demarcation after the patient has turned onto his or her side. It is usually more convenient for percussion if the patient turns away from the examiner. Some abdomens are so massive, however, that reaching to percuss is easier if the patient turns toward the examiner.

Liver

Estimating the size of the liver on physical examination is difficult and notoriously inaccurate. The body habitus and positions of the diaphragms make a difference. While dullness to percussion of the wide upper surface is easily determined, finding the normally thin lower edge by percussion is more difficult. Given that determining liver size by physical examination is at best a rough approximation, the main value to palpation is to define its texture [21].

Recommended is beginning to palpate the liver over the mid-abdomen at the right

mid-clavicular line. The hand then works its way upward to the costal margin. The point is that massive hepatomegaly is sometimes missed when the examiner begins palpation at the costal margin.

In most normal people, the liver edge will extend below the costal margin by about 5 cm at the midclavicular line, but the thin edge is palpable in only half [22]. The normal edge will be sharp and be only slightly tender, if at all. On full inspiration, the liver can descend two or three finger-breaths below that.

A firm but not hard liver is typical of cirrhosis where the distal edge generally remains fairly sharp, if somewhat blunted. While the cirrhotic liver is somewhat smaller than the normal liver, its edge is palpable in almost every case because of the thicker, stiffer edge. In passive congestion from venous hypertension of right-sided cardiac insufficiency, the texture of the liver is more plastic and the edge more rounded. A stone-like hardness of the palpable liver reveals a tumor; a nodular edge usually indicates that it is metastatic.

Marked hepatomegaly can occur without a major disruption of liver functions. Included are some cases of alcoholic cirrhosis and schistosomiasis. A prominent feature of these conditions is the diversion of blood flow from the liver, resulting in portal hypertension.

Gall Bladder

Most typically, tenderness in the right upper quadrant from cholecystitis is limited to near the anterior mid-axillary line at the right costal margin. A supportive diagnostic sign is observing the patient grimace with discomfort and abruptly stop a deep inhalation just at the time that the descending gall bladder confronts the examiner's fingertips as they press into the subcostal margin (Murphy's sign). If the test is truly positive, the responses will not occur when the same test is performed on the left side.

Enlargement of the dilated gall bladder detected by palpation in the presence of jaundice indicates obstruction within the bile duct system rather than from hepatocellular disease (Courvoisier's law). Icteric patients with enlarged gall bladders are likely to have neoplastic disease; the remaining have biliary tract obstruction from gallstones or from inflammatory thickening of bile ducts [23].

Pancreas

Acute Pancreatitis

The normal pancreas is not palpable; it is palpable only indirectly in acute pancreatitis by the overlying, rigid abdominal wall. Even this sign, however, may be absent.

Patients with acute pancreatitis are markedly uncomfortable; they walk slowly and bent forward and position themselves by shuffling onto a table laterally and with great caution. They have increased discomfort when lying supine and find more comfort when sitting.

Pancreatic Carcinoma

Carcinoma at the head of the pancreas usually becomes evident by causing obstructive cholestasis with jaundice. This relationship is commonly referred to as Courvoisier's Law, although the original description related to common duct obstruction by stones, not pancreatic cancer. Isolated jaundice, in fact, is often the only indication of pancreatic cancer in the primary care setting [24]. Associated, however, may be a vague sense of discomfort including malaise or deep upper abdominal pain with anorexia, nausea, and loss of weight, partly from intestinal malabsorption. Acute pancreatitis and unstable glucose control are potential complications.

An early complication of pancreatic cancer can be superficial or deep vein thrombosis/phlebitis of cryptic origin. The diagnosis should certainly come to mind when thrombophlebitis is recurrent, and/or it occurs in an unusual place. An example of cryptic cancer of the pancreas is

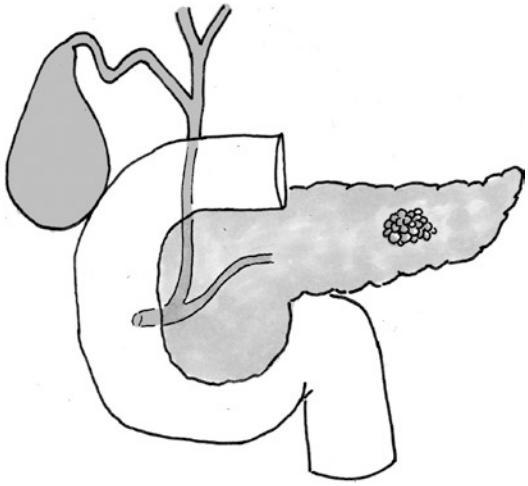

Fig. 20.4 Pancreatic carcinoma

thrombophlebitis of the penis, simulating Peyronie's disease [25] as the first manifestation of pancreatic carcinoma. The presence of gallstones or a history of cholecystectomy is decidedly a risk factor for this cancer [26].

When carcinoma originates in the tail of the pancreas, the condition is almost always far advanced before the tumor is large enough to palpate. The bulk of pancreatic cancer at its tail may compress the splenic artery, producing a murmur over the left upper quadrant. Jaundice, the sentinel symptom of cancer at the head of the pancreas is late to come, if at all (Fig. 20.4).

Spleen

Unlike the normal liver that may be palpated at the costal margin, the palpable spleen is probably enlarged. It is noteworthy that a small percentage of the normal population has a palpable, non-pathologic spleen [27]. At the end of a deep breath, the patient may feel a "catch" as the spleen tip bumps up against the examiner's fingers. The spleen moves readily with deep inspiration, helping to identify spleen from an enlarged kidney, renal cyst, or retroperitoneal tumor.

If the spleen is not palpable but a condition is suspected in which splenomegaly is common, then a modified procedure for increasing the success of palpating the spleen is advised. This modification also pertains to any case of unexplained lymphadenopathy, fever, or anemia. Here, the patient lies on the right side toward the examiner with the knees and hips fully flexed. The non-palpating hand applies forward pressure from behind at the left costal-phrenic area while the patient inhales to the fullest extent. Patients often require strong urging to take the deepest breath possible.

While an enlarged spleen may not be palpable, massively enlarged spleens surely are. As with the liver, extreme enlargement of the spleen may be missed on physical examination if the clinician begins the examination at the costal margin rather than farther or down the abdomen. Certain diseases are prone toward developing the "megaspleen." These include chronic malaria, myelofibrosis, and lymphoma.

The palpable spleen when caused by hemorrhage, infarction, or recent inflammatory disease will be tender and feel spongy. Chronic splenomegaly from portal hypertension or hematologic or malignant disease, by contrast, is usually firm with a fibrotic consistency; it is often not tender.

The spleen when ruptured may or may not be palpable. Signs and symptoms of shock are usually present, although they can be completely absent. Tachycardia may be the only clinically corroborating clue after a fall or other plausible causes of spleen trauma. Certainly a ruptured spleen should be considered in any person regardless of age who has sustained trauma near the left upper quadrant of the abdomen.

Infectious mononucleosis is an indication that splenomegaly should be suspected as an early non-specific sign. If coupled with recent-onset malaise, pharyngitis, cervical adenopathy, and petechiae in the palate, this diagnosis is highly likely.

Kidneys

Normal-sized kidneys in their normal position are rarely palpable. In the adult, they are palpated only when massive. No matter the

cause – cystic, obstruction, or neoplasm – the kidney will not move with respiratory excursions as does the spleen.

Hydronephrosis

Hydronephrosis should be suspected when a mass is palpated in the flank. It is the most common intra-abdominal mass in the newborn. Typically, a palpable hydronephrotic kidney will have a smooth rubbery surface and is not tender. It may be large enough to displace the bowel medially.

Polycystic Kidneys

Kidneys that are palpable bilaterally most likely represent polycystic disease. The polycystic kidney may be giant-sized. Typical is its nodularity. The polycystic kidney is mobile and can be rocked between two hands when one hand is placed over the subcostal margin and the other at the flank. Hypertension that may be highly resistant to treatment is an expected coexisting condition (Fig. 20.5).

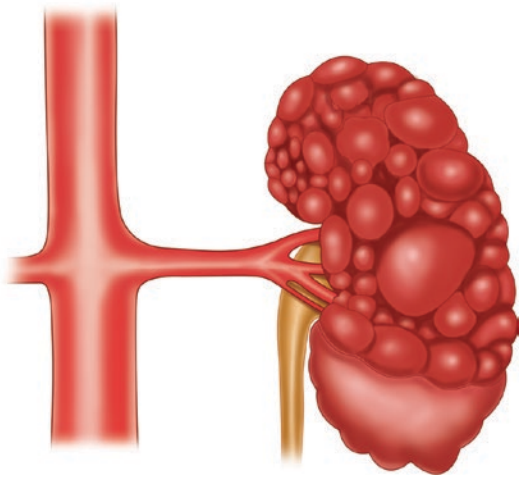

Fig. 20.5 Polycystic kidney (Modified from Phillips [52])

Acute Pyelonephritis

"Punch tenderness" at the costovertebral angle seldom adds any further information to the diagnosis of acute pyelonephritis diagnostically beyond history and urinalysis. It is unnecessary in the periodic physical examination when there is no suggestion of a urinary tract disorder.

When acute pyelonephritis is present, there will likely be sensitivity elicited by simply percussing over the costovertebral area. When appropriate for greater stimulation, lightly punch with the ulnar aspect of the loosely clenched fist. The resulting experience of discomfort may be due to acute pyelonephritis, calculus obstruction, or forniceal rupture. When a renal or perirenal abscess is responsible, even a soft blow can cause intense pain. It is important to remember that CVA percussion or punch tenderness can also be caused by retroperitoneal appendicitis and sometimes by an inflamed diverticulum in the ilium. To test by percussion in the costovertebral angle, of course, requires that the patient sit or to turn onto one side.

Renal Cell Carcinoma

Renal cell carcinoma (formerly called "hypernephroma"), if palpable, is predictably rough on the surface and will feel stony hard. A small minority of cases (about one in ten) will present with the diagnostic triad of flank pain, flank mass, and hematuria. Vascular congestion may be an associated finding, sometimes manifest by a recent development of a scrotal varicocele. Thus, a varicocele on new onset is a clue to obstruction of the testicular vein by the tumor.

Many cases of renal cell cancer are first announced by its propensity to metastasize to "improbably" sites. For example, an intractable nosebleed led to a nasal tissue biopsy that proved to originate from a renal cell carcinoma [28]. Among other sites of metastases reported are the lung, pancreas, bone, stomach, adrenal gland, vagina, cervix, and salivary glands. It is important to note that metastases from a renal cell carcinoma can appear even long after the primary tumor has been removed [29].

Umbilicus

Diastasis Rectus

Stretching of the abdominal rectus muscles, most common during and after pregnancy, will result in a ridge along the vertical midline of the abdomen. The diagnosis depends on a gap between the long muscles of at least two fingerbreadths. It remains soft when the rectus muscles alongside are tensed. Implications of the condition are limited to the esthetic.

Umbilical Hernia

Small bulges in the umbilicus are simply "outies" with no implications of abdominal wall hernia. Umbilical hernias occur where the connective tissue of the umbilical ring is defective, either naturally or after a surgical procedure. The ventral hernias are defined by lack of muscular resistance on pressing over the umbilicus and by a bulging at the site during straining of the abdominal recti muscles in the effort to raise the trunk from the supine position. Ventral hernias are common in the first 3 years of life; in most cases, they subside spontaneously [30].

Post-surgical Hernia

Incisional hernias are defects in the fascial wall of the abdomen following surgery. They can occur after laparoscopic intervention [31]. Hernias may occur some distance from the healed incision. Midline incisions, including laparoscopic ones less than 2 cm, may be associated with a large hernia slightly more cephalad to the incision.

Nodule

Bumps along the rim of the umbilicus can be the earliest sign of an intra-abdominal malignancy. It was this association that earned the name "Sister (Mary) Joseph's nodule." Sister Joseph was a surgical assistant in the early twentieth century who called attention to the frequent incidence of umbilical nodules in cases of intra-abdominal malignancies [32]. They usually originate in the ovary in a woman and the stomach in a man [33]. Umbilical nodules can be the first sign of pancreatic carcinoma [34].

Omphalith

If allowed to accumulate, lint in the umbilicus can eventually develop into a firm mass. Known as the "Pseudo Sister Mary Joseph's nodule" [35], it may contain keratotic material as well as hair, cotton, and other debris. The PSMJN poses no significance other than cosmetic, although cellulitis from a belly button bezoar has been reported [36].

Intestine

Despite its sheer size and complexity, the intestinal tract serves in a remarkably unobtrusive role from day to day. Yet, it is susceptible to a host of adversities while even a small and limited incursion on its normal function can cause profound disruption of the entire system.

Obstruction

Signs of the obstructed bowel are typically high-pitched tinkles interspersed with long silent periods. Tenderness to palpation and percussion are expected findings. While visible peristalsis is seldom present, it is highly specific for obstruction. Among the most common causes of the obstructed bowel are herniation into the inguinal ligament, the umbilicus, or the weakened area at an abdominal wall scar. If the hernia cannot be reduced by gentle external pressure, it is considered incarcerated, with high risk of necrosis, rupture, and peritonitis. Trying to reduce a small hernia that is resistant to external pressure is fraught with danger.

Incarceration of the bowel that results in ischemia is referred to as "strangulated."

Diverticuli

Diverticuli in the colon are very common. Most do not cause symptoms and are discovered only incidentally during colonoscopy. The clinical presentation of diverticulitis of the colon is highly variable, from minor symptoms to the full picture of the febrile acute abdomen. There are no specific physical signs of diverticulitis and suspicion of the diagnosis is based upon recurring episodes of abdominal pain that subsides spontaneously [37]. Rupture of a diverticulum with resultant peritonitis is a rarity. Chronic abdominal symptoms from recurring diverticulitis, however, are fairly common [38].

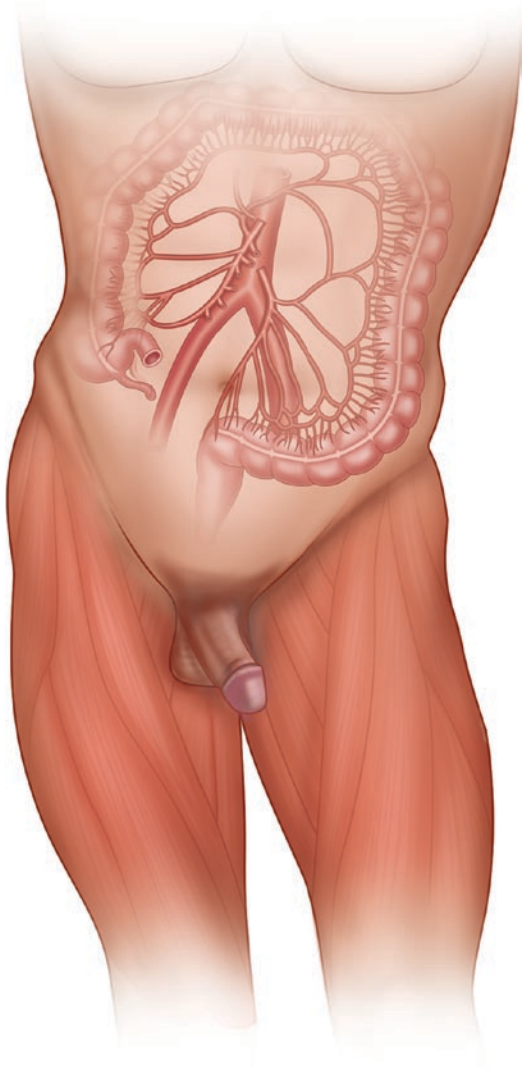

Fig. 20.6 Arteries to intestinal tract (Modified from Phillips [52])

Ischemia

Three large arteries with extensive collateral circulation supply the intestinal tract. When atheroma-induced ischemia occurs, it is assumed that all three arteries are involved. While there are no physical signs of intestinal ischemia, one can expect to find evidence of atherosclerosis elsewhere in the peripheral circulation along with the loss of weight (Fig. 20.6).

The cardinal symptom of intestinal ischemia is epigastric and periumbilical pain after eating that lasts for a few hours. Certain medications – digitalis, beta adrenergic blocks, and ergotamines – by causing even slight vasoconstrictor actions may exacerbate symptoms.

Acute Colonic Pseudo-obstruction

Dilation of the colon without apparent mechanical obstruction is rare. Generally, it occurs in the presence of other systemic disorders such as infection, electrolyte imbalance, and neurologic events (Ogilvie's syndrome) [39].

Appendix

Tenderness in the right lower region deserves respectful consideration of an inflamed appendix; it can occur at any age. Typically, the site is on a line between the anterior spinal process of the ileum and the umbilicus. Further evidence of appendicitis is evoked by gentle percussion and by pain in the same area after a cough or Valsalva maneuver. Rebound tenderness here provoked from the left lower quadrant further supports the diagnosis of appendicitis.

Patients with appendicitis often prefer to lie flexed towards the right side. They tend to resist returning to the supine position for the hands on examination.

Fever and malaise may or may not be present in appendicitis. Pain often begins in the epigastrium and migrates to the right lower quadrant. The typical site of tenderness was precisely delineated by a surgeon at Roosevelt Hospital in New York in the late nineteenth century, and it became known as "McBurney's incision" [40]. Even so, the inflamed tip of an appendix can

make itself known in aberrant locations such as in the retroperitoneal space. A very long and inflamed tail of the appendix may extend into the middle or left abdomen where it causes pain and tenderness. At the same time, the clinician must also consider other causes of pain and tenderness in the right lower quadrant, namely, diverticulitis and an ovarian abscess or hemorrhage.

Two additional signs help define acute appendicitis: the psoas sign and the obturator sign. Indeed, these tests have at least a high specificity for appendicitis as the cause of right lower quadrant tenderness:

1. Psoas sign: right lower quadrant pain is accentuated by hyperextending the right upper leg with the patient lying on the left side (Fig. 20.7).

   The presence of an abnormal psoas test, along with fever and pain migrating into the right lower quadrant, adds substantially to the diagnosis of acute appendicitis [41]. This sign, however, is not infallible [42].

Fig. 20.7 Psoas sign

Fig. 20.8 Obturator sign

2. Obturator sign: with the right knee flexed, the upper leg is rotated laterally while the lower leg is stretched medially. The maneuver exaggerates right lower quadrant pain in appendicitis (Fig. 20.8).

Acute appendicitis tends not be recognized in the elderly as readily as in younger patients. Yet, they are in much greater risk of complications and mortality [43]. Physical signs are less suggestive in this aging population, peritonitis occurs more often, and a delayed diagnosis is more common than in the younger patient.

A bottom line: appendicitis is one of the clinician's most daunting challenges. The patient may present with mild symptoms and minor signs but have an appendix that is ready to burst. On the other side of the spectrum is the very ill patient with intractable vomiting, severe pain, and high fever. Any hint of appendicitis – no matter the severity – deserves expedient evaluation.

Aorta

The abdominal aorta is normally palpable above the umbilicus but only in relatively thin individuals. The elasticity of the aorta is an important factor in maintaining a relatively high pressure throughout the cardiac cycle. With the reduction of elastic fibers of the aortic wall from aging, some loss of compliance (and ease of palpation) occurs.

Atherosclerosis

The aorta is highly susceptible to accumulation of atherosclerotic plaques. Obstruction, however, rarely occurs because of the vessel's width. Where the obstruction is most likely to occur is at this terminal branching, in the common iliac arteries. Major obstructive atherosclerosis here at the bifurcation is likely to produce claudication (usually in the hips) and, in men, impotence (Leriche's syndrome).

The atherosclerotic aorta is susceptible to disruption of plaques. Complications include tearing of the intima, formation of thrombi, and embolization of atheromatous content. In addition, the weakened wall predisposes to development of an aneurysm and/or to dissection.

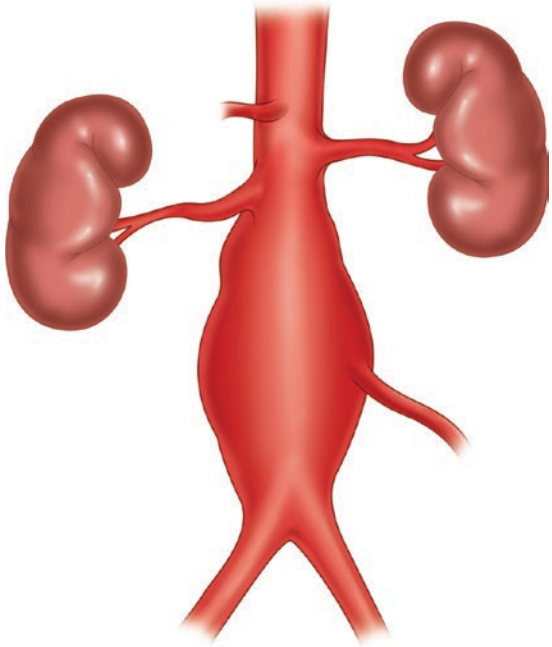

Fig. 20.9 Aortic aneurysm (Modified from Phillips [52])

Aneurysm

The normal width of the aorta in adults is about 2.0 cm at the root with some tapering to about 1.5 cm in the abdomen. An increase to 3.0 cm or more is defined as an aneurysm. Palpating an aneurysm is not possible until it is much larger and even that is difficult when the abdominal wall is thick. As a rule, suspect an aneurysm when the aorta is palpated distant to the umbilicus. Usually what calls attention to an aneurysm is pain when it is expanding rapidly (Fig. 20.9).

The aortic abdominal aneurysm is often referred to as the "triple A disease" or "AAA." It occurs with few exceptions only after the age of 50. Younger persons with conditions that involve weakened connective tissue, however, are susceptible. These include Marfan's syndrome and Ehlers-Danlos syndrome.

Dissection

Blood dissection through an intimal tear and along the media can occur anywhere along the length of the aorta. In the abdomen presentation, dissection may begin just distal to the subclavian artery or distal to the renal arterial branches. The extent and swiftness of the dissection will determine the presenting signs and symptoms. Pain, often severe and of rapid onset, is generally referred to the lower abdomen and leg. Tenderness in the central, lower abdomen may be elicited where an aneurysm can sometimes be felt. Leg weakness can be profound with pedal and popliteal pulses reduced or unequal (Fig. 20.10).

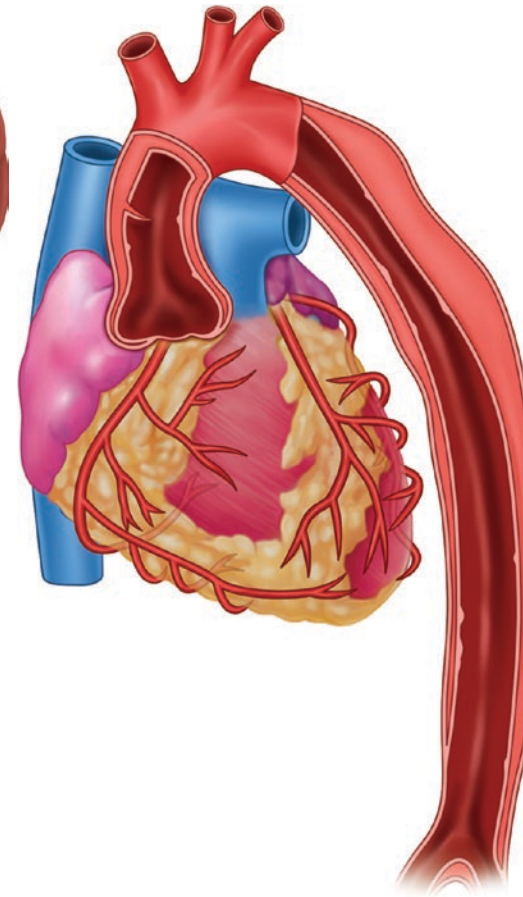

Fig. 20.10 Aortic dissection (Modified from Phillips [52])

While dissection of the aorta usually occurs in the setting of hypertension, the patient can develop symptoms and signs of hypotension, including shock, soon after the onset of pain. All this may happen in a patient who has had no other manifestations referable to cardiovascular system.

Mass

The diagnostic possibilities of an abdominal mass are so extensive that meaningful generalizations are inadequate. The origins range from the stem cell layer of the epidermis to the intestinal mucosa and include every organ system in between. They can be large enough to obscure the organ of origin [44]. Some are common; some are vanishingly rare. All are worrisome. Each one merits a definitive workup.

Clinicians of the pre-imaging era had to rely on surgical exploration as the only means to define an abdominal mass. Could any one of them begin to conceive of the diagnostic precision of today's instruments any more than we can imagine what the practice of medicine will be like in a hundred years from now?

Incidentaloma

Indeed, imaging techniques have opened up a new category of disease: the "incidentaloma." Identification of unsuspected lesions while scanning for another condition is now quite common. For example, the majority of tumors of the adrenal gland are discovered in this way. These include subclinical (or at least overlooked) forms of Cushing's syndrome, aldosteronoma, and pheochromocytoma as well as androgen-producing tumors [45]. Definitive diagnosis after imaging identification of adrenal primary carcinoma and metastatic lesions to the adrenal gland generally must await surgical resection and cytological studies [46]. It is not unknown that a pregnant uterus is discovered on ultrasound workup for persistent abdominal symptoms.

Testicular Tumor

An abdominal mass discovered in a young man dictates directing attention to the possibility of a metastatic testicular tumor. An undescended testicle raises the possibility of its eventually developing cancer increasing by a factor of 40 [47]; the tumor may present as a mass in the lower abdomen.

Impacted Colon

"Phantom tumor" is the impression on palpation of a mass that is caused by impacted stool. This finding is almost always restricted to the left lower quadrant, namely, along the descending colon.

Pregnancy

The possibility of pregnancy should never be far from mind on examining virtually any women of appropriate age for abdominal complaints. Symptoms "typical" of early pregnancy are highly variable. Once in a blue moon, the patient does not suspect being pregnant even in the later months, but rather attributes various symptoms and her increased abdominal girth to another cause such as just gaining weight.

Fetal heart sound by Doppler ultrasound might be heard by the 8th week of gestation (i.e., 10 weeks after the last menstrual period). The normal rate is somewhere between 110 and 160 beats a minute. The tip of the enlarging ovarian fundus is palpable above the symphysis pubis beginning about the 10th week of gestation. It will reach near the umbilicus by the 20th week and the sternum by the 36th week.

Ectopic pregnancy further complicates the diagnostic problems of abdominal symptoms. As the extra-uterine fetus grows, blood vessels rupture, creating a source of intermittent abdominal pain at varying sites that may vary from mild to severe. Pain in the middle of the abdomen decreases the likelihood of ectopic pregnancy, whereas tenderness in the lateral or bilateral tenderness increases the possibility [50]. It is fairly common that women with ectopic pregnancies have pain in the left shoulder from irritation of the diaphragm by free peritoneal blood.

An additional concern is that other enlarging intra-abdominal conditions may be assumed to be from a pregnant uterus. Such conditions include uterine fibroids, adenomyosis, and endometrial cancer. In a nutshell, the clinician must be keenly aware of the confounding symptoms and signs of pregnancy.

Listing of Abdominal Masses

In an attempt to summarize the diverse origins of an abdominal mass, the following are listed:

Tumor (benign or malignant)
Infectious (abscess from bacteria, fungi, parasite)
Cystic (pancreas, kidney, ovary)
Vascular (aberrant vessel, aneurysm, hemangioma)
Distention (gall bladder, urinary bladder)
Inflammation (gall bladder, pancreas, gastrointestinal tract including diverticulitis)
Organ enlargement (liver, spleen)
Intestinal entrapment (volvulus, hernia, intussusception)
Neural (neurofibroma, neuroblastoma in children)
Harmatoma (rhabdomyoma, adenoma, hemangioma, lympangioma) [48]
Residua of surgery [49] (a "gossypiboma," the name referring to *cotton* or perhaps to *gossip*)

Groin

The final attention in the routine abdominal examination is to palpate the inguinal areas for enlarged lymph nodes. It completes the survey testing for adenopathy at the three major sites: cervical, axillary, and inguinal.

Lymphadenopathy

Lymph from the genital and perineum regions drains into lymph nodes just below the inguinal ligament. These are referred to as the "horizontal" lymph nodes. Distal to these are the "vertical" lymph nodes. They serve the legs. A small segment of intestine herniated here (and noted above) can be mistaken for a lymph node.

By far, the most common cause of inguinal lymphadenopathy is fungal infection between the toes that may have superimposed bacterial infection. Otherwise, palpable lymph nodes in the groin are a worrisome clue to malignant disease, most especially lymphoma. The possibility increases substantially if inguinal lymphadenopathy is bilateral.

Hernia

The supine position is not optimal for detecting a "sliding" inguinal hernia; rather, checking with the patient standing is far more rewarding, as noted in Chap. 27. Supine, however, may be the necessary position in a very sick patient.

If incarcerated, a small loop of herniated bowel can become gangrenous and initiates peritonitis. The loop may be mistaken for a lymph node, described above under section "Lower Abdomen." This event is more likely to occur in the frail elderly and may be present with minor symptoms and without signs of peritonitis; in contrast, it may be responsible for a full-blown picture of the acute abdomen with sepsis.

Urinary Bladder

The urinary bladder when full is about the size of a grapefruit. It holds about half a liter of urine. It can be further stretched but only with keen awareness of the need to urinate. Emptying the bladder requires a complex neurological action in which parasympathetic stimuli cause the detrusor muscles to contract; this action occurs simultaneously with relaxation of the internal sphincter, a sympathetic function, and voluntary relaxation of the external sphincter. Obviously, any compromise of this synergistic action promotes urinary incontinence.

Distention

At the pubis symphysis, midline abdominal pressure over a distended urinary bladder tends to increase the sense of needing to void. Enlargement can then be confirmed by palpation and percussion, remembering that giant pseudocysts of the urinary bladder as well as pelvic tumors can give the impression of a distended bladder.

Obstruction to urinary bladder outflow is generally due to prostatic enlargement in men. Digital palpation of the prostate gland per rectum

is the critical part of this evaluation. In women, several forms of muscular dysfunction in the pelvic floor may be responsible for outflow obstruction.

Acute urinary outflow obstruction can be the side effect of a drug superimposed on another disorder, such as an enlarged prostate or defect in the pelvic musculature. Drugs most likely implicated are those with anticholinergic activities and beta adrenergic blockers.

A distended urinary bladder can also be atonal, the result of a neurogenic dysfunction. An autonomic neuropathy involving tracts within the sacral spinal tracts 2–4 may be responsible. The umbilical scratch test will help sort out upper neuronal disease from peripheral neuropathies. Contraction of the abdominal wall toward the stimulus remains intact in disorders of the corticospinal tracts; it is absent in peripheral neuropathies (Fig. 20.11).

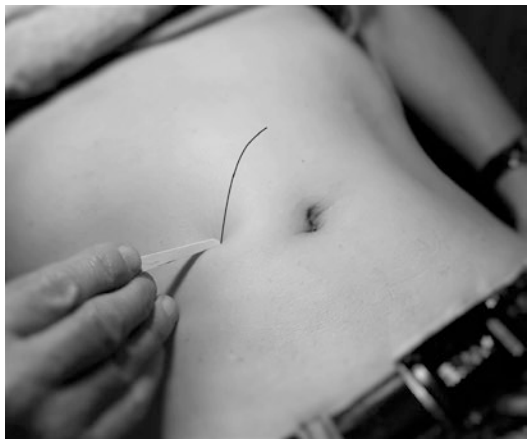

Fig. 20.11 Abdominal scratch test

Situs Inversus

The clinician is reminded of a rare condition that, nevertheless, can be exasperatingly confounding when present. In situs inversus, all the organs of the chest and abdomen are in mirror image of "normal" (Fig. 20.12).

Evidently, medical issues are not more common in situs inversus except as they relate to their oversight in management. Distressing experiences have happened to patients before this perplexing diagnosis was detected. In one such emergency operation on a mid-aged woman for a left upper abdominal gunshot injury, a surgeon at first thought the many foreign bodies encountered were pellets from birdshot [51]. He soon realized that the pellets were actually gallstones. The patient had situs inversus. Such riveting stories are surely common among those with incidental discovery of their anatomical anomaly during a medical emergency.

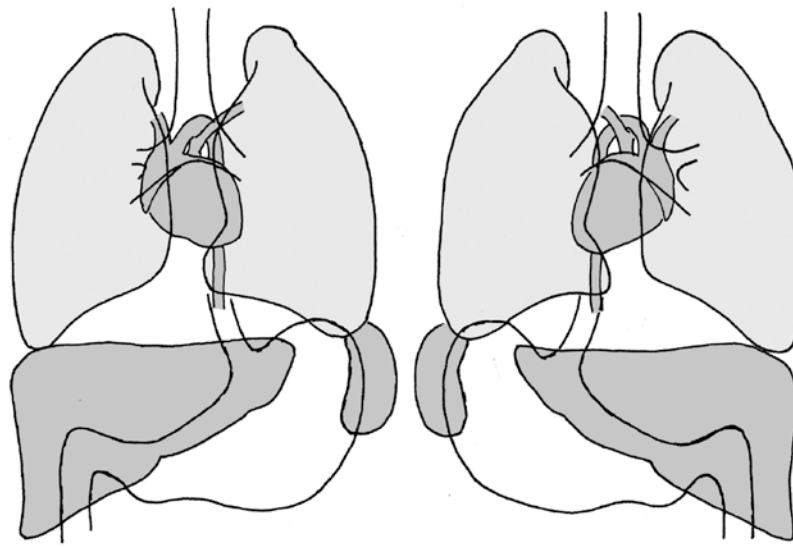

Fig. 20.12 Situs inversus. *Left*: Normal, *Right*: Situs inversus

When present, resonance to percussion will be present over the right upper quadrant (from air within the stomach and over the splenic flexure of the large colon). A clue may have already been appreciated during the earlier part of the physical examination: heart sounds louder in the right chest than on the left.

A distinction is made between situs inversus and dextrocardia. The latter condition occurs when only the heart and proximal vessels are anatomically reversed and are positioned mostly in the right chest. Congenital heart disease is commonly associated, especially those involving transposition of the great vessels.

A right-sided heart can also come from failure of the lung to develop on that side. The heart and great vessels, although displaced to the right, are oriented as normal. Bronchiectasis and underdevelopment of the frontal sinus are commonly associated, increasing the propensity for lung and sinus infections.

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Leg: Supine Position

21

In general, the leg examination is better performed when the patient is sitting. There are two conditions, however, when it is advantageous to evaluate while the patient is supine. One is to check for a difference in leg length, the other to apply a diagnostic leg raising maneuver in the patient with low back pain. It is recommended that leg length be evaluated at each new patient physical examination; testing by straight leg raising need not be routinely performed on patients without symptoms suggestive of sciatica.

Essentials

Leg length
[Straight leg raising]

Leg Length

Leg Length Discrepancy

A difference in the length of the legs (anisomelia) is quite common. Discrepancies of 1.0 cm or more have been demonstrated radiographically in 14.5% of the general population [1]. Such asymmetry for weight-bearing may contribute to wear and tear on joints, causing degenerative changes in the knee or hip over the years. The loss of articular cartilage and development of osteoarthritis is part of the process. Such may explain the frequent occurrence of unilateral arthritis of a knee and hip, beginning with insidiously encroaching symptoms in early middle age and increasing in the years and decades to follow toward a serious disability.

The traditional standard for comparing leg lengths is the full-leg radiograph. In addition, measurements with tape and various methods of determining pelvic leveling (observation, palpation, instrument) are used [2]. Comparing the relative positions of internal malleoli of the supine patient for relative leg length, however, can be performed in seconds with acceptable accuracy. The observation is conveniently included in a routine physical examination, particularly while the patient is supine. There is, incidentally, no significant difference between measurements obtained on standing and supine radiographs [3].

Place the thumbs snugly against the lower aspect of the medial malleoli. The position of the thumbs will be at the same level if the legs are of equal length. Here it is essential that the entire long axis of the body be aligned precisely – from nose to great toes. A slight degree of misalignment can substantially distort the symmetry (Fig. 21.1).

A difference in leg length of 1.0 cm is considered clinically meaningful in the wearing of joints from uneven and excessive loading of knee, hip, and lumbar articulations [4]. On walking, there is a higher stress of ground

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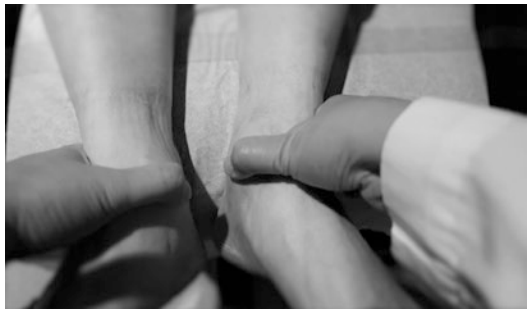

Fig. 21.1 Leg length discrepancy, supine

impact on the shorter leg. Studies reveal that the knee on the shorter leg is at greater risk of developing osteoarthritis [1]. Even slight degrees of differences may also induce low back pain or trochanteric bursitis, but the evidence is less certain.

Detecting a discrepancy in leg length may be especially practical in trekkers and long-distance runners. The problem of the long-term effects of leg length discrepancy is common among persons with scoliosis [5], the elderly among whom osteoarthritis is prevalent [6], and workers who stand for long periods [7]. In total hip arthroplastic procedures, orthopedic surgeons take special care to minimize postoperative leg length differences [8].

Can shoe modification ease discomfort and perhaps prevent further joint degenerative changes when the legs are of different lengths, however slight? The answer awaits further trials. Yet, it is possible that a wedge insert to lift the heel of the shorter leg and fully or partially correct the difference could then be considered as a long-range, inexpensive investment. First, the problem must be identified. Certainly, it is an attractive thought that one of the major reasons for debility in the older population could be prevented by a simple intervention that began decades before.

Sciatica: Spinal Causes

By "sciatica," is meant pain in the buttock area caused by compression on the sciatic nerve.

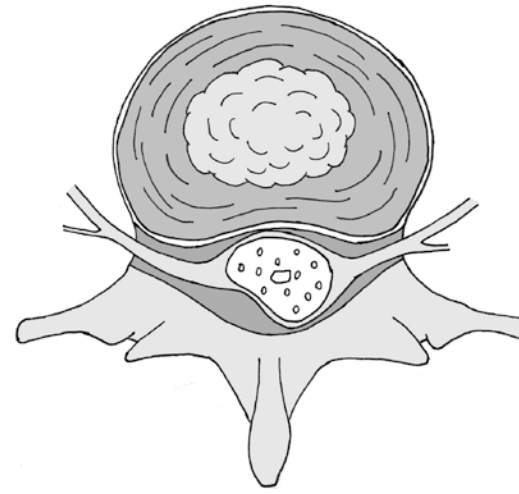

Fig. 21.2 Sciatica

The great majority of cases involve the vertebral spine, and these are caused by protrusion of the pulp through a tear in the cartilage ring of the intervertebral disc [9]. The ring forms the tough annulus fibrosis around the disc. A bulging herniated disc compresses the nerve root at the lumbar of sacral outflow (Fig. 21.2).

Another common cause of sciatica is vertebral osteoarthritis that tends to narrow the outlet of the nerve roots from the spinal cord. More rare are causes that affect the spine: misalignment of a vertebral body (spondylolisthesis), cystic disease of synovia, and tumors of the spinal cord [10].

The most common localizations for spinal causes of sciatica are the lumbar nerve roots between the fourth and fifth lumbar vertebrae and to a lesser degree between the fifth lumbar and first sacral vertebra. Helping to pinpoint the site of an impinged nerve root are the distribution of pain radiation, the pattern of tingling or other paresthesias, the place of weakness, and the abnormal tendon reflexes.

L4 nerve root compression: pain radiates to the anterior-lateral aspect of the thigh. If muscular function is compromised, there will also be a reduction or loss of the tendon reflex at the knee.

L5 nerve root compression: pain radiates to the dorsal-lateral aspect of the thigh. Weakness may

be severe enough to cause foot drop. Changes in deep tendon reflexes are unpredictable.

S1 nerve root compression: pain radiates to the posterior leg. The condition may result in reduction or loss of the ankle reflex. Weakness of the gluteal muscle may affect walking by tilting the pelvis.

If the sciatic nerve is compressed at any of these sites, pain will probably be elicited or aggravated by coughing or by the Valsalva maneuver.

Straight leg raising (Lasèque's sign) is the standard test to perform where there is any suspicion of vertebral/spinal cord disease affecting the lower back. First described in Serbia in 1880 [11], it consists of passively and slowly elevating the extended leg of the supine patient. The raised leg stretches the sciatic nerve root and induces muscle contractions. Pain along with sudden resistance to continuing elevation of the leg indicates further compression of the nerve root. If these events occur as the straight leg moves from 30 degrees to 70 degrees from horizontal, there is a high likelihood that a discogenic disorder is present. This sensitivity is increased when the foot is held in dorsiflexion through the elevation. On the other hand, ruling out sciatic nerve compression in the absence of a symptomatic response by the straight leg raising test is not reliable [12] (Fig. 21.3).

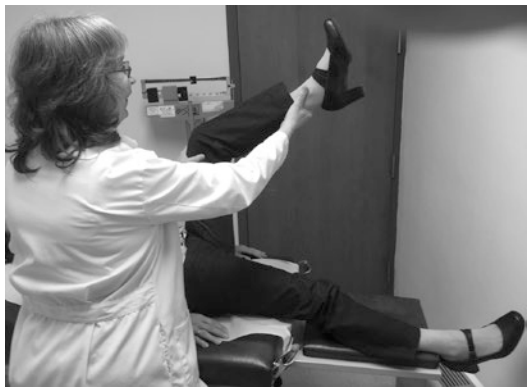

Fig. 21.3 Straight leg raising

Raising the straightened leg greater than 70° can bring out diffuse discomfort from stretching of the hamstring muscles. Arthritis of the hip can also be aggravated at the more extreme lifting angle. Clearly, there is no point in raising the leg above 70° in testing for sciatica.

Sciatica is almost always unilateral. When it is bilateral, the cause is likely herniation of a disc at the midline, not at the usual lateral site. Degenerative disease of the vertebral column can also produce bilateral sciatica by extensive stenosis of neural outlets. Discomfort from either condition may produce a symptom on walking that simulates arterial claudication (called "neurogenic claudication").

Sciatica: Non-spinal Causes

Piriformis Syndrome

The piriformis is a flat muscle that stretches from the ilium and sacral vertebrae to the greater trochanter; it serves to rotate the thigh laterally. The sciatic nerve runs beneath it (or rarely through it). There is a long-held concept that spasm of this muscle could compress the nerve and present as full-blown sciatica. Although the reality of this mechanism is debated [13], the piriformis syndrome may be a frequently recurring clinical entity. Perhaps it is responsible for the sudden back pain sustained from a simple trunk-twisting motion that gradually clears up within several days, no matter what we do regarding rest and activity.

There are some features suggestive of the piriformis syndrome that may be observed during the physical examination [13]. They include unilateral pain in the buttock with tenderness deep in the sciatic notch as well as increased pain on sitting and on lateral rotation of the thigh against resistance [14]. A sharp cough can exacerbate the pain. About half of those assumed to have piriformis-induced sciatica have an abnormal straight leg raising test (Fig. 21.4).

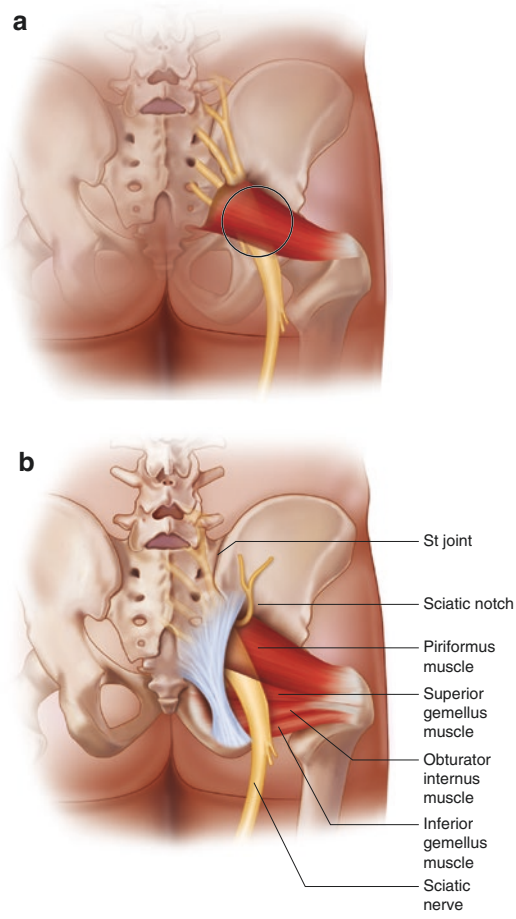

Fig. 21.4 (**a**, **b**) Piriformis syndrome

Credit-Carditis

Habitually sitting on a thick wallet or other bulky pocket items can produce enough pressure on the sciatic nerve to cause a pressure neuropathy. Known as "back pocket sciatica," definitive treatment is rather straightforward.

Other Causes

Low back pain with elements of sciatica presents diffuse etiologies. Causes to consider include trauma from a pelvic or femoral fracture, hip dislocation, muscle hematomas, and injury to the proximal hamstrings. Sciatica from injections in the buttock is, thankfully, a thing of the past. Gynecologic disorder must also be considered: endometrial growth, ovarian cyst, and uterine enlargement in late pregnancy. Transient sciatica can also be experienced in the immediate postpartum period.

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Part IV

Patient Sitting, Again Facing Examiner

Leg: Sitting Position

22

Position #4 *Patient returns to the sitting position at edge of the bed or examining table.*

Mention is made in this chapter of conditions that have a predilection for the legs.

Sitting is a favored position for detailed examination of the legs, including a continuation of the vascular and neuromuscular examination completed in other regions. The bed has already been raised to optimal height at the first part of the examination. Here, the clinician may find it advantageous to use a chair or stool. A flexible lamp that can optimally illuminate the lower legs provides a critical advantage.

This chapter is developed according to the major functional systems: skin, vascular, and neurologic. Conditions that cause edema can involve any of these systems and are covered within each. In addition, guidelines are presented for measuring girth when there is a difference in size. For the expedience of description, the knee and foot are covered in separate chapters.

Essentials

Skin
Size
Arteries
Veins
Lymphatics
Neurological system

Skin

Dermatologic Lesions

Lesions listed here that produce spots on the skin have a predilection for the legs.

Ecchymoses/Petechiae

Evidence of trauma in the lower leg is certainly common. Who wouldn't agree that the main purpose of the shin is to locate furniture while walking in the dark? As they do in other regions, bruises on the lower leg that are unexplained by recent trauma raise concern for coagulopathies and for fragile blood vessels. Rupture of a popliteal cyst can produce a massive ecchymosis, a condition expanded upon in Chap. 23.

Petechiae in the leg suggest a disorder of platelet numbers or function. An alternate explanation is small emboli from the heart or major arteries leading to the foot. These are most likely to appear in the distal foot and toes. Petechiae that are raised indicate that an inflammatory vascular etiology must be considered. The important distinction of macular and raised petechiae is covered in some detail in Chap. 6.

Cellulitis

Cellulitis is inflammation of the subcutaneous and dermal skin recognized by a poorly demarcated erythema that is painful, tender, warm, and

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somewhat raised. A feature of severe cellulitis is cutaneous edema that simulates the granular texture of an orange peel. Proximal lymphadenopathy with linear, erythematous streaks (once known as "blood poisoning") sometimes complicates the picture.

Cellulitis usually occurs in the legs, although it can occur anywhere. Introduction of an infectious agent through the skin is assumed to be responsible, although bacteria and fungi are seldom obtained by aspiration or biopsy. Pathogens most likely to initiate a strong inflammatory reaction in the skin are quickly suppressed by the immunologic defenses. The portal of entry (noticed or not) may be at the site of cellulitis, or it may be at a distal site such as a fungal infection between the toes. It is a frequent complication of self-injected drugs [1].

Plethora of the skin from venous insufficiency can appear as cellulitis, and indeed, it may be a contributing cause of cellulitis. For help in differentiating them: cellulitis will retain its erythematous appearance when the leg is elevated above the heart level. The area rapidly blanches when venous insufficiency is the sole cause of erythema. This test can prove extremely helpful in sorting out inflammatory vs. venostasis causes.

Erysipelas (Greek erythros Red + *pella* Skin)

Erysipelas, known for its bright red rash, affects the deeper skin layers. It is most probably a reaction to a bacterial exotoxin, usually group A streptococci [2]. The onset of a rash along with regional lymphangitis and an intense, febrile illness is typical. The rash has well-defined margins that are raised; these characteristics help separate erysipelas from cellulitis. The lesion may become vesicular or bullous but not purulent.

Of historical note is the alternative name of erysipelas, "St Anthony's fire." It comes from the Middle Ages when the infection was evidently common. Victims, their sudden illness highlighted by a brilliant, red rash, appealed to the saint for cure.

Ulcers

Cutaneous Ulcer

Ulcers of the legs are virtually confined to the distal territory. Many have distinguishing features that are important etiologically. They can be cutaneous, neuropathic, arterial, or venous in origin. Treatment depends upon the correct diagnosis, a principle sometimes overlooked in a one-treatment-fits-all mindset of the "industrial setting" for treatment of leg ulcers.

Persistent ulcers on the leg that are confined to the skin are generally infectious. They are purulent complications of a scratch or superficial laceration. A red, tender streak proximal to the skin break indicates lymphadenitis. Fever and enlarged and tender, inguinal adenopathy are common cofeatures.

Chronic wounds will often harbor multiple organisms, some aerobic, and others anaerobic. The malodorous nature of a persistent ulcer may provide a clue to its bacterial origin. *Proteus*, for example, emits an ammonia-like smell and is likened to that of corn tortillas. *Pseudomonas* is fruity (like Concord grapes) or almond-like and has been described as "sickly sweet." Infections from *E. coli*, of course, reek of sewage. The pungent and foul odor of *Clostridium* warns of is highly destructive nature.

As appropriate for all ulcers, it is imperative to note the size (preferably documented by ruler and photo), depth, borders, surface appearance, and odor. Details for describing ulcers are contained in Chap. 28.

Pressure Ulcer

The pressure ulcer occurs at sites where unremitting pressure is exerted. In the bedridden patient, the sacral area and the heel are most susceptible. Ill-fitted prosthetic limbs pose a similar liability.

The early warning sign of a pressure sore in a light-skinned person is a red blush that does not blanch after compression. The area in those with dark complexion may be blue or purple. Blistering at the site indicates a more advanced stage. Other signs of impending ulceration – temperature, tenderness, and texture – are highly variable.

Discoloration

Gouty Erythema

Acute gouty arthritis typically causes intensive erythema over the involved joint. It is characterized by tenderness as well as extreme pain at the slightest movement of the joint. Without checking the joint for swelling and pain on motion, this condition can be mistaken for cellulitis.

Lipodystrophy

Spotty light-colored depressions in the thighs appear from atrophy of fatty tissues in the subcutaneous layer. They usually appear at sites where insulin has been injected.

Kaposi's Sarcoma

Patches, plaques, and nodules on the skin that have a purplish hue can be signs of an indolent malignancy referred to a Kaposi's sarcoma. The lesions usually affect the lower legs and feet, although oral, gastrointestinal, and pulmonary complications may occur. Immunodeficiency is the underlying condition in which there is a greater susceptibility to herpesvirus.

Kaposi's sarcoma was once an uncommon disease affecting mainly elderly men. The incidence escalated with the emergence of HIV/AIDS, a trend that has been greatly quelled in the era of HAART [3].

Necrobiosis Lipoidica

Raised red-brown plaques with sharp borders found mostly over the shin are typical of necrobiosis lipoidica. A particularly distinguishing feature is a yellow deposit at the center of the plaque. Over the years, the lesion enlarges at glacial speed, but it can result in telangiectasia formation, atrophy of skin, and eventually ulceration. Squamous cell carcinoma has evolved from long-standing necrobiosis lipoidica lesions.

Necrobiosis lipoidica occurs predominantly in persons with diabetes. Even those with glucose intolerance – the "prediabetic" – are susceptible. Comorbidities include those conditions that make up the metabolic syndrome: hypertension, obesity, and dyslipidemia [4].

Pretibial Myxedema

A localized red to purplish patch of swelling over the tibial area resulting from stimulation of fibroblasts and infiltration of interstitial fluid is known as pretibial myxedema. The skin is thickened, and rough in texture, resembling an orange peel [5]. There is a tendency for it to occur in areas of trauma as well as in dependent areas during prolonged standing, thus accounting for the predilection of the shins.

Pretibial myxedema is part of an autoimmune disorder. This name is unfortunate since; in reality, the lesion is a complication of hyperthyroidism, more specifically, Graves' disease. It has been described in euthyroid Hashimoto's thyroiditis [6].

Erythema Nodosa

As the name indicates, these lesions are nodules that are reddened or purplish. They tend to occur on the extensor surfaces of the lower legs in a bilateral and symmetrical distribution. Individual lesions can coalesce to form large areas of hardened skin. Most notably, the lesions are generally painful and exceedingly tender.

The pathology of erythema nodosa involves inflammation of fat cells beneath the skin, a form of panniculitis [7]. The lesions can occur anywhere where there is fat under the skin. Delayed hypersensitivity reaction to a great variety of antigenic stimuli seems to underlie its emergence. While the causal event cannot be identified in about half the cases, febrile illnesses from infection (bacterial, viral, and fungal) appear to be an inciting factor. Implicated, also, are such far-ranging conditions as pregnancy, medications, carcinoid syndrome, pancreatic cancer, and non-Hodgkin's lymphoma.

Patients with chronic diseases such as pulmonary tuberculosis, sarcoidosis, or inflammatory bowel disease are particularly prone to develop erythema nodosa. The possible connection should come to mind in a patient with respiratory symptoms or diarrhea who develops red bumps over the legs. Whatever the cause, these lesions gradually disappear within 6 weeks.

Pyoderma Gangrenosum

While rare, this recurring ulceration can simulate that of venostasis, while its management is totally different. It occurs nearly anywhere, but pyoderma gangrenosum usually involves the leg. The surface exhibits an irregular cribriform pattern (i.e., sievelike). Most notable, it has grayish borders that are raised and undermined. They are notoriously painful. The lesion is also notable for its recurring breakdown and healing.

The ulcer of pyoderma gangrenosum is caused by an inflammatory reaction [8]. The onset can be sudden and rapidly progressive or gradual. It is not a variety of vasculitis and it is not a sequela of defective venous valves. While there may be some similarities to each of these conditions, management of pyoderma gangrenosum requires a wholly different approach.

Necrotizing Fasciitis

What may seem to be cellulitis at first could actually be the beginning of necrotizing fasciitis. This is a rapidly spreading, life-threatening soft tissue infection that involves the subcutaneous tissue and that tends to dissect along fascial planes of muscles. A minor penetrating injury is usually the initiating event. Prompt intervention is imperative to avert loss of limb or life [9].

Necrotizing fasciitis may be recognized as the appearance passes from the early erythematous, hot skin stage to mottled pallor and rubor, accompanied by severe pain and coolness. The patient rapidly develops systemic toxicity with fever and prostration. Evidence of skin necrosis along with bullae that may be hemorrhagic emerges. Damage to sensory nerves causes cutaneous numbness. The pathology may be confined to the foot or it may affect the entire leg. Gangrene, rhabdomyolysis, and disseminated intravascular coagulation are possible late sequelae. With progression, respiratory and renal complications occur.

Although dubbed in headlines the "flesh-eating infection," necrotizing fasciitis is the result of bacterial toxins that destroy, not eat, tissue. In about half the cases, the invasive organism is group A streptococci [10]. Multiple organisms, however, are recovered in some cases. When anaerobic, gas-producing clostridia organisms are present, crepitus in the skin may be felt. In only about half the cases is the site of a bacteria-introducing wound identified.

Size

Leg Length Discrepancy

Comparing the legs for discrepancy in length was described in the previous chapter where the supine position gives the clinician a distinct advantage for aligning the body. A valid comparison of leg length, however, can be made with the legs dangling, thus not breaking up the regional sequence of the examination.

Comparison of leg length is most easily made by bringing the ankles together and then placing the thumbs snugly beneath the medial malleolus. In legs of equal length, the thumbs will line up within 1 cm.

Leg Girth Difference

A studied glance at the legs should reveal any difference in girth. It is prudent to measure circumferences, if only for future comparisons. Easy and reproducible measurements recommended are circumferences made at the maximal calf and the minimal ankle size, as shown. It is important to insure that the tape is exactly horizontal at each measurement. Included here is an example of a simple schematic for recording the measurements (Fig. 22.1):

	R/L (cm)
Max calf	38.6/40.2
Min ankle	22.1/24.2

Alternatively, girth can be determined by using the anterior iliac crest as a reference point and measuring with a tape to the mid-calf and the ankle just above the lateral malleolus. This method is more cumbersome and probably has no practical advantage.

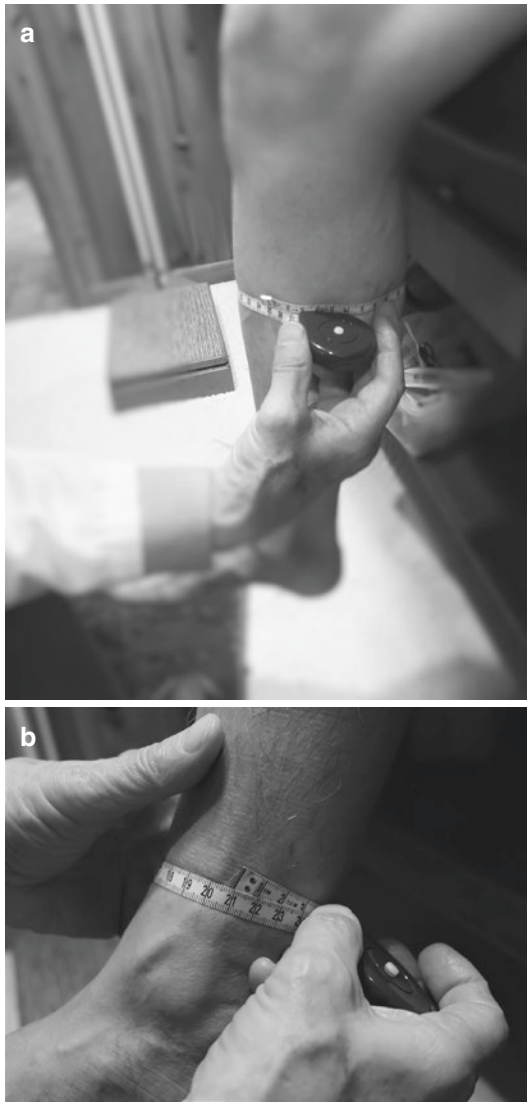

Fig. 22.1 Measurement of calf and ankle girth: (**a**) Maximum calf; (**b**) minimum ankle

Edema is the predominant cause of leg swelling. Its formation is determined by a deviation of one of three physiological principles, singularly or in combination. Clues to help distinguish these causes will usually be evident on physical examination. They are:

1. Reduced osmosis pressure, essentially from a critical lowering of plasma albumin.
2. Increased venous pressure, impaired blood flow to the heart as well as the additional hydrostatic pressure of gravity, as it occurs in chronic venous insufficiency.
3. Capillary fragility. Any injury or immunologic compromise of the capillary wall allows leakage of intravascular fluid. All forms of vasculitis may be accountable.

The presence of "pitting edema" indicates interstitial fluid retention. Usually, the optimal place to test for pitting in the leg is immediately proximal to the internal malleolus by exerting firm pressure of the thumb for several seconds. The degree and extent is recommended according to the following standard:

Pitting edema
1+ = barely visible
2+ = definite by slight
3+ = moderate
4+ = severe (deeply pitting)

In edema of recent or intermittent fluid accumulation, return to normal after pitting occurs within 30 seconds. Chronic edema, however, allows fibrin-inducing reactions in the subcutaneous space; the return of pitting to surface level is long delayed.

It should also be kept in mind that a patient can have massive systemic edema with but minor edema in the lower legs. This disparity of minimal edema in the lower legs in the presence of tense ascites is not uncommon.

Bilateral Leg Edema

Orthostasis is a benign cause of lower leg edema. It is the product of hydrostatic pressure superimposed on hemodynamic pressure that results in movement of intravascular fluid into the interstitial space. In effect, gravity overcomes the internalizing "pull" of fluid that osmotic pressure exerts within the dependent capillaries. The transfer of fluid between intravascular and interstitial spaces is slow. While the weight of blood in the legs is greater when fully upright than

when sitting, gravity-induced edema of the legs from standing is not common since we tend not to stand for long periods. There are exceptions, of course, in occupations where prolonged standing with little walking is required, as demanded, for example, of surgeons, some assembly line workers, and cosmetologists. In contrast, people sit for long periods.

Certainly, the most common cause of edema is prolonged sitting. It is almost universal among inactive people, such as the semi-ambulatory in elderly care facilities. A long airplane flight is guaranteed to result in ankle swelling for all but the flight attendants. While orthostatic edema is bilateral, it can be somewhat asymmetrical when legs are habitually crossed.

The problem of orthostatic edema is exaggerated in hot weather. Acclimatization to heat results in an expanded plasma volume, and this, in turn, increases the outward "push" of intravascular fluid.

The clinician must also consider impeded venous blood flow caused by pericardial disease and right-sided myocardial failure as possible causes of bilateral leg edema. Renal insufficiency can result in edema from an overload of blood volume. Thrombosis of the thoracic or abdominal aorta must be considered. The cause of edema may be within the legs: obstruction of the deep femoral vein near the pelvis or, more likely, venous valve dysfunction.

Hypoproteinemia provides an osmotic basis for bilateral edema; it has a host of etiologies including liver disease, nephrotic syndrome, and nutritional deficits. In patients with severe hypoproteinemia, edema of the upper body, particularly noticeable in the face, often coexists.

Unilateral Leg Edema

Edema in one leg implicates a localized condition. The major causes are acute venous obstruction and inflammation (in a word, "thrombophlebitis") and chronic venous insufficiency (or "venostasis"). Even when venous insufficiency is bilateral, the resulting edema is almost always asymmetrical (reinforcing the utility of obtaining careful leg girth measurements). If leg swelling occurs gradually over several weeks or months, suspect a venous-obstructing neoplasm in the groin or abdomen.

Swelling from lymphedema is much less common than from venous reflux disease. Details on both conditions are provided later in this chapter.

Arteries

Color at the toes of the dangling leg is an important indicator of the distal arterial circulation. Normally, the tips of the toes will be pink with the legs dependent. Discoloration of the legs is a cardinal feature of both acute and chronic arterial disease.

In a relatively young person with pink toe tips and without leg symptoms, there seems little point in palpating for pedal pulses. Feeling for both the dorsalis pedis and the posterior tibial pulses on all patients beyond middle age is, however, recommended in the survey examination. Each artery branches from the superficial femoral artery below the knee. The dorsalis pedal artery may not be palpable in one of a hundred normal individuals; an absent posterior tibial pulse is rarely absent [11] (Fig. 22.2).

The competency of arterial flow in the lower leg can be estimated by blanching a toe with firm pressure and checking the time required for the normal color to return. It is assumed that a capillary refill time on the great toe less than 5 sec is within normal limits; in the diabetic patient this standard was found to be unreliable [12].

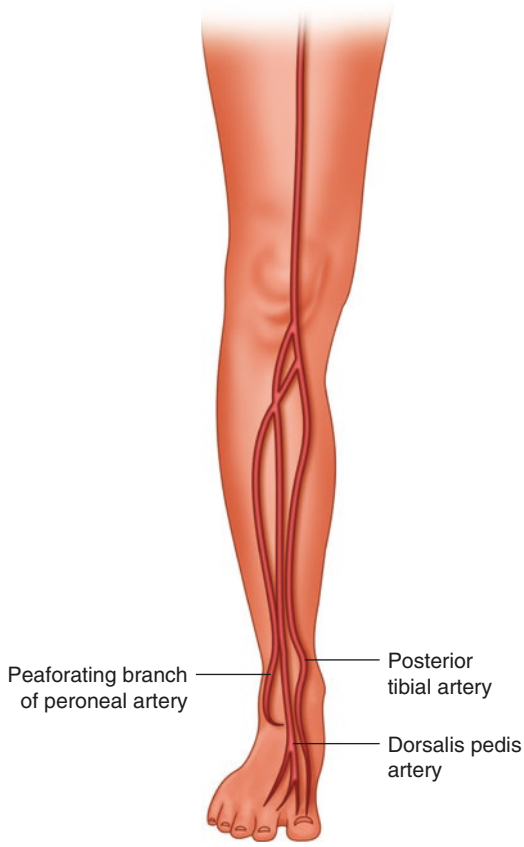

Fig. 22.2 Arteries to leg

Acute Arterial Insufficiency

In the leg, a large thrombus or embolism will produce sudden pallor or mottled discoloration from cyanosis distal to the obstruction. Coolness, pain, numbness, and weakness in the corresponding area are experienced. The transition from palpable warm to cool skin is usually over a narrow span, consistent with the site of arterial occlusion. Pedal pulses will be absent (or at least markedly reduced) on the affected side [13].

The most common site of origin of a thrombus is from a large atheroma in the iliac or femoral arteries. An embolism is usually of cardiac origin and most often associated with atrial fibrillation. An intra-atrial myxoma is a rare cause; the vascular, gelatinous tumor easily fragments, often causing catastrophic results in the peripheral circulation.

In principal, the smaller the emboli, the more distally they will travel. It must also be appreciated that a large thrombus or embolism can break apart, shedding smaller particles into even smaller vessels.

There are some forms of acute peripheral ischemic disease where the pedal pulses are palpable. These include consumptive coagulopathy [14], cholesterol embolism [15], and calcific uremic arteriopathy (calciphylaxis) [16]. The syndrome may present as diffuse purpura or mottling of skin, perhaps taking on a netlike appearance. In these cases, the ischemia can progress on to gangrene with peripheral pulses still palpable.

Chronic Arterial Insufficiency

Seldom is significant arterial obstruction to the legs caused by atheromatous disease within the aorta. Obstruction usually occurs with narrowing at the iliac arteries, the terminal branches of the aorta or further distally (Fig. 22.3).

In chronic arterial insufficiency, the characteristic color at the toe tip is rubor. Rubor, a consequence of reactive hyperemia, is a reliable sign. The color is easiest described as a purplish hue somewhat between erythema and cyanosis. It tends to disappear when the patient is supine. Blanching of the toes and sole on elevation of the leg is more pronounced with significant arterial obstructive disease. This commonly practiced raised leg maneuver is, however, difficult to interpret as a quantitative test for arterial competence. Doppler ultrasonometry provides sensitive changes in arterial waveforms from compromised arterial blood flow, but the changes are difficult to express in recordings. Used to derive

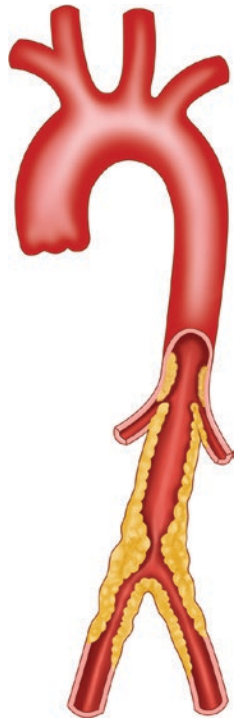

Fig. 22.3 Atherosclerosis of aorta (Modified from Phillips)

the "ankle/brachial index," the same technology provides a more convenient measurement. The index compares the systolic pressure of the brachial artery and that of a pedal artery. Reduced arterial flow in the leg is demonstrated if the pressure at the ankle is less than that in the arm. An ankle pressure of 50 mm/Hg indicates serious ischemic disease [13]. The procedure is described in the Chap. 28.

In addition to rubor, arterial pulsations in the ankle and foot are weakened or non-palpable in chronic arterial insufficiency. This finding obliges the clinician to feel for more proximal pulses: the popliteal and the femoral arteries.

Corroborative signs of chronic leg ischemia include loss of hair over the dorsum of the foot and distal lower leg. The skin here becomes thin and takes on a shiny appearance. The presence of a bruit along the femoral or the popliteal artery may be helpful diagnostically as a sign of arterial obstruction, but exceptions are common.

Characteristic of arterial insufficiency in the muscle, generally, is that it is painless until the muscle is becomes active. At some point of exertion – depending on the severity of arterial flow restriction – discomfort occurs, the symptom referred to as "claudication." (The term "claudication" is named after the Roman emperor Claudius who limped.) Most often, arterial claudication is described as aching or cramping. Some, however, experience heaviness or numbness of the legs during walking. Within several minutes, the pain or other discomfort typically goes away. When pain on effort is more prolonged, it is more likely to be from a muscle or articular strain. This generalization holds true, incidentally, for arterial insufficiency elsewhere such as in the arm and jaw and in obstruction of the subclavian artery or giant cell arteritis of the masseter artery. That is, pain occurs on exertion of the ischemic muscle and subsides within a few minutes on resting.

In claudication, the location of the symptom in the leg reveals the site of arterial obstruction with a reasonably high degree of certainty:

Symptom	Obstruction
Buttocks to thigh	Aorta and iliac artery
Pain on sitting	Iliac artery
Calf	Femoral artery (large majority of cases)
Foot	Tibial or peroneal arteries (usually a combination)

The cause of chronic arterial insufficiency is, of course, predominantly from atherosclerosis. Large, obstructing plaques are most common in the iliac arteries and the femoral branches. Thromboangiitis obliterans – a disease almost exclusively that of smokers – affects the middle-sized and smaller arteries. It is more likely to occur at a younger age than is obstruction from atherosclerosis. Another cause of claudication is fibromuscular dysplasia. Although fibromuscular dysplastic arteries are found more often in the carotid and renal systems, it does occur in the legs and can produce typical symptoms of claudication [17].

Ischemic Ulcer

An arterial obstruction anywhere along the way can result in the ischemic ulcer. A common denominator is pain at the ulcer site that is relieved somewhat by keeping the leg down. An ulcer may occur anywhere on the distal lower leg, the dorsum of the foot, and, quite typically, on the tip of the toe. Small and rounded, the typical ischemic ulcer has fairly well-circumscribed margins. Lacking robust arterioles, the base of the ulcer has little granulation, differing in this way from ulcers with an inflammatory or venous basis. It may be covered with a yellow-gray discharge or a black eschar. The depth can range from shallow to deep enough to expose tendon and bone.

Vasculitis

An arterial ulcer can have an inflammatory origin, associated with any form of vasculitis. The margins of these ulcers tend to be more irregular than ulcers from ischemia. Petechiae or purpura that are palpable and urticaria around the ulcer are supporting diagnostic features.

Veins

Pressure in the veins of the ankle during recumbency is 0–5 mm/Hg. With the patient sitting upright with the legs down, hydrostatic pressure is added to the hemodynamic, increasing the pressure to about 60 mm/Hg. The vertical column of venous blood is from the heart to the feet (minus the length of the thighs). Pressure on standing increases to about 90 mg/Hg; it is higher in tall people. These increased pressures of sitting and standing tip the balance of normal osmotic "pull" of interstitial fluid into the circulation and favor transudation of intravascular fluids into the interstitial space (Fig. 22.4).

It is important to realize that these pressures are intermittent. Each step in walking contracts muscles in the calf, thereby compressing the deep venous system. Blood is forced upward, and local pressure falls precipitously. During the relaxation cycle of the step, the superficial veins empty into the lower pressure perforating veins and then into the deep veins. The functional architecture of the venous system in the calf has led to its being called the second heart. The action of this two-cycle pump occurs with each step so that the venous return matches that of the cardiac output; the volume of blood flow increases as the pace increases (Fig. 22.5).

Both the intermittent expansion of the chest with respiration and the negative pressure of right atrial diastole compliment venous return. These functions are critical during standing, but because of the gravitation pooling of blood, they are not enough to maintain the circulation during prolonged upright. The compressive force of the calf muscle must be enlisted within minutes.

Thrombophlebitis

The preeminent worry with a swollen, painful leg is the possibility of thrombophlebitis. Any injury to a vein tends to induce an inflammatory reaction which in turn will likely cause formation of a thrombus. A thrombus forming in a vein will induce an inflammatory reaction. Thus, the combination term "thrombophlebitis" is aptly named.

In superficial veins thrombophlebitis causes swelling. It is usually accompanied by linear pain, tenderness, and increased warmth along the affected vein. Sometimes a subcutaneous thrombosed vein (or cord) can be palpated.

In deep vein thrombophlebitis (DVT), signs are highly variable and usually obscure. The calf is the most common site of origin. Calf swelling with pain, erythema, tenderness, and increased warmth are typical findings, although any or all may be absent. Deep palpation is probably unwise

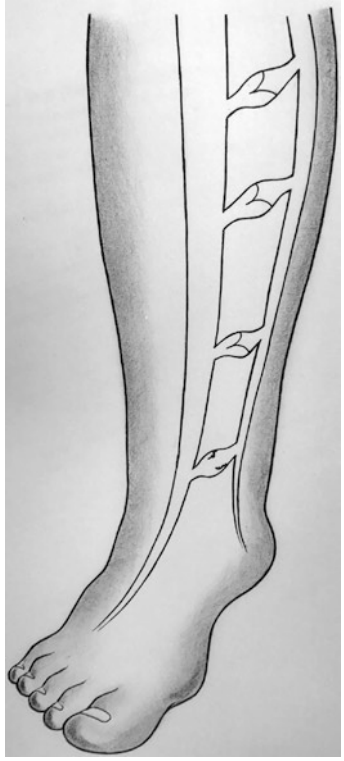

Fig. 22.4 Superficial, deep and perforating veins. Note damaged valve in lowermost perforating vein)

since it could break up a thrombus. Traditional dorsiflexion of the foot (Homan's sign) as a test for thrombophlebitis is too nonspecific to merit diagnostic conclusions; this maneuver can elicit pain in any inflammatory condition of the calf.

Embolization of a thrombus to the lung is most likely to occur when the thrombus in the calf extends into the thigh. There, a large extension of the calf thrombus may flap loosely in the venous stream and cause no physical signs. Its release into the major vein is an immediate threat. Venous clots are principally made up of fibrin and are extremely friable and prone to disintegration and embolization. This texture of a "red clot" contrasts with that of an arterial thrombus, the "white clot," made up mainly of platelets that form a tightly adherent mass (Fig. 22.6).

The physical examination is limited and nonspecific in deep vein thrombophlebitis. When the clinician suspects such a potentially serious disease from history or from nondiagnostic physical signs, he/she is obligated to enlist blood and imaging studies.

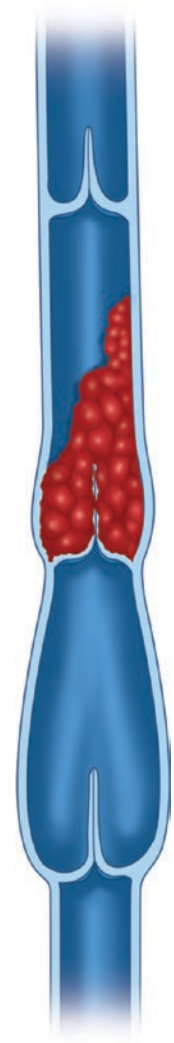

Fig. 22.5 Venous thrombosis (Modified from Phillips)

Certainly, risk factors for deep vein thrombophlebitis should be considered in diagnosis. These are predisposing problems of recent leg injury and immobilization (e.g., a cast) or recent major surgery or a history of cancer. Then, there are thrombogenic conditions: smoking, contraceptive medications, and inherited defects of thrombolysis. The relationship between prolonged sitting and thrombophlebitis has led to the term "economy class" air flight. Also, other major diagnostic possibilities that cause rapid-onset symptoms and signs in the leg should be reasonably excluded;

Fig. 22.6 Venous valves: abnormal (Modified from Phillips)

these include arterial embolism, cellulitis, necrotizing fasciitis, and a rupture of a popliteal cyst.

Rarely, the swelling of the leg from a massive thrombosis or embolism in the proximal deep veins may create enough tissue pressure to obstruct the arterial circulation. Called "phlegmasia cerulea dolens," the condition is characterized by a rapid onset of tense and painful swelling of the leg with a diffuse cyanotic hue and diminished arterial pulses. This is an uncommon form of compartment syndrome, ascribed in part to any of the hypercoagulability states [18], to impairment of venous return in pregnancy [19] and to venous catheterization [20]. Certainly, recognition of phlegmasia cerulea dolens ("venous gangrene") is critical for timely limb- and life-saving intervention.

Fig. 22.7 Venostasis syndrome (Modified from Phillips)

Venous Reflux

Venous valvular insufficiency is a disease of gravity. That is, the normal anti-gravitational system in the veins is counteracted by reverse flow of blood in the valves. The once delicate and flexible venous valves, after an inflammatory injury, become fibrotic and contracted. The damage to the valves resulting in venous reflux is the sequela of thrombophlebitis. Most people with venous reflux disease have a history of pregnancy or injury to a leg, during which the complication of thrombophlebitis may not have been recognized (Fig. 22.7).

When the valves in the veins are disabled, the hydrostatic pressure at the gravitational dependent end of the system is persistent. The once normal one-direction flow to the central circulation is reversed during standing and walking. Tissue

destruction occurs from the excessive pressure although it may take many years to develop.

Varicose Veins

Defective valves in the superficial venous system are responsible for varicose veins. Varicose veins are distended only when the legs are in the dependent position, reinforcing the recommendation that the patient sit during the general leg examination. The veins can be small knuckles, mostly over the lower legs. They can involve the entire greater and/or lesser saphenous veins.

Varicose veins are seldom the cause of debilitating discomfort with upright activity or of substantial edema or tissue injury. The cause may be inherited or acquired from an inflammatory event, namely, phlebitis. Also, valvular reflux in the perforator veins may be responsible by placing an "overload" on a superficial vein, thereby forming a varicosity.

Venostasis Syndrome

The venostasis syndrome involves the reflux of a series of valves in the deep or perforating veins. Over time, the lower leg becomes swollen, mostly prominently just above the ankle. Any difference in girth of the lower leg that is chronic should bring to mind the possibility of venous insufficiency, an extremely common problem in the older population.

Venostasis will cause some degree of plethora in the lower leg. Even a faint blush over the calf in the sitting or standing patient may represent a serious degree of valvular reflux in the deep venous system.

Edema, erythema, and increased warmth in the lower leg in venous insufficiency can be separated diagnostically from other conditions such as cellulitis and thrombophlebitis. With the patient returning to the supine position, raise the leg to above heart level. If plethora is from venous insufficiency, venous blood will run off. Erythema and increased warmth will rapidly diminish or simply disappear but will return soon after the resuming of sitting. In the other conditions, these signs will persist during leg elevation.

To summarize: edema from venous insufficiency is most likely to occur from the reflux of damaged valves in the perforating and deep veins, the result of permanent damage to the venous valves from thrombophlebitis. Physical signs are a plethoric discoloration, edema resistant to pitting, and a brownish hyperpigmentation distributed mainly just above the ankle. The pigment is hemosiderin deposit containing iron from exudation of blood into the subcutaneous space. These sequelae of venous insufficiency make up the syndrome "stasis dermatitis."

As the tissue damage increases, the involved skin becomes thinner with loss of dermis and subcutaneous fat. The skin becomes hardened and scaly, non-pitting, and readily injured. At this point, the condition is described as "lipodermatosclerosis." Weeping of serum occurs as the fragile tissue breaks down. These destructive changes in the overdistended venous system of the lower leg provide an auspicious setting for the skin to break down (Fig. 22.8).

Venous Ulcer

The ulcer of venostasis is quite characteristic and identifiable by ancillary signs. It is usually just above the medial malleolus although there are frequent exceptions. Often the knuckle of a perforating vein can be found near the ulcer. Venous ulcers are generally not painful in contrast to ischemic ulcers.

A whitish, moist crust or exudate usually covers the venous ulcer although a red granular surface may be visible. A granulomatous texture at the base reflects an active arterial regrowth. The depth of the ulcer is variable and in the advanced stages exposes the underlying bone. A foul odor is usually present (Fig. 22.9).

Weeping of the ulceration with the leg dependent is common and certainly the bane of the patient. Weeping can be dramatically stopped, however, by placing the patient supine and lifting the leg above heart level. Demonstrating this position-determined phenomenon to the patient reinforces the importance of leg elevation in management.

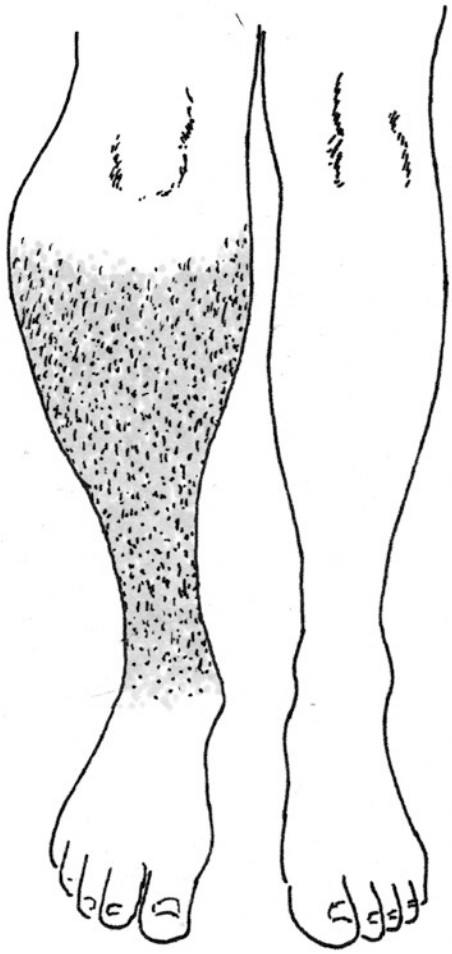

Fig. 22.8 Lipodermatosclerosis

A chronic venous ulcer severely impairs self-esteem and social graces [21]. Despite the unsightliness and massive inconvenience of the venous ulcer, it can persist for years. Continually applying the methods of counteracting gravity prove not easy. Complications will eventually occur and may include cellulitis, osteomyelitis, and – from incessant weeping in large ulcers – hypoalbuminemia. Transition from a long-standing venous ulcer to a squamous cell carcinoma is also possible.

Lymphatics

Lymph is an ultrafiltrate of plasma in the interstitial spaces. It is a protein-rich fluid with a high concentration of leukocytes. The lymphatic drainage vessel system transports interstitial lymph, including leaked protein, to the central venous flow in the left upper thorax. When that system is disabled, lymph accumulates in the interstitial spaces and eventually produces lymphedema.

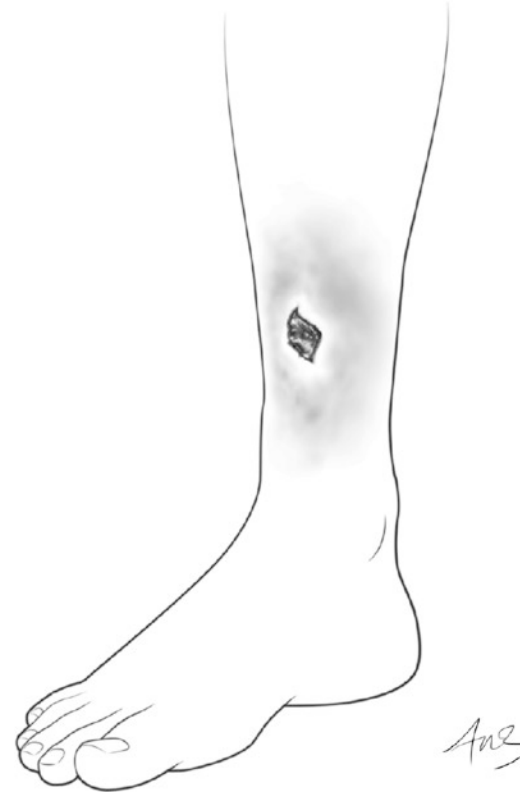

Fig. 22.9 Venous ulcer

Lymphedema

The skin of chronic lymphedema has a rather pale appearance, and the indurated skin resists pitting. There is an absence of pain and lack of increased temperature, helping to differentiate it from thrombophlebitis. It is mostly confused, however, with the swelling of chronic venous insufficiency.

Lymphedema tends to affect the foot and toes, whereas venous insufficiency usually spares them. Hyperpigmentation, if any, is relatively light. Ulcerations of the skin and diffuse weeping are found only in the most advanced stages.

These severe cases, admittedly, are practically impossible to differentiate with confidence. Furthermore, lymphedema and venous insufficiency can coexist.

The cause of lymphedema may be an inherited defect in the lymph system or result from inflammatory destruction or mechanical obstruction. In acquired cases, pitting edema occurs initially. Fibrosis supervenes after months and the swelling develops a more solid texture without pitting. Eventually scarring and hyperkeratosis can occur with massive enlargement of the entire leg. It is known in its extreme as "elephantiasis." In the tropics, the cause is usually a filarial infection.

Neurologic System

The patient in the sitting position with legs dangling provides an advantageous position to evaluate the lower extremity for weakness. The long length of motor and sensory tracts makes the legs most susceptible to any condition that promotes central and peripheral neuropathies.

By this time in the physical examination, the clinician has gathered an impression of general neurological competence. It is highly unlikely that patients with a normal gait, with no indications by signs or symptoms of neurological disease and with no conditions such as diabetes or chronic alcoholism that tend to cause peripheral neuropathies will have abnormal neurological signs in the legs. Certainly at this point, a detailed neurological examination of the legs of patients who fit this description can be omitted in good conscience. Standing tests to come will challenge any subtle neurological issue.

Should further neurological evaluation be indicated, meaningful screening can be rapidly performed and can provide a credible assessment. As a reminder, adhere to strict symmetry, comparing each neurological test with its contralateral side, proceeding step by step. To help with remembering the results, always compare specific points by moving in the same direction.

Weakness

Test for strength by flexion and extension of the great toe against the mild resistance applied by the examiner. This testing is akin to that described for testing upper extremity strength with thumb movement. If weakness is detected on one side, then flexing and dorsiflexing the foot and raising and lowering the lower leg – all against slight resistance – will more fully explore the motor functions. Additional and more demanding tests of lower extremity power with the patient standing are described in Chap. 26.

This section of the evaluation of weakness in the legs is organized according to levels of the neurological control: (1) upper motor and (2) lower motor.

1. *Upper motor neuron weakness*

Upper motor neurons carry signals originating in the cerebral cortex. The stimuli descend into the internal capsule (or pyramidal tract) to the brain stem and end in the spinal cord. There at the contralateral lower lumbar and sacral levels, they synapse with effector nerves that comprise the lower motor neurons.

Sudden interruption anywhere along this corticospinal tract causes weakness with spasticity, hyperreflexia, and an abnormal plantar reflex. These signs are hallmarks of upper motor neuron lesions. Affected muscles tend to maintain their original bulk, although some atrophy from disuse will occur. Causes are usually a cerebrovascular hemorrhage or thrombus. Multiple sclerosis and a neoplasm are possible but far less likely.

Spasticity is evident when the leg resists passive flexion and extension of the knee and hip. Sometimes the muscles that are resistant to passive movement will suddenly give way, a feature known at the "clasp knife" effect.

In lesions of the corticospinal tract, there is typically a residual imbalance of muscle tone in which the extensors of the legs are stronger than the flexors (in contrast to the flexors of the arms being stronger). The patient is unable to flex the leg properly and so must "throw out" the leg laterally to clear the foot with each step.

At the same time, the arm is held stiff and flexed.

The deep tendon reflexes provide additional evidence of corticospinal interruption. For effective testing, the strike force on the reflex hammer must be symmetrical with a direct, not a glancing, blow. Normal reflexes can be brisk in the knee and minimal in the ankle – or vice versa. The point is made: it is the lack of symmetry of these reflexes that is significant diagnostically. For reliability, the patient should be as relaxed as possible. Inconspicuous deep tendon reflexes can be brought out by having the patient pull his or her hands apart vigorously just as the stroke begins.

The reflex "jerk" is a reaction to sudden stretching of the tendon that stimulates the nearby muscle spindle cells. It reflects a completed arc from afferent stimulation to spinal cord to effector motor stimulation. Deep tendon reflexes are superficial reflexes; that is, they circuit the spinal cord and do not enlist any long tracts. Complex upper motor relays, including cerebellar, affect their degree of reactivity by providing an inhibitory action. It is loss of this dampening effect by the upper motor pathways that leads to hyperreflexia.

For illustrative purposes, one form of common cerebrovascular event – the internal capsule hemorrhage – is described in some detail. Hemorrhage into the internal capsule (or pyramidal tract) was once the most common form of stroke. Modern worldwide control of hypertension has markedly reduced its frequency. The arterial supply of this pathway from the motor cortex is from tiny branches. They emerge at right angles from the large middle cerebral artery, an architecture that renders them vulnerable to high pressure. A rupture of such a minute vessel into the densely packed motor pathway of the internal capsule has a devastating effect. The result is hemiplegia but without interference of the sensory or coordination systems. For a sketch of the involved neuroanatomy, see Chap. 11 Weakness: Pyramidal Tract.

Grading of deep tendon reflexes should be consistent. Recommendations are as follows:

Grade	Reaction
0	No reflex elicited, even with reinforcement maneuvers, always abnormal
1+	Hypoactive, borderline normal – abnormal
2+	Low normal
3+	High normal
4+	Hyperactive
5+	Markedly hyperactive with clonus (repetitive contractions)

Patella Reflex

Use the pointed end of the standard reflex hammer to best stimulate the broad patella-tibial tendon. Keep one hand on the distal quadriceps. A slight contractile response may be more easily detected from feeling the quads than is seen in movement of the leg. The major relay is through the spinal cord at lumbar nerve four (L*4)*.

Ankle Reflex

Here, the target is the distal end of the Achilles tendon immediately above its insertion on the calcaneus. Use the wide end of the reflex hammer to insure stimulation across the entire tendon. The reflex is most easily evaluated by enforcing slight dorsiflexion of the foot with the non-striking hand. This maneuver tightens the Achilles tendon beforehand and exaggerates the provoked extension. The reflex arc is mainly through the first sacral nerve (S1).

It is noted that an absent deep tendon reflex can occur immediately after disruption of an upper motor neuron lesion. This effect is transient, owning to the initial shock and before there is time for synaptic recovery in the spinal cord.

Deep tendon reflexes tend to be hyperactive in states of increased metabolism. These conditions include hyperthyroidism and the effect of a stimulating drug. The hyperreflexia is symmetrical.

Plantar Reflex

Commonly referred to as the "Babinski response," the test is performed by stroking the lateral sole starting at the heel and moving anteriorly, then curving medially across the base of the toes. The stimulation falls within the sensory territory of the S1 dermatome. Here, the pointed end of a

twisted apart tongue blade makes an effective instrument; it will not penetrate the skin as might a metal point.

The stimulated plantar arc will normally result in dorsiflexion of the great toe. Upward movement of T1 is a highly reliable indicator of corticospinal tract disease. The up-going great toe, however specific for upper motor neuron disease, is a relatively insensitive indicator; that is, flexion of the toe does not exclude a defect in the corticospinal tract (Fig. 22.10).

An acute vascular disease that is lateral will cause a unilateral abnormal plantar response. Some diffuse dysfunction of the cerebral cortex, however, may result in a bilateral reflex.

The normal response to the plantar stimulus is simultaneous flexion of all toes. In upper motor neuron disease, the toes 2–5 instead spread or fan out. This sign is not as reliable as the up-going toe. It can be present in the absence of neurological disease. The examiner must also be careful that reflexive withdrawal of the calf or thigh is not misinterpreted as a plantar reflex.

2. *Lower motor neuron weakness*

Lower motor neuron disease is basically the process of denervation. Defects anywhere along this system produce characteristic findings of neuropathy. They include atrophy of the muscles served by the nerve along with hypotonia and fasciculations. Another telling feature of lower motor neuron disease is the reduction or absence of deep tendon reflexes.

A defect in lower motor neuron disease may be in sensory input from the stimulus or in the efferent pathway within the spinal reflex loop. Dysfunction can originate in the anterior horn cells of the spinal cord, along the projecting nerve or in the neuromuscular junction. Causes of weakness from lower motor neuron disease include diabetes, alcoholism, polyneuropathies, nerve entrapment, and trauma (Fig. 22.11).

The source of lower motor neuron disease may also be located at the neuromuscular junction. The origin of dysfunctions at this level can often be traced to myasthenia gravis, drug overdoses, thyroiditis, and the polymyositis syn-

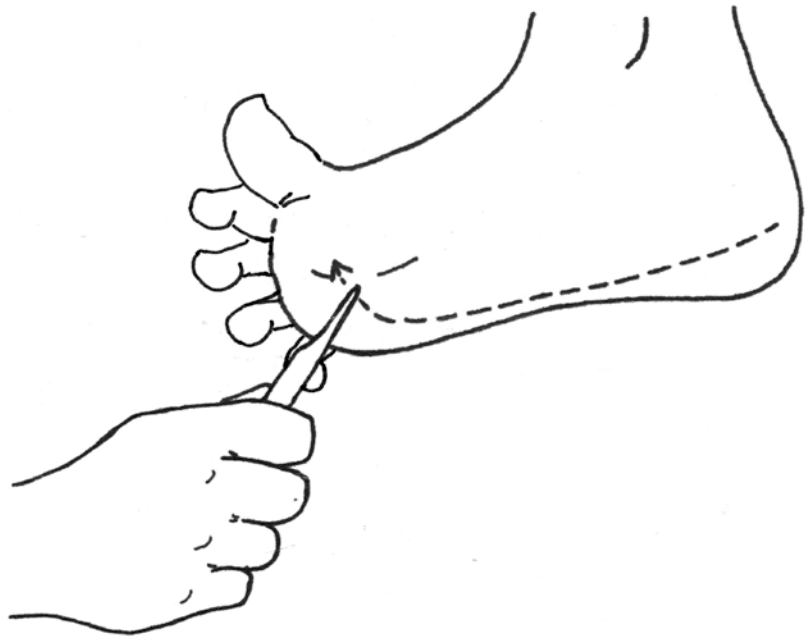

Fig. 22.10 Plantar reflex

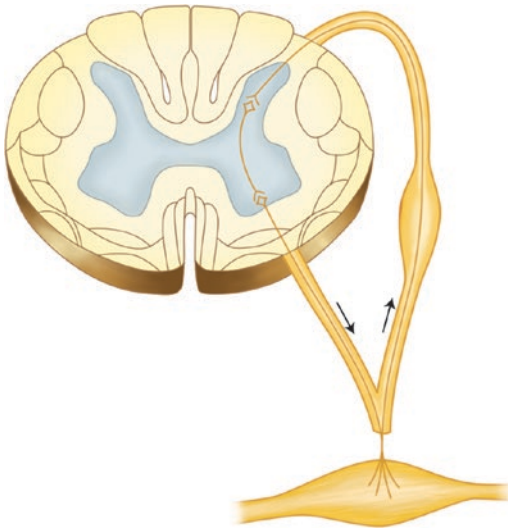

Fig. 22.11 Neural arc of cord

dromes. When lower motor neuron disease is bilateral, disturbances of bladder, bowel, and sexual functions occur.

Slowed deep tendon reflexes are highly suggestive of myxedema. This response involves both the contractile and the recovery components. Perhaps the most obvious sign of delayed deep tendon reflexes is found in the ankle. Look especially for a delayed return to neutral position after the initial reflex ankle flexion.

Amyotrophic Lateral Sclerosis

A neurodegenerative disease with highly specific features, amyotrophic lateral sclerosis affects the motor neurons of both the central and peripheral nervous systems. Weakness and muscle wasting are cardinal features, but the onset and progression of the disease are highly variable, usually involving at first either arms or legs. Sometimes, however, the illness begins with bulbar dysfunctions such as difficulty in speaking, chewing, and swallowing. Eventually, the distribution of muscular involvement is global [22].

Physical examination in amyotrophic lateral sclerosis may reveal any of the following manifestations:

(a) Upper motor neuron: spasticity, muscle cramps, and increased tendon reflexes. There is an extensor plantar response in about half the cases.
(b) Lower motor neuron: muscle wasting, hyporeflexia, and muscle cramps. Fasciculations may be prominent.
(c) Brain stem neurons: dysarthria, dysphagia, drooping of the palate, and depressed gag reflex along with pooling of pharyngeal secretions and fasciculations of the tongue.

"Amyotrophic" refers to muscle atrophy, while "lateral sclerosis" refers to the corticospinal tracts that transverse the lateral columns of the spinal cord. Criteria for the diagnosis require that there are defects in both the central and the peripheral nervous systems. In addition to diffuse dysfunctions of motor activity (upper and lower) along with muscle atrophy is the absence of (or minimal) sensory defects.

Sensory

The sensory system of the legs is evaluated here along three distinct tracts to the cerebral cortex. They are represented by testing perception for sharp, light touch, and proprioception. Vulnerable to metabolic and traumatic injury, these long tracts represent a practical sampling of the sensory system, and the testing can be quickly performed. While the sensory examination is said to be the "weakest" part of a neurological evaluation, it can be conducted with creditable reliance with patients who are able to cooperate.

It is helpful first to demonstrate each of the testing stimuli on the patient's fingertip so that he or she can know what to expect when it stimulates the toe. If the stimulus is not felt at the tip of the great toe, repeat it proximally until it is: malleolus to mid-shin to knee. There is no need to proceed proximally if the sensation is perceived in the toe.

Sharp

The pointed end of the twisted apart tongue blade used to elicit the plantar reflex is handy for testing sharpness. There is no need to use more than light pressure. Recognition of sharpness reveals an intact lateral spinothalamic tract. Fibers of this track carry sensations for both pain and temperature.

Touch

The ventral spinothalamic tract is tested by applying light touch. A facial tissue or cotton ball is a sufficient tool for stroking the toe tip. More exacting tools for quantifying sensations are available. Made of filaments that have measured pressure for bending, they apply a calculated and predictable amount of pressure. Such precision is desirable for a neurologist or in research but is hardly necessary in the routine physical examination. This instrument-guided evaluation is described in more detail in Chap. 28.

Proprioception

For proprioception, the tuning fork serves well as a sensitive test. Stimuli are carried to the cerebellum by the posterior spinal column tracts. A low-frequency fork at 128 cps is recommended. The fork must be pressed firmly against the bone, either the tip of T1 or its metatarsal joint. For screening, the strike force on the fork can be light at first, repeating it with increasing force if at first not perceived. It is helpful to demonstrate the vibratory sensation on the patient's finger to give an idea of what to expect in the toe.

Perception of vibratory sensations in the finger but not in the toe is sufficient to conclude that there is some degree of proprioception deficit. Placing the fork then on the malleolus and then to higher levels along the tibia will help map the extent of the anesthesia. Neurologists may test vibratory sensation by timing the disappearance of vibratory sensation during prolonged contact. This method is probably too exacting for the general clinician's evaluation. Proprioception will be reexamined again later during the standing tests.

These three sensory tracts are depicted in the cross section of the spinal cord at the level of the fourth lumbar disc (Fig. 22.12).

Neuropathic Ulcer

To ascertain that an ulcer of the lower leg has a neuropathic basis, there needs to be a demonstrable neurologic defect. If so, there will be a loss of sensation to sharp along the edges of the crater.

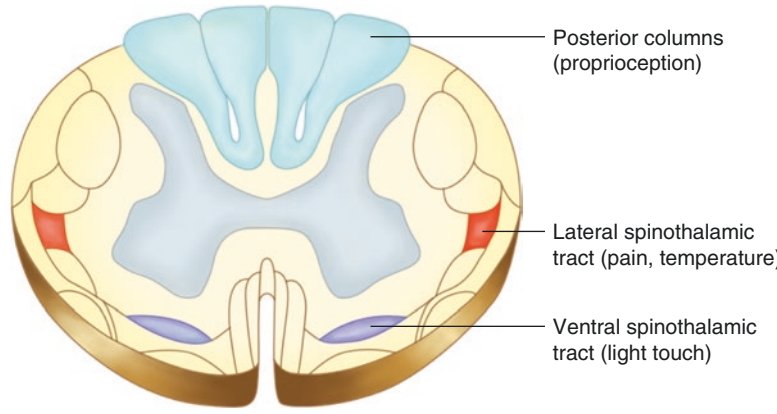

Fig. 22.12 Sensory tracts: composite of spinal cord

When sensation in the long tracks is absent, the patient may be unaware of excessive pressure exerted on a small area of the foot by a misfit shoe or a small, undetected object in the shoe. This commonly held concept is, in fact, the essence of the diabetic foot prevailing concept although the diabetic may have foot problems of diverse etiologies.

Muscle

Primary disorders of muscle involve destruction of the myocyte by demyelization. All forms have a genetic basis and involve muscle wasting in varying patterns. Progression occurs at differing rates with relapses, at least in the first few years. Most forms of muscular dystrophy begin during childhood but can appear at any stage of life.

Myotonic Muscular Dystrophy

In adults, myotonic form of muscular dystrophy is the most common. In this "classical" adult-onset version, the predominant symptom is distal muscle weakness in the limbs [23]. While weakness can appear anytime during life, it usually is first noticed by the age of 20. It progresses slowly over the years with, at first, some relapses. In the leg, dorsiflexion of the ankle is affected early and leads, eventually, to foot drop. Walking becomes a major dysfunction, and falls are frequent [24].

A characteristic of myotonic muscular dystrophy is some delay of contracted muscle to relax, causing a slow release of a grip or return from a foot lift. Impaired speech and swallowing are late complications. Non-muscle manifestations at multiple sites further complicate the illness; these may include intolerance to insulin, cataract, cardiac arrhythmias, hypogonadism, and disrupted cognitive function [25].

Recognizing muscular dystrophy on the physical examination requires identification of the weakness, coupled with atrophy of the weak muscle group and the absence of fasciculations. Since there is no defect within the spinal reflex, both sensations and tendon reflexes are preserved (unless the muscle is too weak to respond). Notwithstanding this generalization, cases of dystrophic muscle disease have been detected in which there were both motor and sensory defects.

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Knee

23

Knees, the largest joints in the body, hold up admirably well over a lifetime of wear and tear: Still, the stresses of ordinary weight-bearing, flexions, twists, and turns take their toll. By the age of 50, the majority of people have at least early evidence and often symptoms of degenerative knee disease. Each step doubles the weight borne on one leg, a fact that correlates with the obese having an increased incidence of degenerative arthritis in the knee. In the musculoskeletal system, only the back can boast of having more complaints against it than can the knee.

The clinician would do well to take a moment routinely to check for anomalies of the knee. Simple measures can sometimes identify a problem that when left unchecked could lead to an insidious progression of pathology over the years. This sitting position with the legs dangling relaxes the muscles, tendons, and ligaments of the knee, facilitating the examination.

Essentials
Size
Shape
Palpation
Movement

The simple hinge that makes up the knee has a remarkable architecture, offering an amazing combination of strength and mobility. Side-to-side stability is provided by the lateral and medial ligaments. The anterior and posterior cruciate ligaments restrict fore and aft movement. Between the weight-bearing femur and tibia, the thick and rubberlike lateral and medial menisci serve as shock absorbers while distributing weight across the tibial plateau. The sliding patella alters the direction of the quadriceps muscle tendons, providing just enough mechanical advantage to amplify the quadriceps' force of extension on the tibia. The powerful combination of the hamstring muscles provides flexion. Lining it all is the synovial membrane that makes up several bursae; the membrane secretes an oily fluid that keeps the knee well lubricated (Fig. 23.1).

Signs that the knee may be susceptible to accelerated wear and tear were mentioned in previous descriptions on examination of the legs. Leg length discrepancy, misalignment, imbalance of supporting muscles, tendons, and obesity were already noted. Once a problem is identified, the patient can enlist appropriate measures that consider lifestyle, physical mechanics, and medicines. Such usually easy-to-apply interventions may stave off the inexorable progression of degenerative disease of the knees, certainly preferable to waiting for "joint death" when knee replacement becomes the inevitable choice [1].

The common denominators of acute inflammation in the knee include pain (especially on movement) and vasodilation (with erythema and warmth). Inflamed synovial membranes become thickened. Fluid accumulates in the synovial bursa and the joint swell. In chronic inflammation,

R.E. Phillips, *The Physical Exam*, https://doi.org/10.1007/978-3-319-63847-8_23

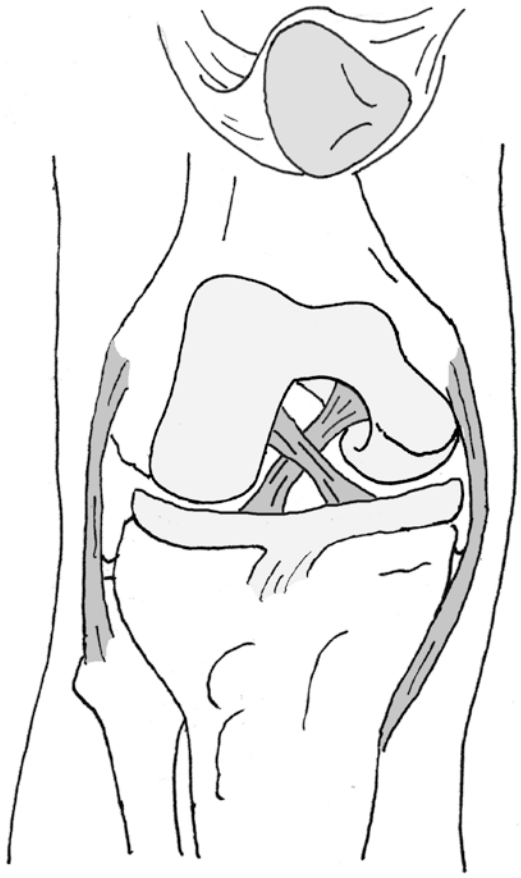

Fig. 23.1 Knee: patella, and quadriceps tendon reflected

the cartilage is destroyed and regional bone is resorbed and remodeled.

Knee injuries are a common condition in the emergency departments. They may be uncomplicated when self-healing is fully anticipated; they can involve, however, more serious damage to regional bone and supporting structures. Criteria have been developed to help decide when an imaging study should be obtained. Six guidelines together have proven highly sensitive and reliable [2]. They are [3]:

- 55 years of age or older
- Tenderness at the head of the fibula
- Isolated tenderness of the patella
- Inability to flex the lower leg to 90 degrees
- Inability to bear weight immediately after the injury
- Inability to take five steps under observation

Size

Swelling

In the normal knee, there is a slight depression flanking each side of the patella. This hollow is filled by swelling, even when mild. The swelling may be a product of inflammation or excessive synovial fluid. With joint effusion is the sense of sponginess when pressing the patella against the femur. It becomes much more prominent when there is joint effusion (Fig. 23.2).

The "patella click test" is used to detect an effusion within the knee. To perform the test on a sitting patient, the knee is extended and the ankle placed on one's own flexed knee that is supported on a stool. (Alternatively, the patient can lay back on the examining table or bed.) It is important to apply strong pressure with one hand spread over the distal quadriceps while compressing the patella sharply with the other. Detecting a click

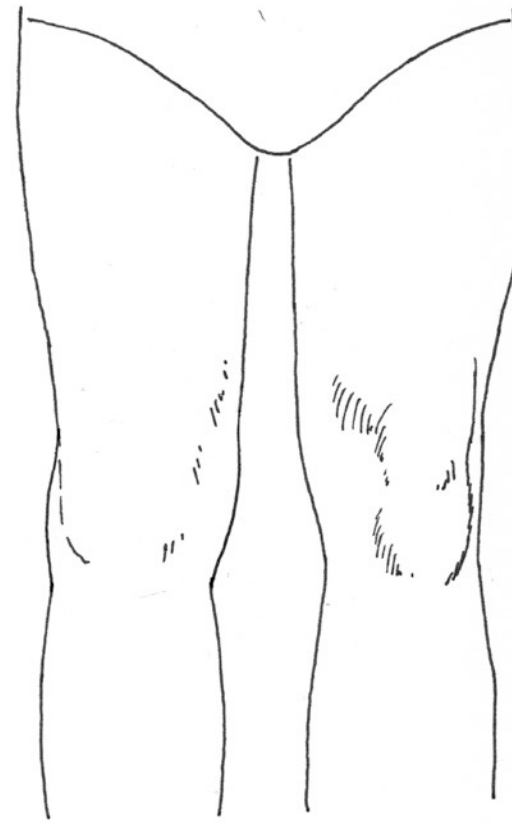

Fig. 23.2 Knee, swelling

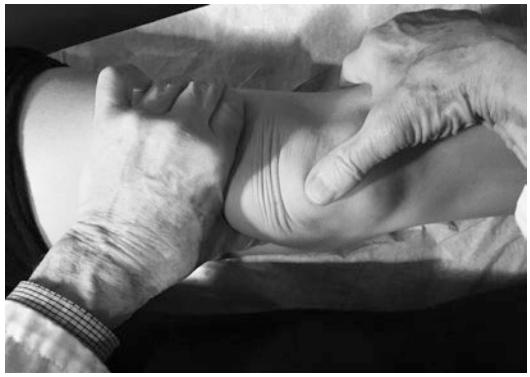

Fig. 23.3 Patella click

as the patella bumps against the femur indicates excessive joint fluid. The displaced synovial fluid immediately returns to the previous distribution upon release of the patella (Fig. 23.3).

Chronic knee disease may be obvious by its gross enlargement due to bony projections, distended synovia, and deformation. The short leg, if not already detected when the patient was supine, can be identified with the patient sitting. Use the same thumbs-below medial malleoli to compare relative positions.

Temperature

Increased Temperature

A sensitive sign of inflammation in the knee is an increase in warmth. To check by comparison with the other knee, cup the knee with one palm, and then use the same hand cupped on the other, moving back and forth several times. This method enables the examiner to detect slight differences in temperature. A barely detectable increase can reflect extensive inflammation in this large joint.

Tenderness in the knee may be elicited by firmly pressing the patella against the femur and then running both thumbs around the periphery. Tenderness could be limited to the patella and its supporting tendons; it could involve the deeper joint, indicating arthritis. Irregular ridges that may be tender along the bony margins of the tibia or femur are exostoses from osteoarthritis.

Movement

The rounded medial and lateral condyles of the femur act in a rocker motion on the slightly depressed surfaces of the menisci. The patella glides smoothly within the groove (trochlear) that runs between the condyles when flexing and extending the knee.

Pain or a palpable sensation of grinding or crepitus on passive flexion and extension of the knee can originate from the patella bursa or from within the joint. The finding indicates the roughened surfaces of the synovial membranes or of loosened concretions.

Keeping one hand cupped on the kneecap, extend and flex the lower leg, feeling for normal tracking. The patella slipping off line or popping fully out of the groove indicates subluxation, an important sign of knee instability. Its causes may be from an uneven contraction of the quadriceps or hamstrings or from a knock-kneed stance.

Patella

The patella (Latin. *shallow dish*) is a sesamoid, discoid-shaped bone around which the broad tendon of the quadriceps muscle wraps itself. The tendon then extends beyond the patella to anchor directly onto the tibia. The patella has an important function by providing the quadriceps with greater leverage for extending the lower leg. It has been discussed previously as it pertains to the overall function of the knee. This section covers pathology that relates directly to the patella.

Subluxation

While the patella can be pushed slightly toward either side, excessive movement or misalignment can reflect deterioration or injury to the supporting ligaments. Subluxation of the patella occurs when it moves out of the trochlea groove during extension and flexion of the knee. The displacement will resolve spontaneously once the leg is fully relaxed (Fig. 23.4).

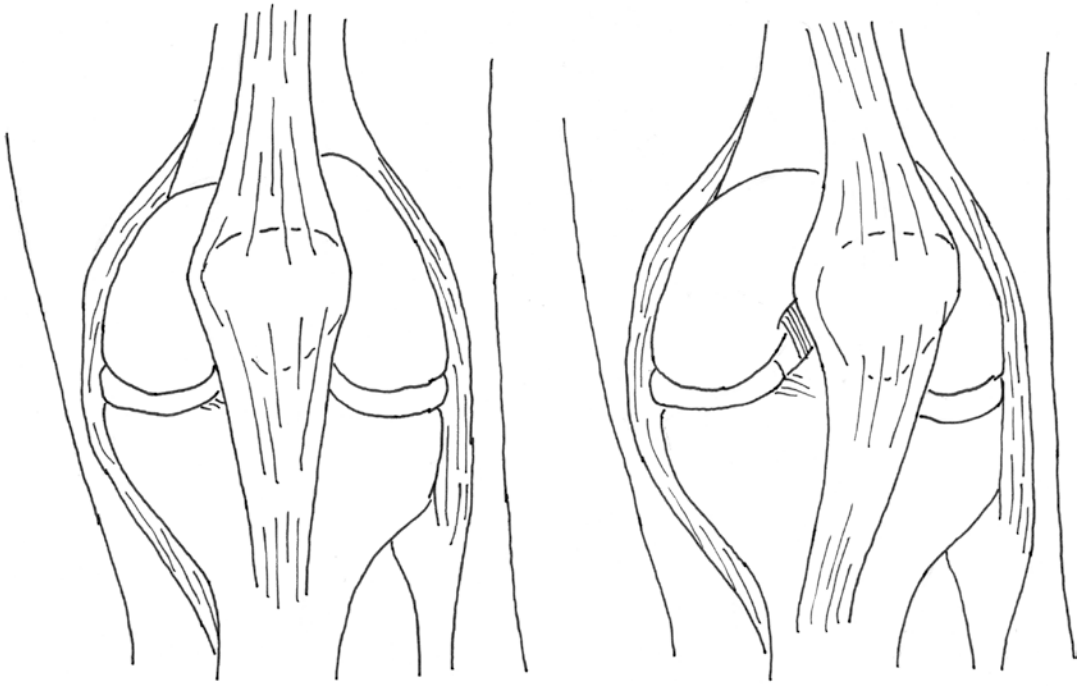

Fig. 23.4 Patella subluxation

Dislocation

Since the patella is at the apex of a large triangle when the knee is flexed, there is a small wonder that it is susceptible to impact injury. It is commonly affected in falls and on deceleration injuries in sports and on collisions with a dashboard.

A dislocated patella results from trauma serious enough to rupture the ligaments that normally define its space-limited movement. A patella when dislocated following blunt trauma is obvious on inspection. Tenderness over the kneecap is an expected finding. The injury greatly impedes the ability to extend the lower leg, and the effort is painful. Its resolution requires surgical intervention (Fig. 23.5).

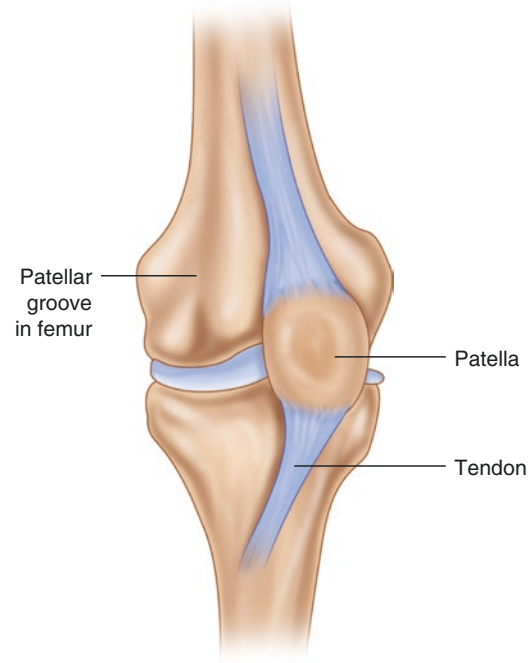

Fig. 23.5 Patella dislocation

Fracture

After an acute injury, a fracture of the patella is likely if there is a hematoma over or around it.

The severity ranges from a minor crack or the shattering of the patella. In most cases, tenderness and pain on extending the lower leg, as in a dislocation, will be present. The serious complication of hemarthrosis should be suspected when the degree of pain and disability is severe.

Pre-patella Bursitis

Familiarly known as "housemaid's knee," prepatella bursitis is the result of extensive kneeling on hard surfaces. The bursa is a thin sac that covers the anterior surface of the patella. Inflammation occurs from protracted direct pressure. Erythema and direct tenderness over the patella is typical while synovial effusion is usually slight. A distended patella bursa may prevent full flexion of the lower leg [4].

Patello-Femoral Inflammation

There are two forms of stress activities that affect the kneecap: (1) heavy, abrupt strain and (2) extensive, protracted overuse from daily activities.

1. Excessive force on the extended knee produces stretching of the upper and lower tendons in which the patella is suspended. The cause is usually athletics that involve jumping. Tenderness will probably be found in the overstretched tendons, not on the patella. The knee may feel stiff and bending is painful. There is a tendency for slack ligaments to allow the knee to buckle under pressure.
2. Low-impact overuse, most commonly the result of long-distance running, causes bursitis on the inner surface of the patella. The most telling physical sign is pain on compression of the patella against the femur. A sensation of grinding or crepitus may be felt by both patient and examiner.

Both stretched tendons and bursitis of the knee are associated with pain on climbing and descending stairs and on kneeling and squatting.

Chondromalacia Patella

This term refers to a degenerative condition within the cartilage that may result from sustained overuse and, in fact, be a further complication of "runner's knee." The cartilage softens and deteriorates. Pain is at first confined to the outer and lower margins of the patella but can worsen to involve the entire knee. Diagnosis at the time of the physical examination is provisional and further definition requires imaging studies.

Popliteal Fossa

Using the hands clasped around the knee, palpate the popliteal fossa for tenderness or for a bulge or pulsation. This test is not necessary if there is no symptom such as pain or swelling in the knee or claudication of the leg.

Arterial Disease

Generally, the fingertips must be firmly impressed in order to feel the normal pulsation of the popliteal artery. A diminished or absent pulsation may indicate obstructive disease by thrombus or atheroma in the superficial femoral or the iliac artery. Palpation of the femoral artery in the groin will help locate the obstruction.

An exaggerated pulsation in the popliteal fossa can result from a hyperactive circulation from any cause or from an aneurysm or kink in the popliteal artery.

Venous Disease

The venous channels in the popliteal space are extensive, and venous "lakes" can be large. Tenderness in the popliteal space should entertain the possibility of localized thrombophlebitis. The proximity of the femoral vein, where venous thrombi are highly dangerous, underscores the need for an expedited definitive workup when popliteal thrombophlebitis is suspected.

Popliteal Cyst (Baker's Cyst)

Synovial fluid can leak from the bursa in the posterior knee and form a cyst. It usually goes unnoticed until it becomes large enough to restrict flexion of the lower leg or impair venous flow or when it ruptures. Such cysts tend to occur more often in knees that are abnormal from arthritis or previous injury [5]. They can appear at any age. Compared with ultrasound imaging, the physical examination has a low sensitivity for determining a small popliteal cyst [6] (Fig. 23.6).

The bulge from the cyst can best be seen from behind with the patient standing. It is mainly on the medial aspect where the semimembranosus muscle attaches to the medial condyle of the tibia. It is palpable as a soft mass by ballottement, and there is generally little if any tenderness. On flexion of the knee, the examiner will feel an increase in volume when fluid from an anterior bursa is forced into popliteal bursa.

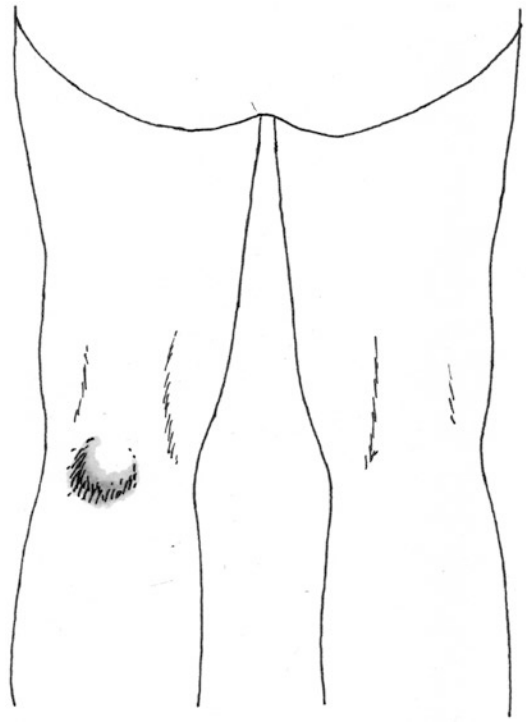

Fig. 23.6 Popliteal cyst

Rupture of a popliteal cyst is a serious problem. Forceful compression during heavy exertion is the usual cause. The resulting sudden pain, leg swelling, and discoloration can simulate the findings of thrombophlebitis [7]. Indeed, thrombophlebitis is a frequent complication.

The rupture of a popliteal cyst in this highly vascular area can cause extensive ecchymosis over the lower legs. Alternatively, the blood may dissect along the fascial planes and appear only as a purpuric area around the malleoli.

Arthritis

Common to all forms of chronic arthritis of the knee is stiffness with difficulty on bending and straightening the lower leg, pain on motion and weight-bearing, swelling, and an increase in heat. Clicking or grinding may be experienced or detected on flexion and extension. There are some landmarks, however, which help distinguish the different kinds of arthritis. Some of the major diagnostic points separating the various forms of arthritis are noted here.

Osteoarthritis

Stiffness in the knee with or without pain is present on arising in the morning. In the early stages, it lasts no longer than 30 minutes in contrast to the protracted symptoms in rheumatoid arthritis. A decreased ability to flex the knee easily and fully is an early sign of osteoarthritis. A revealing clue is a slight increase in warmth over the affected knee. Tenderness in osteoarthritis is variable.

As the condition advances, the intra-articular cartilage becomes rough. Bony enlargement, mostly at the tibial head, occurs with formation of osteophytes at the edges. Crepitus from intra-articular inclusions is common. Effusion occurs but is not generally as prominent as other forms of inflammatory arthritis (Fig. 23.7).

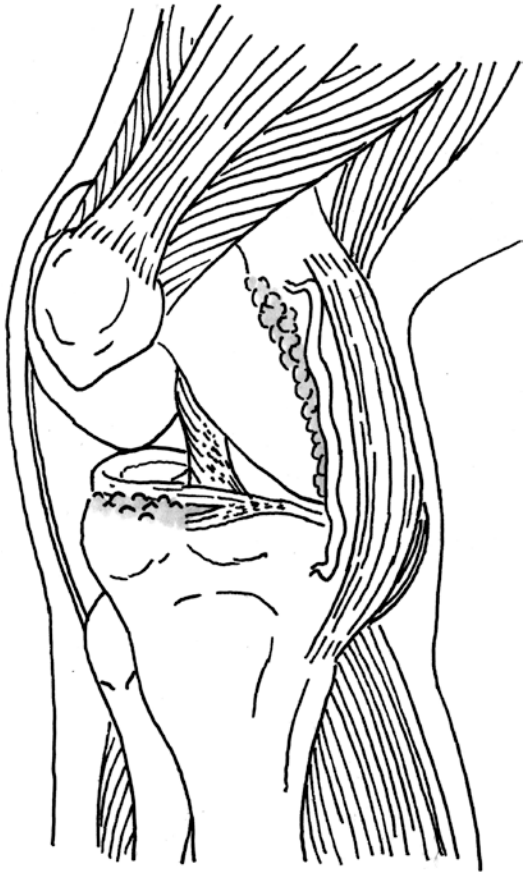

Fig. 23.7 Osteoarthritis

Rheumatoid Arthritis

Painful tendonitis and synovitis in the knee can be an early sign of rheumatoid arthritis. A highly distinctive feature of rheumatoid arthritis is that the symptoms and physical findings are bilateral and fairly symmetrical.

The loss of full extension of the knee is an early sign of rheumatoid arthritis. Eventually, major deformities from stretched ligaments occur with the lower leg turned inward or outward. Atrophy of the quadriceps is common. Rheumatoid nodules tend to occur along the Achilles tendon. Diagnostic changes in the interphalangeal joints are generally present.

Increased pain in rheumatoid joints before it rains is such a common experience that the association cannot be dismissed as a myth. On the other hand, no explanation by change in atmospheric pressure or other physical phenomenon has been convincingly established.

Gouty Arthritis

Deposits of urate crystals form tophi that precipitate in cartilage, synovia, and tendons, the knee being one of the predominant sites [8]. In chronic gouty arthritis, tophi are generally not tender. The thin skin overlying a tophus is very pale owing in part to the white, chalky material beneath. Overall, the surface of the tophaceous knee becomes markedly irregular.

Acute gouty arthritis is notorious for its rapid onset and the extreme severity of pain and tenderness in the fiery red and swollen joint. While it is the great toe that is the most typical site, the knee is involved fairly often. The most intense inflammation usually subsides after several days to a week.

Calcium Pyrophosphate Deposition Disease

This form of arthritis is fairly common but is frequently not identified. Formerly called "pseudogout arthritis," its pathogenesis is the precipitation of calcium pyrophosphate crystals in the synovial fluid, the knee being the most likely site [9]. The crystals damage cartilage and synovia. A definitive diagnosis is made, like gout, by identifying culpable crystals obtained from aspiration of synovial fluid.

Pyrophosphate deposition disease causes acute arthritis in other joints, such as in the wrist and shoulder. Such locations of pain are not similar to those affected by osteoarthritis or gout. Unlike acute gouty arthritis, the pain tends to last for several weeks. When the pyrophosphate deposits occur in multiple joints, as it often does, the joints are not affected symmetrically as they typically are in rheumatoid arthritis.

Calcium pyrophosphate deposition disease is a disease of the elderly. If a patient younger than 60 years develops it, then an underlying, etiologic risk factor should be looked into: hyperparathyroidism, hemochromatosis, and hypomagnesemia.

Septic Arthritis

While septic arthritis is mentioned after more common forms of acute arthritis, it is the first diagnosis to think of in any acute arthritis. The infection can destroy a joint virtually overnight; it can certainly pose a danger to life.

The knee is the most likely site of large joint infection. Septic arthritis usually occurs in a joint already damaged, most especially from rheumatoid arthritis. The pathology consists of hyperplasia of synovial membranes, rapid destruction of cartilage, and eventually erosion of bone. Pain, tenderness, and swelling are cardinal features along with fever, an increased temperature of the joint, and erythema. All signs may develop within a single day. Fever is highly likely but can be slight if present at all in the elderly and the immune-compromised patient.

The clinician must be mindful that a septic joint can easily be passed off as a crystal arthropathy, as a flare-up of rheumatoid arthritis or as a recurring systemic connective tissue disorder. It can occur when attention is on the more typical features of a systemic illness. Complications of viral infections (rubella, HIV), endocarditis, and reactive arthritis (Reiter's syndrome) can include septic arthritis.

Staphylococcus aureus is usually the infectious organism in a septic joint. Gram-negative bacteria, however, are more likely among intravenous drug users and in the immunocompromised. Infectious arthritis from *Neisseria gonorrhoeae* is atypical in that it tends to migrate from one joint to another, including the knees; it is also highly destructive. In sickle cell disease, a septic joint complicates vascular-occlusive events, and it occurs most often in the knee. Sometimes septic joints are multiple with the knee most frequently involved and then the elbow, shoulder, and hip [10].

The tick-borne infection of Lyme disease often presents with large joint polyarthalgias that are transient and accompanied by constitutional symptoms with or without the characteristic rash, erythema chronicum migrans. The spirochetal infection does have a predilection for causing an acute arthritis in one joint, and that joint is usually the knee. In some patients, responses of the cellular and humoral immune system lead to chronic arthritis with erosion of cartilage and bone.

Reactive Arthritis

A bacterial infection can cause an autoimmune reaction that in turn causes acute non-septic arthritis 2–4 weeks later. Reactive arthritis is mostly a disease of the younger generations.

Antigens to various microorganisms have been detected in the synovial membrane of affected joints, but the synovial fluid is sterile. An infection of the genitourinary system, the gastrointestinal tract, or the nasopharynx is implicated [11]. In many cases, however, no preceding infection can be identified, and the diagnosis depends on establishing serial serological testing [12].

Formerly called "Reiter's syndrome," reactive arthritis usually occurs in the knee and/or ankle, although the fingers, wrists, and vertebrae may also be affected. Usually, several joints are involved and not in a symmetrical distribution. There are otherwise no distinguishing characteristics of the inflamed joints evident on the physical examination. Non-articular manifestations that are clues to the diagnosis are often present. These include fever, weight loss, uveitis, and mucocutaneous ulcers in the mouth and glans penis.

Hemarthrosis

A wrenching twist or blow to the knee may cause bleeding into the joint. Hemophiliacs and those on anticoagulants are more susceptible to hemar-

throsis of the knee from much lesser degrees of trauma. Even a small bleed into the joint can cause severe pain, particularly on movement. Bruising around the knee may or may not be visible. Whether damage to cartilage and chronic arthritis is a complication of hemarthrosis or a contributing factor to the bleed is indeterminate.

Neuropathy

Neurogenic Arthropathy

Loss of feeling in the feet will eventually blunt the defensive reflexes of walking and weaken muscle tone. These changes promote the development of abnormal weight-bearing surfaces. Bone and cartilage undergo destructive changes with the knees being highly susceptible. As expected, neurogenic diseases of the knee and foot are relatively painless. To establish the diagnosis of neurogenic arthropathy, it is necessary to find regional defects in motor and sensory functions.

Peripheral neuropathy can affect the quadriceps, causing weakness of extension of the lower leg as well as atrophy in the muscle. A decrease in sensation in the anterior medial thigh may be associated. Referred to as the "patellar-femoral syndrome," this condition has its basis, most likely, in diabetes.

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Foot

24

The foot is an often forgotten territory of the standard physical examination – unless the patient calls attention to a problem there. Even with no symptoms, however, a quick assessment of the foot is worthwhile. Sometimes a problem can be identified that may eventually affect other parts of the weight-bearing system. A relatively minor defect in the foot is often easily correctable, an intervention that could possibly prevent an incremental disability in the years and decades to come.

Circulatory and neurological issues in the foot have been presented in Chap. 22. In this chapter, emphasis is on the infectious, inflammatory, and physical aspects that are localized to the foot. The details are presented geographically, fore to aft. Even so, keep in mind that the foot functions as an integrated unit of all its parts (Fig. 24.1).

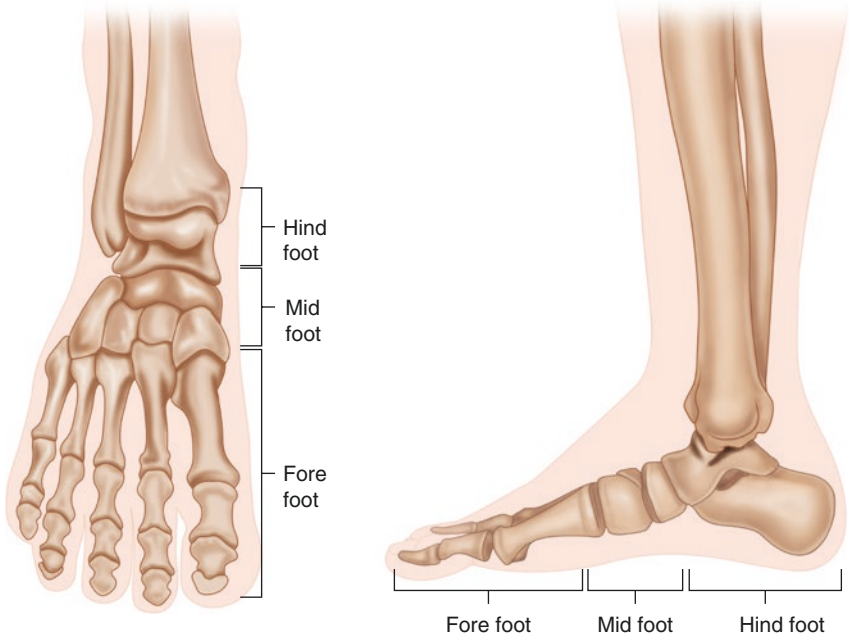

Fig. 24.1 Regions of foot

R.E. Phillips, *The Physical Exam*, https://doi.org/10.1007/978-3-319-63847-8_24

Essentials

Toes
Ball
Arch
Heel

Forefoot

Through most of the day, the toes are confined to a damp, hot space. They are often cramped into footwear designed as much for looks as for function.

For convenience and consistency, the toes are designated by descriptive abbreviation, as were the fingers with an "F." Examples are "RT1" for the right hallux and "LT5" for the left pinkie.

Onychomycosis

Thickened, totally opaque or discolored nails that are brittle result from indolent fungal infections. The ingrown toenail (almost always on the great toe) is a potential site for invasive bacteria or fungi. A discharge – either serosanguinous or purulent – at the edges of an ingrown nail indicates that the skin has already been breached.

Tinea Pedis

Tinea pedis (or "athlete's foot") is a fungal infection between the toes that produces peeling of white skin, leaving a red, macerated base often with a fissure and crusting. It is almost always in the T4–5 interdigital space and can extend to other spaces. Tinea pedis can affect the nails and spread onto the sole where it has earned the name "moccasin foot." This extended infection is notable for marked scaling and cracks in the thickened skin. An odor emitted from the affected skin is attributed to a superimposed bacterial infection.

The infection thrives in the closed environment of the shoe where warmth and humidity are high. Without a change in ambience, it proves stubbornly resistant to treatment.

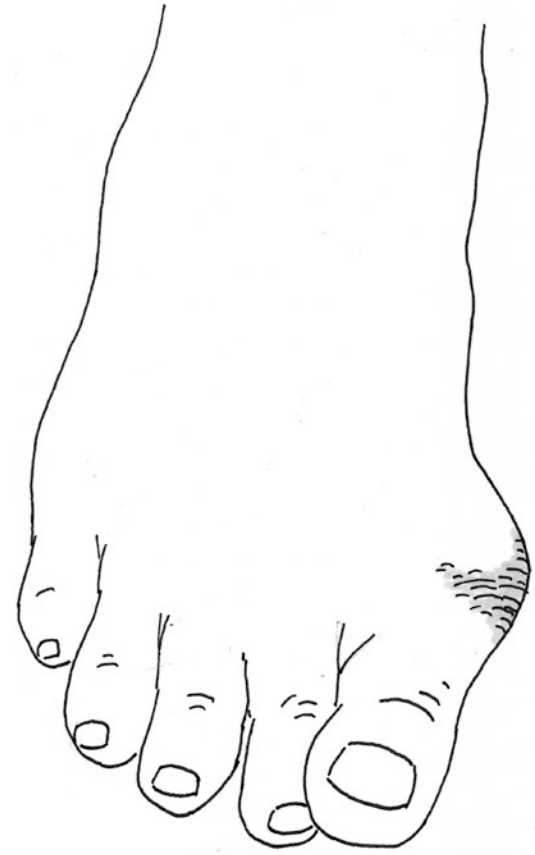

Fig. 24.2 Bunion

Bunion

A bunion is a lateral bony protrusion at the base of the hallux. The toe points medially. The deformity has most likely developed over years from ill-fitted shoes. Any condition that affects the supporting structure of the foot, however, can lead to formation of a bunion. Rheumatoid arthritis is a classic example (see below). The deformity from a bunion can be severe enough to cause the great toe to cross over on top of the second toe (Fig. 24.2).

Corn

A dome-shaped corn is a thickened knob of skin, usually over the dorsal aspect of the toes. It occurs over a bony prominence, the result of friction from a tight shoe. High heels, among

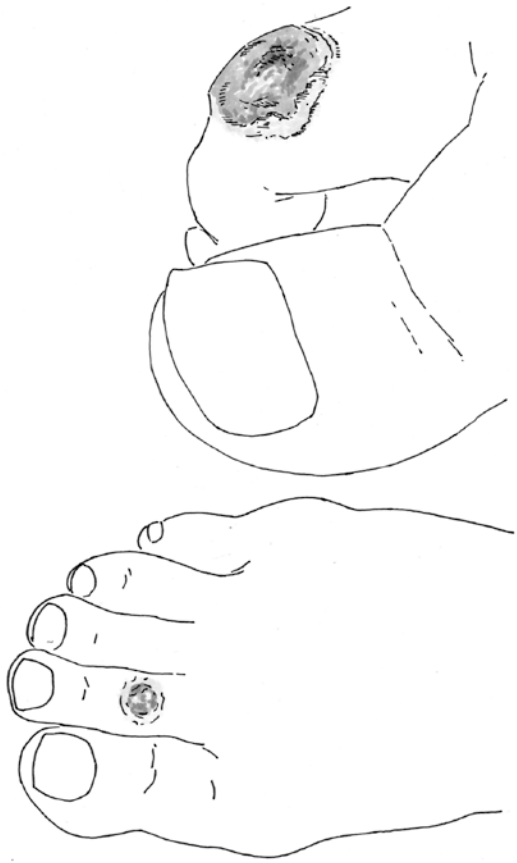

Fig. 24.3 Corn

other unnatural burdens placed on the foot, increase the rubbing atop the toes exponentially (Fig. 24.3).

Hammer toe

The hammer toe is propped up proximally and bent down in the middle joint. Usually the toes involved are T2 and 3. The prominent proximal metatarsal joint is particularly exposed to a friction-produced corn (Fig. 24.4).

Claw toe

Toes are bent at both the middle and distal joints. Damage to a peripheral nerve in the foot causes the toes to clench. Any or all of the smaller toes may be involved (Fig. 24.5).

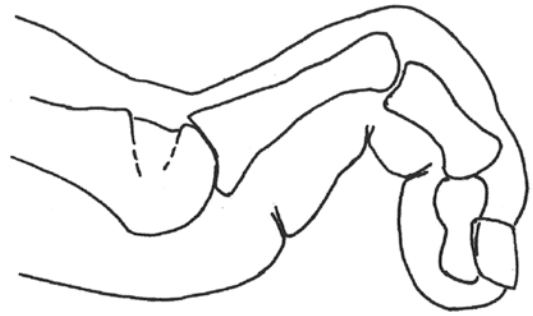

Fig. 24.4 Hammer toe

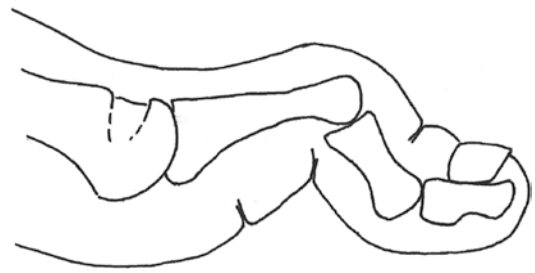

Fig. 24.5 Claw toe

Both the hammer toe and the claw toe can result from an imbalance of muscles in the foot or from an exceptionally high arch. Perhaps the most common cause, however, comes from ill-fitted shoes.

Ischemia

Rubor of the toes is characteristic of arterial obstruction with the legs in a dependent position. Ischemic injury in the toes, including necrosis, can result from obstruction anywhere along the extent of the aorta and its major branches. An extensive collateral circulation among the three arterial branches in the foot spares the toes to some extent from obstruction in these vessels.

As with the fingers, the toes are susceptible to damage from diseases of small arteries and arterioles. In these conditions, pallor is the outstanding characteristic. The degree of small vessel disease can be limited to one or two toes but may extend to all. Ischemia of the toes may be episodic or unremitting. Causes include

cryoglobulinemia, Raynaud's disease, dermatomyositis, and various forms of collagen vascular disease.

Rheumatoid Arthritis

The earliest symptoms of rheumatoid arthritis occur in the small bones of the hand and feet in the great majority of people. In many, symptoms at first are only in the feet and ankles. There they tend to occur simultaneously in both feet and involve multiple joints.

Virtually anyone with long-established rheumatoid arthritis will have serious issues with the foot, including deformities of the toes. The foot becomes stiff. The arch collapses and the foot turns outward. Thickening of the synovia and weakening of ligaments result in a hammer toe, claw toe, callus, or bunion, often in combination. The rheumatoid foot is more susceptible to stress fractures of the metatarsals from osteopenia.

Gouty Arthritis

Of course, acute gouty arthritis comes to mind on seeing an extremely painful and tender swelling of the metatarsal-phalangeal joint of the great toe. All joints of the foot, however, are susceptible to attacks, as are the ankle and the knee. And not all acute arthritis flairs in T1 are due to gout.

Sesamoiditis

Flanking the underside of T1 are small sesamoid bones. These can be inflamed and painful after hard use. The condition is most prevalent among those placing greatest stress on the toes: runners and ballerinas (Fig. 24.6).

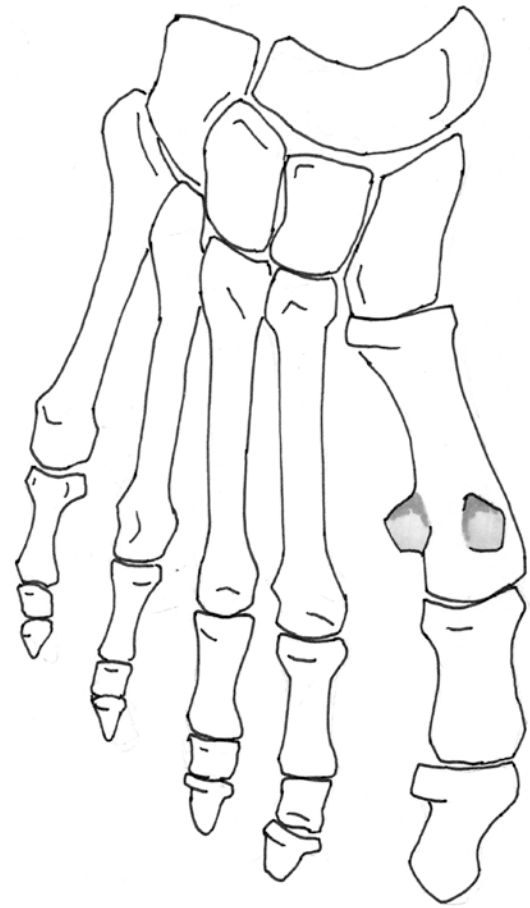

Fig. 24.6 Sesamoiditis

Midfoot

Ball

Normally, weight-bearing of the ball of the foot is distributed fairly evenly across the metatarsal heads. Two common conditions arising from an abnormal distribution of weight are the "plantar wart" and "Morton's neuroma." Both, by the way, are misnomers, explained as follows.

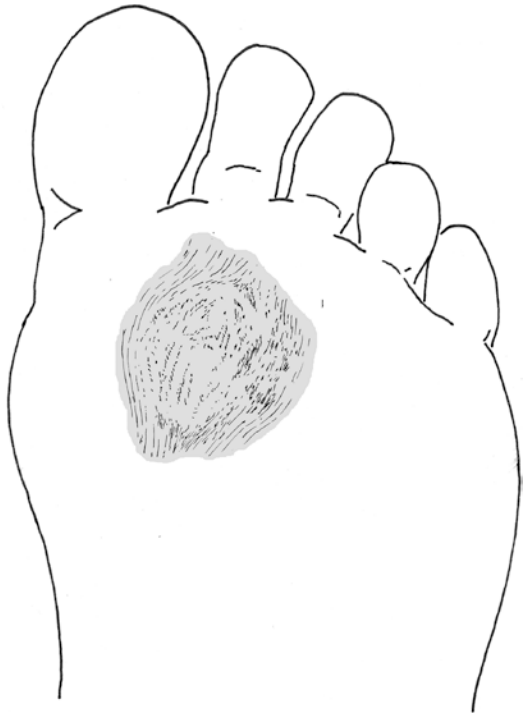

Fig. 24.7 Callus

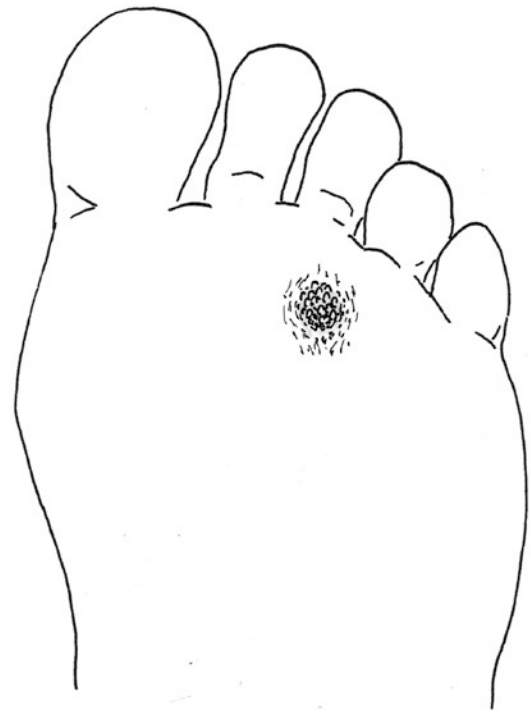

Fig. 24.8 Wart

Callus

A callus can develop on the ball of the foot where the load is more concentrated, usually over the distal metatarsal area of the great toe. It is a waxy-appearing thickening of skin that can be mistaken for a verrucous and indeed is referred to as a "plantar wart." A callous differs from a corn in that it occurs over areas where skin is normally thick, namely, over ball or heel (Fig. 24.7).

Wart

A wart (verrucous) is a hard, papillary lesion having a granular surface. Typically, the surface of a wart is studded with minute black dots that are capillary loops. A wart can be highly debilitating when on a weight-bearing surface. It is caused by the human papillomavirus. The "plantar wart," however, is not a wart but a callus. Furthermore, it has nothing to do with frogs (Fig. 24.8).

Neuroma

A plantar "neuroma" is another localized thickening at the ball of the foot. It is not a neuroma but an enlargement of ligaments skirting the middle metatarsals. In what is commonly known as "Morton's neuroma," fibrotic ligaments rub against the nerve with every step. The area is tender. The enlargement may or may not be palpable.

Arch

The highly flexible arch of the foot is a shock absorber, providing an effective spring for alternating weight-bearing. Any anomaly of the arch strains other supporting areas.

Pes Cavus

An abnormally high arch places an exaggerated burden on both the forefoot and the hindfoot. On bearing weight, the foot does not flatten normally.

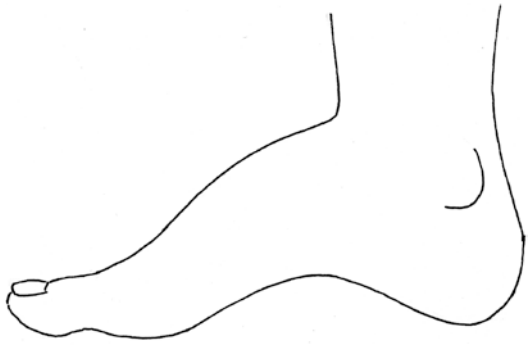

Fig. 24.9 Pes cavus

Callus formation is a frequent complication, as are deformities of the toes, including a hammer toe or claw toe. In severe cases, gait is stiff and unstable. About half of those with pes cavus have an inherited neuropathy involving both motor and sensory systems, Charcot-Marie-Tooth syndrome (Fig. 24.9).

Pes Planus

This condition is the commonplace "fallen arch" or "flat feet." The weakness of the arch causes an overpronation with each step. The strain in turn causes the posterior tibial tendon to lose its alignment. The strain can result in pain from inflammatory symptoms of the broad plantar fascia. Pes planus is probably the most common cause of plantar fasciitis (Fig. 24.10).

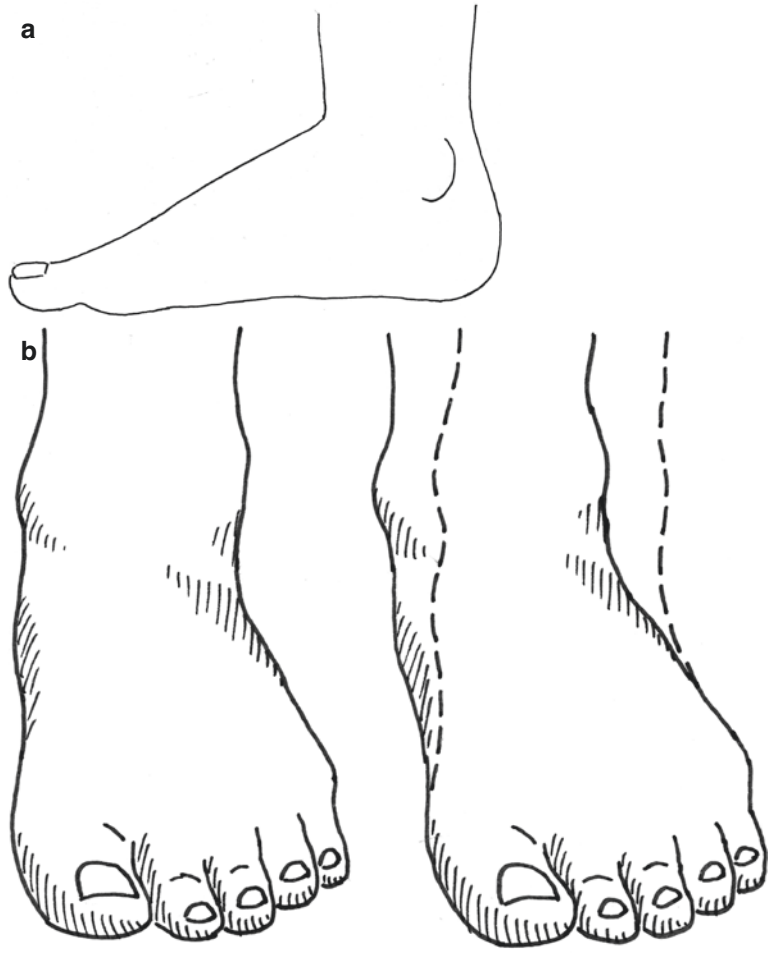

Fig. 24.10 (**a**) Pes planus. (**b**) Foot pronation

Hindfoot

The causes of pain across the heel are many. They are most often the result of repetitive physical stress, including the extremes involved in some sports.

Heel

Achilles Tendonitis

Tendonitis over the Achilles tendon causes tenderness where it attaches to the calcaneus. Dorsiflexion of the foot will exaggerate the heel pain.

Fracture

A stress fracture of the calcaneus, usually from a violent athletic effort, causes severe pain in the heel. Repetitive stress of lesser impact may result in a painful calcaneal bursitis.

Atrophy

The tough but compressible pad, just beneath the calcaneus, is subject to atrophy from underuse when pain in the area limits walking. The aged and the very obese are particularly prone to thinning of this cushion [1].

Neuropathy

If heel pain is associated with tingling, burning, or insensitivity, consider a neurological cause. Entrapment of a nerve, known as the "tarsal tunnel syndrome," is usually responsible. Exploration of the heel for sensory loss is diagnostically appropriate.

The tunnel is formed by a thick outer ligament, the flexor retinaculum pedis, that extends from the medial malleolus to the calcaneous. Through it pass tendons of muscles to the foot, the tibial nerve, and the posterior tibial artery and vein in a tight squeeze. This branch of the tibial nerve provides sensory input over the sole.

Plantar Fasciitis

The plantar fascia is a tough, fibrous band that stretches from the posterior calcaneus and fans out across the sole to the metatarsal heads. There it separates into five ligaments. Conceptually, the plantar fascia is likened to a bowstring that is a little shorter than the bow to which it is bound at each end. The action stabilizes the arch while preventing the arch from flattening excessively on bearing weight. Also, the plantar fascia provides a shock-absorbing spring with each step (Fig. 24.11).

Plantar fasciitis is more of a wear and tear problem than an inflammatory one, evidently caused by microscopic injuries to the collagen fibers [2]. Risk factors are overuse (from prolonged standing or from running) and marked obesity. In addition, tightness of the Achilles tendon from habitually worn high heels may factor in its development by allowing the tendon to gradually shorten and develop an acquired "ankle equinus."

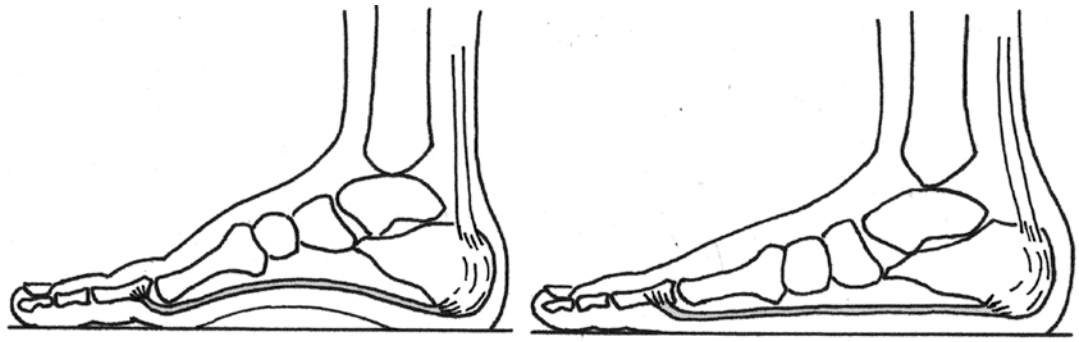

Fig. 24.11 Plantar fascia: normal and stretched

Plantar fasciitis can be diagnosed with reasonable certainty by a combination of factors. These include pain in the heel on the first step of the morning or on standing after prolonged sitting. Sharp pain may be present on palpation of the medial plantar region of the calcaneus. Discomfort by passive dorsiflexion of the ankle and great toe is also characteristic [3].

While the earliest and most prominent area of pain and tenderness from plantar fasciitis is in the heel, it eventually extends forward and can involve the entire sole.

Heel Spur

Calcium can be deposited anywhere along the plantar fascia. Most commonly, concretions occur at first around the calcaneus and extend out along the fascia. The "heel spur" is an extension from this buildup of calcium. The deposits cause tenderness and can be enough to produce disability. Even when extensive, however, they are seldom palpable (Fig. 24.12).

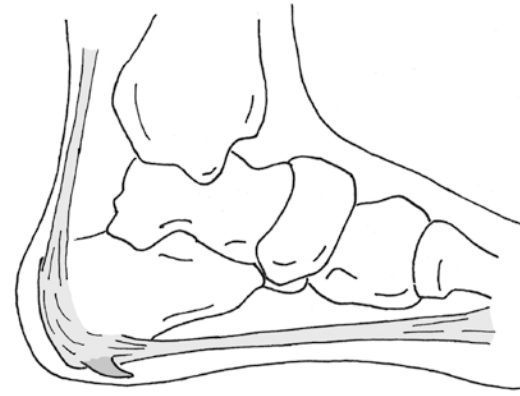

Fig. 24.12 Heel spur

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Part V

Patient Standing

Standing

25

Position #5 Patient is standing.

Remember to lower a raised hospital bed before the patient steps down.

This chapter encompasses those observations that are best or can only be examined with the patient standing. The architecture of the axial system that is *designed* for weight-bearing is looked at *during* weight-bearing. The evaluations can provide meaningful information that might otherwise have gone unnoticed. When treatable problems of structural integrity that affect bearing weight are identified, addressing them could save undue wearing from muscle and skeletal stress over the years and perhaps decades to come.

Essentials
Posture
Limbs
Hernia

A studied glance at the fully exposed back is enough to assess symmetry of shoulder height and thoracic contour. Iliac crests will be at the same level unless there is malalignment of the hip or a short leg. The natural contour of the vertebral column will be evident: a normal slight concavity in the cervical and lumbar spine and slight convexity in the thoracic spine.

Spinal Column

Standing: Erect

It is not necessary to palpate and/or percuss along the column, as is commonly practiced, unless the patient has back issues. Various lumps and spots on the back will likely have been noticed during the chest examination. Two conditions with important implications are mentioned here: spina bifida occulta and neurofibromatosis.

Spina Bifida Occulta

This *forme fruste* of spina bifida is the result of a failure in the development of the vertebral arch to close completely. In this less severe form, there is no protrusion of the meninges or cord. The condition usually involves only one vertebra, the fifth. It occurs in 10–20% of the population but remains completely asymptomatic in the vast majority of these throughout life. Even so, there is some evidence that spina bifida occulta is associated with problems of enuresis in children [1]. In adults, there is greater susceptibility to spondylolysis, such as a linear fracture or dissolution of the involved vertebral body [2].

Physical evidence of spina bifida occulta occurs over the lower back, typically occurring just above the gluteal crease. It may appear as a deep dimple, a vascular nevus or hemangioma,

R.E. Phillips, *The Physical Exam*, https://doi.org/10.1007/978-3-319-63847-8_25

a lipoma, a hypopigmented patch of skin, or a tuft of hair. In a more complex form, a dermal sinus that drains intermittently may be present.

Neurofibromatosis

This neurogenic disorder has been mentioned elsewhere. In the most common form, neurofibromatosis type 1, neurofibroma can appear almost anywhere on the body. These are prominent bumps that are remarkable for their softness. Their origin is a germline mutation in the supporting sheath that envelopes nerve. Although disfiguring, the tumors are benign. Other than cosmetic, they can develop within the spine and cause scoliosis.

The incidence of pheochromocytoma in patients with neurofibromatosis type 2 is far greater than the incidence in the general population [3]. The diagnosis of a pheochromocytoma is often first made by computer imaging as an "incidentaloma," not by clinical intuition. In one study, 7.7% of patients with neurofibromatosis and labeled as type 1 had a pheochromocytoma, although most were asymptomatic [4]. This rare but important endocrine disorder is described further in Chap. 8.

Multiple hyperpigmented macules throughout the body, known as café au lait spots, are a consistent cofeature with neurofibromatosis. These are light brown spots that spread at least 10 cm across and appear just as the name suggests: a spill of coffee with milk. When café au lait spots are present but without neurofibroma, they are probably incidental and not part of the syndrome of neurofibromatosis [5].

Standing: Forward Flexion

Minor degrees of vertebral deviations can be easily missed on the general physical examination. It is helpful to have the patient assume a position in which these abnormalities may be exaggerated and therefore more noticeable.

The patient is asked to bend forward at the hips so that the trunk is about 45° from horizontal. There is no need for the patient to try to touch the toes. The effort may easily strain the muscles and joints that are unaccustomed to it. Extreme forward bending to touch the toes requires more flexion of the hip than flexion of the spinal column. Exceptions may be made in the occupational and the sports physical examination.

Flexibility

The normal spinal column is surprisingly flexible, permitting wide rotation and some degree of flexion and extension. These functions can be quickly checked on during the routine physical examination.

Adding torso movement to a physical examination required for a job more demanding physically than "desk work" is appropriate. Observing the patient rotating the trunk from side to side and bending forward and arching backward are basic procedures in the occupational physical examination.

Stiffness

Difficulty in bending far forward may reveal significant vertebral disease, be it rheumatoid, osteoarthritis, or other bone-affecting disorder prevalent in the aging population. Any restriction of full movement could have a muscular, ligament, neural, or vertebral origin. On the other hand, the lack of physical activity at any age can produce inordinate stiffness.

Ankylosing Spondylitis

Ankylosing spondylitis is a chronic inflammatory condition that most often affects the joints of the spinal cord. The intervertebral discs lose cartilage, ligaments around the vertebrae thicken, and fibrous rings around the vertebra eventually ossify.

The result of this inflammatory polyarthritis is pain and stiffness with tenderness over the sacroiliac joints. On inspection, the slight, normal lordotic curve in the lumbar region is diminished. Flair-ups of ankylosing spondylitis often occur with systemic signs of inflammation, most notably uveitis.

Posture

Abnormal curvature of the spine places a burden on weight-bearing tolerance and – when severe – on respiratory mechanics. Recognition of spinal malalignment in youthful patients may serve to initiate corrective measures that could reduce progression.

Scoliosis (Greek *scoliosis*, Curvature)

Detection of lateral deviation of the spine, if slight, requires careful inspection. Mild degrees of scoliosis can be easily overlooked. To screen for scoliosis, it is helpful to run the pointer finger down the vertebral prominences from neck to sacrum. Any curvature of the spine will become more obvious. Scoliosis may already have been suspected earlier in the examination if corroborating clues of skeletal asymmetry (such as deviation of the trachea and/or a difference in leg length) were detected (Fig. 25.1).

By having the patient bend in forward position, malalignment of the spine becomes much more obvious. The scapula on the convex side of thoracic scoliosis will be somewhat more elevated and lie slightly more cephalad than the opposite scapula (Fig. 25.2).

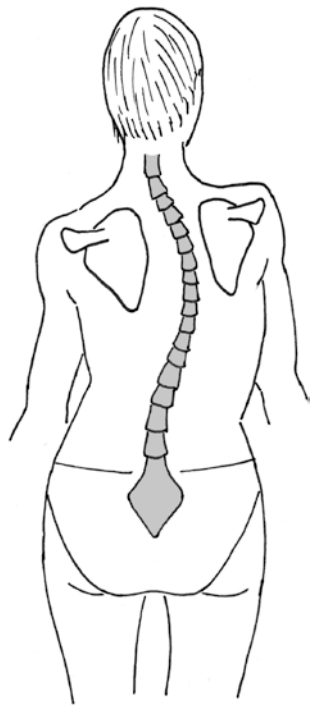


Fig. 25.1 Scoliosis, straight up

People with scoliosis have issues not so much directly with the spine but with the secondary problems of a shorter leg or splaying of the legs that imposes a burden on the feet. An asymmetric load on the pelvis stresses muscles during upright activities. Scoliosis can progress slowly and inconspicuously over many years and, in addition, eventually compromise the capacity of breathing in addition. The finding of scoliosis in a general examination may also be an early clue to a chronic systemic disorder such as neurofibromatosis, osteomalacia, or empyema.

Kyphosis (Greek *kyphos*, Hump)

The normal spine has a slight kyphotic angle in the thoracic region. When this gentle curvature is exaggerated, it is denoted "hyperkyphotic." The term "kyphosis," however, is more commonly used for this condition (Fig. 25.3).

There are several forms:

Posture-based This vertebral condition is the habitual slouch of teenagers and the aged. There are no bony deformities and correction can be at least partially achieved voluntarily but not without considerable effort.

Juvenile Osteochondrosis A developmental disorder of the spine can become apparent during adolescence in which some vertebrae become irregular and wedge-shaped, usually in the mid-thoracic region. Known more commonly as Scheuermann's kyphosis, it is more prominent than the kyphosis incurred solely from long habitual practiced poor posture.

Congenital Abnormal development with fusion of some vertebrae during gestation can result in severe kyphosis in the child. It can encroach on chest expansion and decrease lung capacity.

Osteoporosis With advancing age, low bone mass and degenerative disc disease contribute to

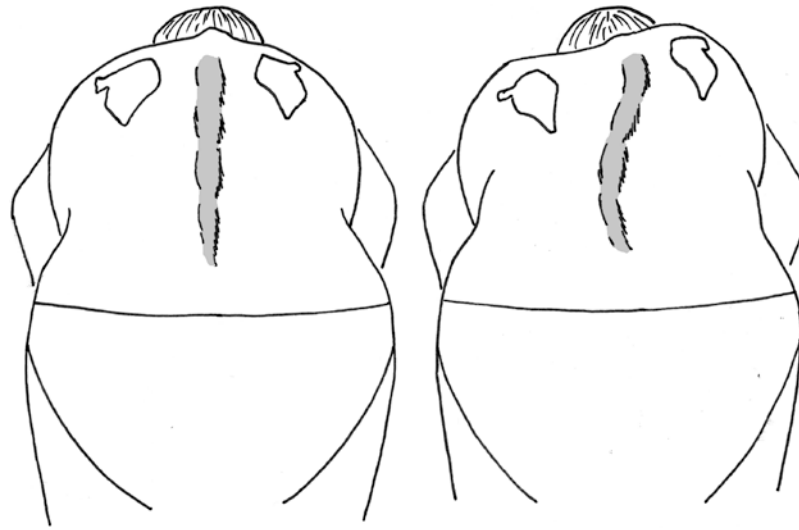


Fig. 25.2 Scoliosis, bent forward

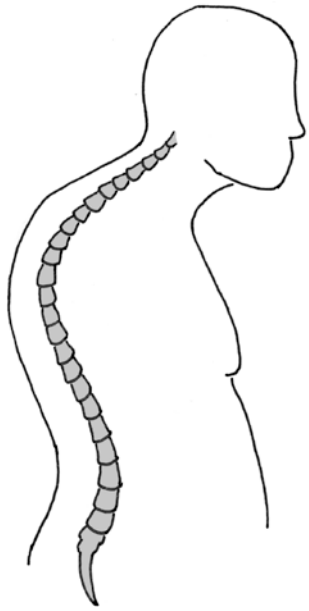


Fig. 25.3 Kyphosis

exaggeration of the anterior curvature of the thoracic spine [6]. The "dowager's hump" is a familiar consequence. Many with severe kyphosis have multiple minute fractures of the vertebrae [7]. Various physical capabilities may be severely curtailed, partly owing to compromised pulmonary function.

Infection Most notorious for destruction of vertebrae and severe kyphosis is tuberculosis. The condition is referred to as a "gibbous" deformity. Destruction of the vertebral body deforms the normal cylindrical shape, causing the spinal column above it to angle into a sharp, forward bend. The renowned hunchbacks of history were probably victims of vertebral tuberculosis.

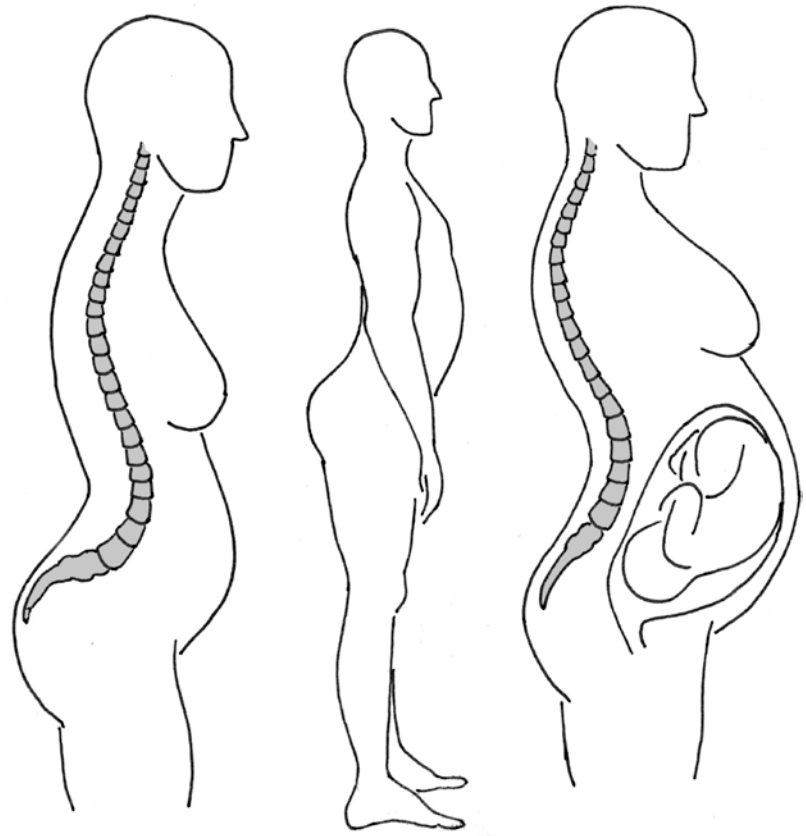
Neoplasm If kyphosis is of recent origin – and is not traumatic – it is imperative to look for a bone-destructive process. A metastatic tumor or multiple myeloma may be responsible.

Lordosis

Lordosis is an exaggeration of the normal inward curve of the spine in the cervical and lumbar regions. The excessive curve is counterbalanced by the back-leaning attitude of the upper body. The deformity strains the muscles of the hips and trunk. Marked obesity is a common cause in which the pelvis tilts forward, while the upper spine inclines backward to compensate for the stress of excessive abdominal weight. Temporary lordosis occurs late in pregnancy. Spondylolisthesis and shortened Achilles tendons from habitually worn high heels are other possible contributing problems. Lumbar lordosis is a significant risk factor for developing low back pain on prolonged standing [8] (Fig. 25.4).

The observation has been reported that at least moderate lumbar lordosis is common among college women who are dancers and

Fig. 25.4 Lordosis



gymnasts. The connection may be the extreme range of motion and repetitive hyperextensions required for these pursuits. There is no information, however, on the relationship of training or incident of injury [9].

Vertebrae

Low Back Pain Syndromes

Pain in the low back rivals all other complaints heard in the clinician's service for adults. It is here that the vertebral column makes its sharpest curve, where muscles and ligaments are stressed and where the longest nerve emerges from the spinal cord. Pain in the low back that is mainly lateral or in the buttock is referred to as "sciatica." It is the radiation of pain into the leg by way of the sciatic nerve that defines the syndrome. This syndrome has been described in Chap. 21, the position in which it is easiest to evaluate for sciatica. This chapter covers other pain-producing low back issues that involve the vertebral column; in general, these conditions do not have as well-defined signs of etiology and are, therefore, less likely to be recognized on the physical examination.

Causes of non-traumatic low back pain are usually of gradual onset, while symptoms tend to be more confined to midline. This separation of midline and lateral causes, however, is highly arbitrary owing to the variability of these conditions.

Herniated Nucleus Pulposa

While vertebral discs usually herniate laterally to produce sciatica, a massive herniated disc can project anteriorly and cause midline low back pain. Such herniation is most likely from the natural degeneration in disc matrix that goes along with advanced aging and weakening of supporting

ligaments. In the younger, healthy person, it takes a violent deceleration injury to cause a disc to herniate.

Typically, a lumbar vertebra will herniate laterally and compress the sciatic nerve, producing a radiculopathy. Compression is distal to the tip of the spinal cord so that features of central nervous system impingement, defining a myelopathy, do not occur (Fig. 25.5).

In the extreme form of disc herniation, sciatic nerve damage is bilateral. One result is the loss of urinary bladder and bowel control. Peripheral neuropathic changes are evident: calf wasting, decreased ankle jerk, and reduced strength of ankle dorsiflexion. With involvement of sacral nerves 2–4, anesthesia in the "saddle" area is an expected finding. This complication of autonomic function when acute is well recognized as a surgical emergency.

Physical signs by both sensory and motor testing, attempting to localize a herniated disc, correlate rather poorly with the findings by imaging and surgery. There is, on physical examination, a low sensitivity for identifying a disc herniation, while the specificity for determining the level of herniation is but moderate [10].

Spinal Stenosis

Narrowing of the intervertebral spaces within the lumbar spine reduces the outlet for neural and vascular elements to pass into the periphery. Causes of spinal stenosis can be the degenerative remodeling of osteoporosis, the thickening of ligaments, a herniated or bulging disc, bone spur, or spondylolisthesis. In more than half of the patients with spinal stenosis, the symptoms are bilateral. Such discogenic etiologies with lateral projections may be misinterpreted as trochanteric bursitis or arthritis in the hip joint. Yet despite the high rates of anatomic stenosis on imaging studies in older people, there are many who are completely without symptoms related to the spine [11] (Fig. 25.6).

In spinal stenosis of the lumbar region, pinching of the sciatic nerve causes paresthesia and weakness that radiate from low back and into the buttocks and legs. Characteristic of spinal stenosis in the lumbar area is leg pain on standing and walking, with or without back pain. Called "pseudoclaudication," it takes longer to recover from a walk than the few minutes it takes for pain to subside in ischemic claudication. Pain is lessened by bending forward and by recumbency. It is not exacerbated from sitting [12].

Neurogenic claudication from spinal stenosis in the lumbar area affects only the nerve roots in the cauda equine since the spinal cord does not extend that far – not like spinal stenosis in the cervical area where the cord itself could be interrupted.

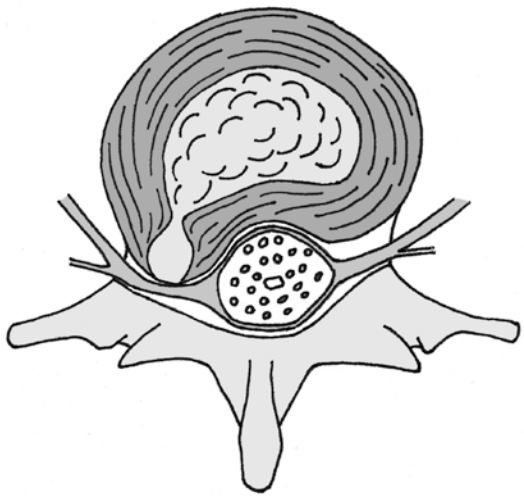


Fig. 25.5 Herniated disc

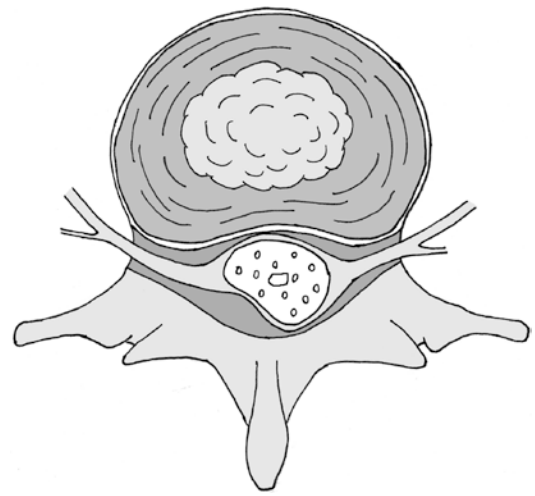


Fig. 25.6 Spinal stenosis

Vertebral Body Collapse

As a complication of systemic osteoporosis, the weakened vertebral body may give way. Pain, often sudden in onset and from slight exertion, ranges from slight to agonizing. Yet images can demonstrate several collapsed vertebra in individuals who have had little or no previous back pain. Nevertheless, they will certainly find themselves a bit shorter. Complications of a collapsed vertebra can be intestinal dilation and constipation.

Spondylolisthesis

Slippage of one vertebra body over the one immediately below usually occurs in the lumbar area. It is almost always involves the fifth lumbar vertebra and often follows a fracture, whether spontaneous or traumatic. The misalignment burdens the muscular and ligament support system for weight-bearing. It may narrow the intervertebral nerve outlet space and result in sciatica.

The result of lumbar spondylolisthesis is general stiffness of the lower back and hamstrings. To relieved pain on weight-bearing, the patient may lean forward in a semi-kyphotic attitude. Further compensation consists of asymmetric guarding in pelvic rotation, giving a waddling character to walking. Atrophy of gluteal muscles is evidence of a peripheral neuropathy as a complication.

Neoplasia

Pain – with or without tenderness – in the spine that is not explained by recent trauma should bring to mind the possibility of a tumor, including a metastasis. It is estimated that one or two persons in ten with cancer of all types have metastases to the spine [13, 14]. The majority occur in older patients from cancer in a visceral organ, while tumors in the younger patients are mostly benign [15]. In many cases, a metastatic lesion in the spine is the initial sign of a malignancy [16]. Multiple myeloma is particularly prone to cause osteolytic lesions in the vertebrae; it is a diagnosis certainly meriting consideration in an elderly patient with pain and tenderness over the vertebrae and who is also anemic.

Pain from malignant disease in the spine has several possible sites of origin, as outlined here:

(a) Regional: The bulk of a tumor can expand in sensitive areas and lead to the instability of supporting elements. The osteolytic properties of the tumor may result in the fracture of a vertebra. Pain from such lesions is generally worse with movement and relieved with rest. In contrast, a low back, pain-producing metastasis has been reported that had osteoblastic properties [17].

(b) Neurological: By compression of a major nerve root, a tumor can produce pain and weakness that has the segmental distribution of radiculopathy. In the lumbosacral spine, it is the sciatic nerve that is most likely involved. Weight-bearing aggravates the pain while recumbency relieves it. If the tumor compresses the spinal cord, signs of myelopathy will also be present. These signs of central nervous system impairment include spasticity of the leg, exaggerated deep tendon reflexes, and a plantar withdrawal response. As the condition progresses, perception of proprioception, pain, and temperature will be impaired. The autonomic functions of bowel and urinary bladder eventually become compromised by the growing tumor.

(c) Inflammatory: Pain of malignant bone disease that is not related to movement and that never lets up may be from inflammatory mediators produced by the tumor [16]. Pain is often described as a deep-seated, aching quality that persists despite rest or relaxation of moving parts. It lasts through the night but tends to lessen during the day, perhaps through distractions.

(d) Concurrent conditions: Postural adjustments for weight-bearing to compensate for back pain place stress on other areas of the axial system, causing additional skeletal discomfort. In addition, drugs used to treat malignancy can themselves result in painful complications. One such worry is the patient on corticosteroids and other immunosuppressive therapies. More susceptible to infection in general, the patient on these medications, for example, may develop tuberculosis of the spine with the associated pain assumed to be from the tumor. Chronic pain in the back

from a long-ago injury – for example, a soldier sustaining trauma from an explosion – may tend to assume that an exacerbation of pain is from the old injury and overlook the possibility of it being caused by a neoplasm.

Limbs

Leg Alignment

It may not be until the patient stands that misalignment of the legs becomes evident. These conditions can have an important long-term impact on the hips, knees, and feet.

Genu Valgum

Better known as "knock-knee," the knees in genu valgum angle inward. Here a problem in terminology comes up: "valgum," a Latin word, refers to outward. In all but the mildest cases, the knees abut when standing and prevent the feet from coming together. Wear and tear of the hips and knees is accelerated, making them more susceptible to injury and to osteoarthritis. The feet tend to pronate, reducing or eliminating the natural arch (Fig. 25.7).

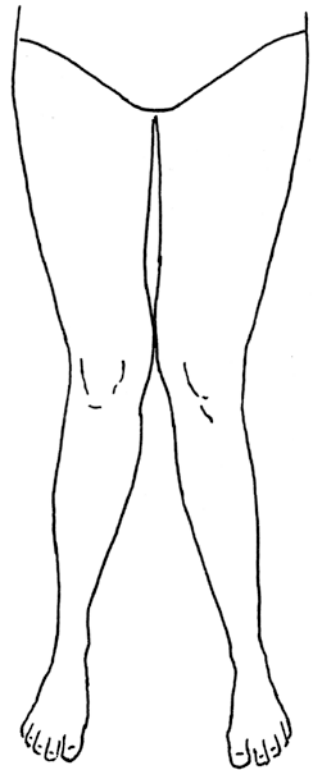


Fig. 25.7 Genu valgum

Genu Varum

The outward angulation of the knees is commonly called "bowed legs." It is typical in the first few years of life. The legs gradually align, usually by the age of three.

Bowed legs may persist because of abnormal growth of bone. Called "Blount's Disease," the deformity occurs mainly in the upper tibia. When severe, weight is borne on the outer rim of the foot, not utilizing the advantage of the entire arch. The misalignment at the hips and knees subjects them to unnatural stress. Here, too, the terminology is confusing for "varum" is Latin for inward (Fig. 25.8).

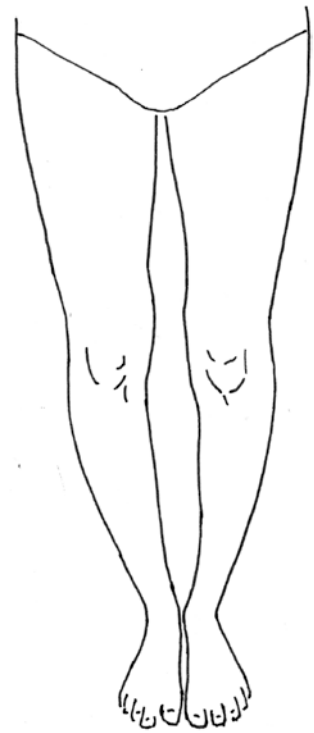


Fig. 25.8 Genu varum

Limb Length

Marfan's Syndrome

It is in the standing position that a disproportional length of arms and legs to torso is most evident. In Marfan's syndrome, the long legs have an

abnormal greater floor-to-pubis than pubis-to-crown measurement. A typical sign of Marfan's syndrome in the digits is shown in Fig. 3.11 of Chap. 3 (Fig. 25.9).

Scoliosis and chest deformities are commonly associated. Disparate abnormalities of connective tissue are frequently associated. These include severe myopia from a displaced lens, redundancy of the mitral valve, and weakness of major arteries, particularly at the aortic root. Because of the high systolic blood pressures generated by heavy exertion, advice for these exceptionally tall people may include avoiding vigorous athletics that could result in exercise-induced arterial dissection.

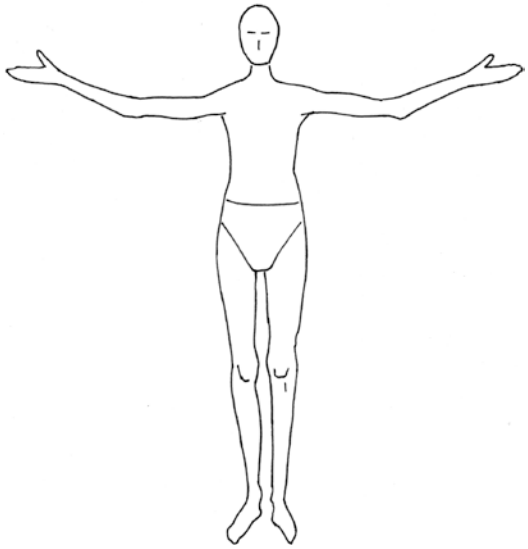


Fig. 25.9 Marfan's syndrome

Limb Length Discrepancy

Over the years of asymmetrical weight-bearing, a difference in leg length can be a major factor in painful wear on the back, hip, and knee. This subject has been addressed in Chap. 21.

Habitus

Body shape has a bearing on risk factors for cardiovascular diseases. One measure has led to the concept of the "apple or pear" habitus. Basically, persons who are larger in the belly than in the hips (i.e., the apple shape) have a greater incidence of the "metabolic syndrome." That is, they are more likely to have hypertension, insulin resistance, hypertriglyceridemia, and reduced high-density lipoproteins. Those who are larger in the hip area than the abdomen (i.e., the pear shape) tend to have fewer risk factors and lesser chance of having diabetes or heart or peripheral vascular disease (Fig. 25.10).

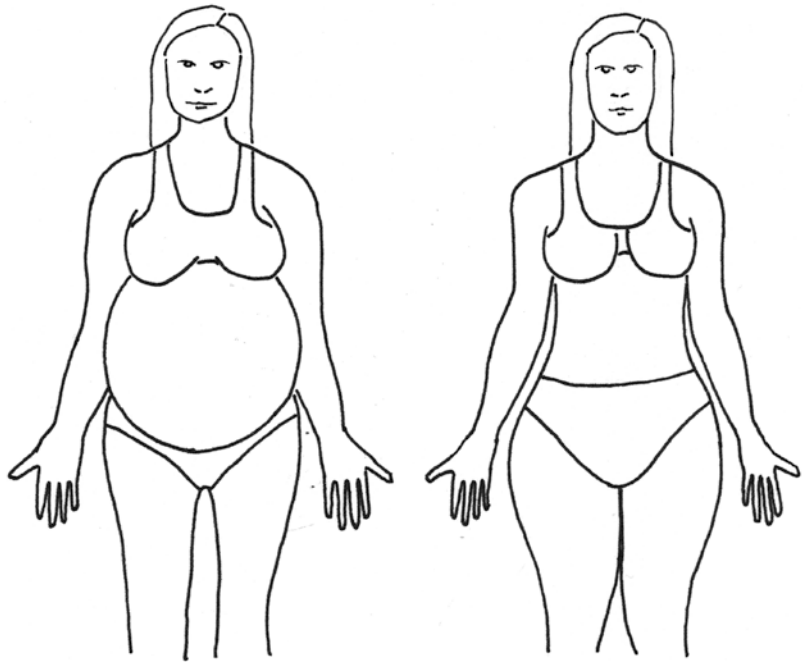


Fig. 25.10 Left: apple and right: pear habitus

Foot

Pronation

If there was any question of a fallen arch during the sitting examination, check for pronation during weight-bearing now that the patient is standing. The "flatfoot" will be quite evident. Further assessment can be made by having the patient walk in place. The examining middle fingers slid under the arch can readily detect excessive pressure as each foot takes on the full body weight (Fig. 25.11).

Inguinal Fascia

Defects in the inguinal fascia are most easily detected with the patient standing. For this advantage, testing for a possible hernia is best performed at this point of the examination.

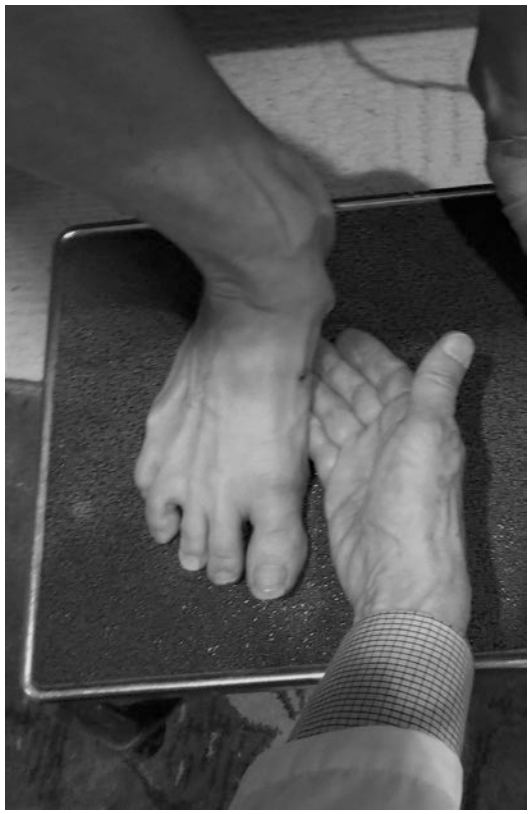


Fig. 25.11 Walk in place to evaluate pes planus

Direct Hernia

A "direct" inguinal hernia defines a weakness or gap of the abdominal muscle fascia where it inserts in the groin. Through it, a portion of the intestine may protrude. A small, protruded bowel will have a dome-shaped, firm bulge that is taut with air. If the hernia is reduced and therefore not palpable, an impulse may be felt over the inguinal ligament from the sudden increase of intra-abdominal pressure induced by a sharp cough.

The direct inguinal hernia is a liability of aging, though it can occur at any age. It is evidently not a product of heavy lifting. The great majority of direct hernias occur in men. There is always the potential of incarceration of the hernia, resulting in acute abdominal obstruction. The incarcerated segment of bowel may be very small, taut with trapped air, gangrenous, and mistaken for an enlarged lymph node.

Indirect Hernia

The indirect inguinal hernia is virtually confined to men. It is performed most conveniently during the genital examination, as is explained in Chap. 26.

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Gait

26

The neurological examination continues by testing sensations conveyed in the longest tracts. The point-specific testing performed earlier is now augmented with observations that require integrating the multiple functions involved in the simple act of walking. Included in this chapter are test procedures that reveal gait-impairing defects: station and tandem walk.

To repeat from the previous chapter, the clinician need not precede this far in an able-bodied patient who has no symptoms of and has not already exhibited signs of a neuromuscular disorder. Even so – and keeping in mind the frequency and disastrous consequences of falling in the elderly population – the clinician is urged to carefully observe gait in patients in their sixty's and beyond regardless of the absence of obvious risks earlier in the examination. Gait assessment is also prudent on patients who have conditions that make them susceptible to neuromuscular complications, namely, diabetes and alcoholism.

In the realm of the neurologist, the observation of gait is one of the most useful tests [1]. Indeed, it has been expressed that a good history along with evaluating walking can provide a diagnosis in the great majority of neurological cases. Not all clinicians, of course, will have the proficiency of a neurologist in the skill of evaluating gait, finding it too complex and/or subtle for diagnostic value. Even so, some simple guidelines to recognize the various gait abnormalities can prove helpful.

Essentials
Station
Tandem walk
Gait
Turning

Station

Proprioception, tested earlier with vibratory stimuli, is evaluated again in another way. The patient faces the examiner with heels together and toes separated in the army's "at attention" position. The examiner touches the patient's shoulders lightly while the patient gets into position. The patient closes his or her eyes, and, having been given prior notice, the clinician removes the hands but keeps them close by. Reassure the patient of your being right there in case of losing balance (Fig. 26.1).

Any reduced sense of proprioception is evident by unsteadiness immediately on closing the eyes. The test emphasizes the dependence upon vision of these patients to navigate upright activity. The demonstration of instability with the eyes closed has a pragmatic implication: at home, the patient must always have a light on when getting up at night.

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Fig. 26.1 Station

Tandem Walk

Have the patient walk, touching heel to toe in a straight line. This test is not easy; the patient should be so forewarned! The test requires the synchronization of several components of neuromuscular control: proprioception, coordination, muscular power, and sense of kinesiology (the sense of body position in space). The challenge of integrating it all with the tandem walk test is a staple exercised by traffic control officers. The test will readily expose a sensory or coordination neuropathy (Fig. 26.2).

An abnormal tandem walk will be evident in the first two or three steps as the patient gropes to find footing. Caution is advised in interpreting this test as tandem step walking can overtax the ability of the inveterate couch potato who may be entirely normal neurologically.

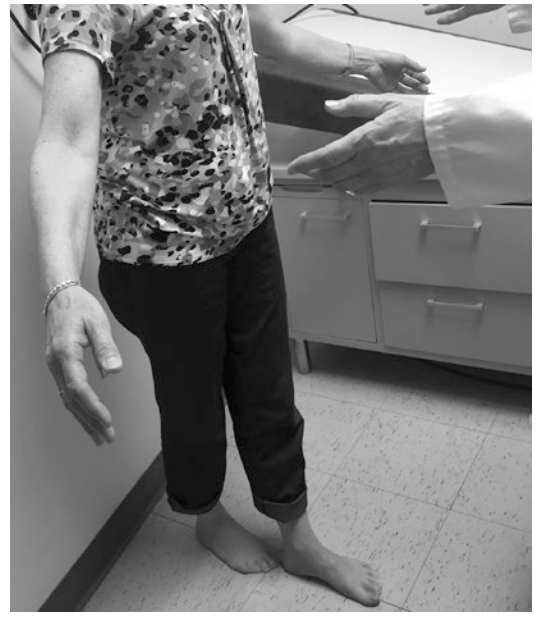

Fig. 26.2 Tandem walk

Gait

The mundane skill of walking is the complex synchronization of several component functions: proprioception, motor, vestibular, visual. Those who cannot integrate all may feel as if they were "repetitively hurling oneself into space" [1].

Gait is best observed while the patient takes about ten steps along a straight line at an accustomed pace. He or she is then told to turn and return.

The speed, length of stride, and rhythmic flow of stepping are cardinal observations of the neurologist. One arm should swing forward as the leg on the opposite side advances (not the opposite comedic sequence perfected by Benny

Hill). Two additional observations are important: (1) the swing of the arms and (2) the ability to turn smartly. Taking three or more steps to reverse course is considered abnormal; it is a common observation in patients with Parkinson's disease.

Walking consists of three highly synchronized phases in which the terms "stance" and "swing" are used:

1. The stance foot is planted on the ground, beginning with the heel. The full weight of the body is accepted with the foot at a plantar flexed attitude.
2. The foot on the opposite side prepares for the swing forward by lifting the heel. The ankle draws up, a dorsiflexed action necessary for the foot to clear the ground.
3. The swing foot moves forward with lifting by the hip and knee. The step-off transfers the full body weight to the metatarsophalangeal joints of the stance foot. The instant that the heel on the swing limb strikes the ground, the stance foot goes into plantar flexion and the cycle begins again.

Of the many types of abnormal gaits, the characteristics of some diagnostic patterns are presented here.

Cautionary Gait

By cautionary gait is meant walking with unsteadiness from fear of falling. Prevalent in the elderly, it is probably the most common of all walking disabilities. It may be called by some "dizziness," although neither light-headedness nor vertigo is involved. Here, the burden of advanced aging slows walking and renders it awkward, the result of declining sensory input, central processing, and motor agility. Frequently adding to these liabilities are vestibular, joint, and visual problems. Anemia, exertional dyspnea, and claudication further contribute to the uncertainty of navigating afoot. And certainly, a habitually sedentary life contributes to the overall disability.

In addition, polypharmacy significantly increases walking disability. It is established that a person who takes four or more different drugs a day (as most elderly people do) incurs an independent risk factor for falling [2]. This relationship exists regardless of the kinds of drugs taken. The increased risk of falls in older people is particularly high regarding sedatives, hypnotics, tricyclic antidepressants, selective serotonin reuptake inhibitors, and serotonin norepinephrine reuptake inhibitors [3]. Risk of falling is less well documented concerning the antihypertensive drugs, although hypotensive, pre-syncopal episodes from these blood pressure-lowering drugs may be far more common that is recognized.

Spastic Gait

The typical sequelae of hemiparesis from a corticospinal (or pyramidal tract) lesion consist of spasticity on one functional set of muscles that overpowers the opposing set of muscles. In the leg, it results in the predominant extension of the knee with weakness of flexion and of ankle dorsiflexion. In the arm, flexion and adduction are dominant. This recognizable pattern of spasticity is the most common form of stroke in hypertensive vascular disease (Fig. 26.3).

In unilateral disruptions of the pyramidal tract, each step exhibits circumduction. That is, the waist is tilted up and away from the forward moving, spastic leg so that the foot can clear the ground. The leg is thus thrown forward in a circular pattern. This slightly rocking motion on the

Fig. 26.3 Stroke, unilateral spastic gait

swing phase of gait prevents the foot from scraping against the floor (Fig. 26.4).

When the corticospinal lesions are bilateral (the diplegic gait), walking is only achieved with a shuffling gait as the adducted thighs cause the knees to bump. The toes scrape along with each step. In the extreme of spasticity in the hips, the patient walks with a scissors gait. Here one leg crosses in front of the other with each step. Weight-bearing falls mainly on the tips of the toes. These patterns are the essential features of cerebral palsy (Fig. 26.5).

Fig. 26.4 Stroke, swing leg

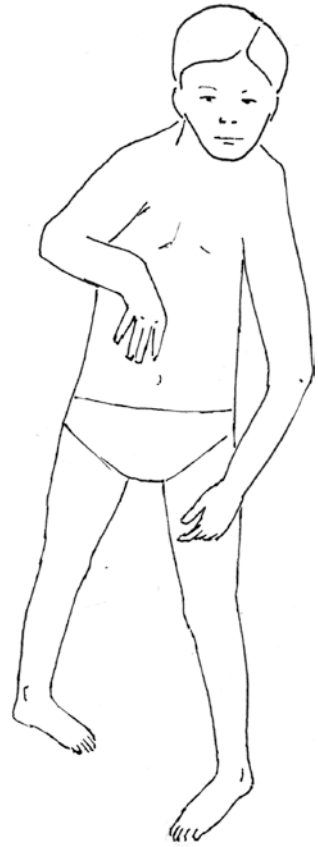

Fig. 26.5 Diplegic spasticity gait

Extrapyramidal Gait

The prototypic disturbance of the extrapyramidal system is Parkinson's disease.

It is manifest by increased flexion tone and rigidity throughout the body. Walking in the advanced stages consists of short steps as if walking on ice. The body is generally bent forward to maintain gravitational balance, causing an involuntary acceleration of walking. Tremor throughout may be exaggerated during walking. The arm swing is decreased, and several steps may be required to reverse direction (Fig. 26.6).

Fig. 26.6 Parkinson's disease gait

While Parkinson's disease belongs to the elderly, drugs can cause Parkinsonism at any age. Most culpable are the anti-psychotic and antiepileptic drugs, stimulators of gastrointestinal motility, and calcium channel blocking agents [4].

Neuropathic Gait

The etiology of the neuropathic gait includes the usual suspects: diabetes, alcoholism, HIV, vitamin deficiencies, and toxic exposures. In practice, the cause of an impaired neuropathic gait cannot be established in about half the cases.

Lower motor neuron disease generally comprises a combination of sensory and motor aspects with one predominating:

Sensory Defect The ability to walk despite a major sensory neuropathy requires visual clues. Gaze is fixed on the pathway ahead. The patient walks on a broad base with the side-to-side footprints greater than 4 inches apart. Tabes dorsalis (a form of neurosyphilis, now rare) predominantly involves the sensory pathways.

Motor Defect A distinctive feature of a peripheral motor neuropathy is the "foot drop" (weakness of foot dorsiflexion). The toe of the foot points downward when the leg is dangling. To compensate and prevent the foot from dragging with each step, the knee is brought up high. The foot is thrust forward and planted with a slap. This characteristic is known as the "steppage" or the "equine" gait by resembling the movement of a horse's forelegs (Fig. 26.7).

Weakness of dorsiflexion in the foot can be tested on the great toe and foot. When weakness in dorsiflexion of the toe or foot is slight or questionable, test further by having the patient walk on the heels. Heel walking clearly demonstrates the defect.

If the peripheral neuropathy causing foot drop is unilateral, suspect a fifth lumbar nerve radiculopathy. If bilateral, consider a demyelinating disease in the anterior horn cells within the spinal

Fig. 26.7 Equine, steppage gait

cord. In foot drop, the major weakness is in the anterior tibialis muscle from a defect in the peroneal nerve, a branch of the sciatic nerve.

Ankle Equinus

A shortened Achilles tendon points the foot downward, so that excessive weight is borne on the toes, akin to the balarina's *en pointe*. The condition is defined by the inability to actively dorsiflex the foot at least 10° from horizontal. Walking that favors placing the weight on the forefoot is akin to the steppage gait of a motor neuropathy.

Ankle equinus is perhaps the most common deformity of the foot and ankle; it is a leading

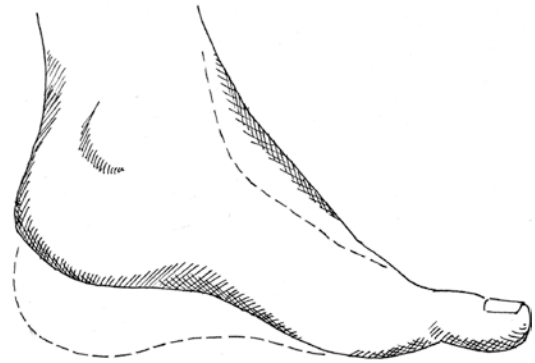

Fig. 26.8 Ankle equinus

contributor to many forms of pathology. Even so, ankle equinus is seldom recognized. Indeed, patients complain not of the underlying condition but rather its complications: pain and deformities that may result anywhere in the foot, the calf, the knee, or the hip.

During the gait cycle, the normal forward pivotal action of the ankle is limited, and the foot does not easily clear the surface in the swing phase. To compensate, the heel lifts off early on initiating each step with a tendency toward "toe walking" and raising the knee high. Strides are shorter than normal, while the gait tends to be a bit slower and typically bouncy. Weight is shifted toward the forefoot while pronation of the foot reduces the need for dorsiflexion (Fig. 26.8).

These compensatory actions may be a major factor in the development of foot and ankle maladies [5]. Possible complications are plantar fasciitis, pronation of the foot, lateral foot pain, Achilles tendonitis, and lesions of excessive load on the forefoot. The latter include arthritis of the metatarsals, sesamoiditis, bunions, callouses, neuromas, and hammer toes. In addition, the shifting of the body's center of gravity may be enough to burden the knee, hip, and back.

In ankle equinus, the tight Achilles tendon is most likely from shortening of the middle bulk of the gastro-soleus muscle complex rather than initial shortening of the Achilles tendon, favoring plantar flexion while limiting dorsiflexion of the foot. Ankle equinus can result from a talus bony deformity, ankle trauma, arthritis, or a bone spur. Spasticity of the posterior calf muscles

(as in cerebral palsy) may be responsible. The same muscles may shorten from disuse, as from crutches or a short leg cast. High heels worn habitually are an important contributory cause, and some loss of muscle fibers has been documented [6].

People with diabetes have a particularly high incidence of ankle equinus that frequency correlates directly with the duration of the diabetes [7]. Ankle equinus is strongly linked to ulcers of the forefoot in patients with neuropathies from diabetes.

Ataxic Gait

A disorder of the cerebellar system causes major difficulties in walking. Stepping is staggering with a tendency to lurch to one side. Grounding has a wide base and turning is erratic. While standing, the body tends to rock back and forth and side to side. Performing the tandem walk is impossible. Corroborating findings of cerebellar disease are dysmetria, dysarthria, nystagmus, and an "intention tremor."

The signs may reflect diseases of the cerebellum itself: neoplasm or degeneration from alcoholism or hyperthyroidism. They may be caused by a hemorrhage within the brain stem. More ordinary and reversible, however, is drinking alcohol to intoxication.

Myopathic Gait

Primary muscle disease tends to affect the proximal muscles of the limb disproportionally to the distal, not like neuropathic lesions. The dominant burden in the leg is weakness in the pelvic muscles. Compensatory lordosis is evident. To accomplish walking, the patient leans the trunk to one side away from the forward moving leg and then alternates to the other side. The result rocking motion is a characteristic "waddle," likened to that of a duck.

Myopathies can be produced by vascular diseases, neoplasia, or by neurologic, metabolic, or toxological diseases. Massive obesity and some orthopedic defects may simulate myopathy owing to the waddling gait.

Choreiform Gait

The irregular, jerky movements on walking are signs of disorders of the basal ganglia. They occur in various forms of chorea, athetosis, and dystonia.

Antalgic Gait

Here, the problem may appear as a neurologic defect when, actually, it is the response to a painful joint. The patient tries to avoid putting weight – or at least lessening the time of weight-bearing – on the hip or knee. Fear of the painful joint suddenly giving way creates an exaggeratedly cautious pattern of walking. In the extreme, a reflex action from sudden pain in the joint may lead to falling.

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Part VI

Male Standing, Female Supine

Genitourinary System

27

Examination of the genitourinary system is often relegated to the last portion of the physical examination (as it is in this text). This practice is understandably related to the social overtones and discomfort anticipated on a detailed examination of a private area. Nevertheless, in whatever sequence the general examination is conducted, careful attention to the genitourinary anatomy may reveal aspects of systemic conditions that might otherwise have been missed.

In perspective, the genital examination is an integral part of the general physical examination. Misadventures from oversight in this area are hardly rare. Missed diagnoses of active HPV infection or new-onset, thrombosis-generated varicocele or testicular mass are examples. Lesions on skin and abnormalities of various organs that can be traced to a genital infection are an ongoing challenge to the clinician. Retroperitoneal tumors from a metastatic testicular seminoma have been resected without the testicles being first palpated. Neurosurgical intervention has been performed on elderly men with acute paralysis before a documented rectal examination revealed advanced cancer of the prostate gland. Dysuria may be assumed "natural with aging" in a late, middle-aged woman when a bimanual examination may have discovered early cervical cancer. These untoward experiences, of course, have medical and medicolegal implications that might have been avoided had a carefully and methodically general physical examination included a genitourinary examination.

Some systemic syndromes that may present with genitourinary symptoms may be recognized by keen clinical acumen. They include diabetes (overactive bladder), cystic fibrosis (absent vas deferens), hepatic cirrhosis (feminization), androgenic hyperplasia (masculinization), Klinefelter syndrome (hypogonadism), and Turner's syndrome (anovulation).

The oft written term "deferred" for the genitourinary component of a hospital admission note adds little to the overall evaluation of a patient but rather reflects the intentions of the examiner, whether eventually fulfilled or not. These areas in a man or woman cannot be checked off as "normal" on physical examination unless the formal examination is performed utilizing standardized criteria.

When the clinician and the patient are of different genders, it is imperative that a second healthcare witness be present during the genitourinary examination. The name of this person must be documented in the record.

Explanation to the Patient

Assuming that the patient has not already undergone a genital examination, the clinician should introduce the procedure with logical, step-wise explanation. For those patients who are uncomfortable with the exam, some simple phrases are helpful: "It is important that our examination include the private parts area." "Because we are

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concerned about your …, we need to look at the genital area." "We need to make sure that …" "A nurse [or other attending] will do the examination with me …" Implied in these approaches is that the genital examination is a natural extension of the overall physical examination while instilling confidence by having a gender-similar healthcare professional in attendance. It is rare for a patient so informed to forego the genital part of the general physical examination.

The introduction is also an opportune moment to speak with a patient about pertinent aspects of sexual dysfunction, birth control, and guidelines for screening for cervical or prostate cancer. It may also be an appropriate time to discuss high-risk sexual behaviors, if relevant.

For minors, the inclusion of a parent may or may not improve the effectiveness of the genital examination. Either way, a clearly thought-out approach emphasizes the need to include at least a limited genital examination within the sociological, legal, and developmental context of the well- or the ill-child visit.

The genital evaluations as described here are general guidelines and reminders appropriate for a comprehensive survey examination. They are not intended as "complete."

Male

The clinician should find it advantageous for both efficiency and access to examine the genitourinary system of a male patient while he is standing. Thus, it can immediately follow the observations on gait just performed. Observations in the general examination – such as gynecomastia – may have direct correlation in the genital examination.

Perineum

Initially, the clinician assesses perineal hygiene, noting the presence of stains from hematuria, incontinence, urinary tract infections, or cutaneous disease. In addition the presence of trauma anywhere around the genital area must be documented in cases having a medical-legal nature.

The "Tanner staging criteria" for sexual maturation is documented in the youthful patient, especially if there is discordance between the patient's alleged age or gender and the physical findings. Tanner I is the prepubertal stage and Tanner V the fully mature stage regarding scrotum, testicles, and penis [1]. The pubic hair escutcheon in the adult male normally extends cephalad, pointing toward the navel.

Skin

Excoriations

Evidence of scratching in the pubic area should prompt a search for abrasions. The cause may be severe pruritis from obstruction of sweat glands and hair follicles by keratin plugs. Such lesions begin as a cluster of flesh-colored papules in the distribution of hair follicles. Scratching ruptures the lesions, causing a reactive inflammation to the secretory content which in turn intensifies the itching. Loss of hair and of sweating in the area are eventual sequelae. The condition is commonly known as the "Fox-Fordyce syndrome" [2]. As a complication of hair removal by laser, the diagnosis is frequently overlooked [3].

Scabies

The burrowing lesions of mites produce scabies, an intensely pruritic condition. Burrows on the penis appear as dark, curly lines ending in a whitish speck, the mite. Papules, vesicles, and pustules can erupt over the scrotum and on the shaft and glans of the penis.

Tinea Cruris

Skin infections of the scrotum by candida fungi have a characteristic bright red appearance with some degree of maceration. Distinctive of candida are satellite pustules that crop up just beyond the borders of the primary lesion.

"Jock itch" is a mycotic infection that is generally limited to the groin. Typically, the erythematous and

scaling lesion spreads with sharp leading edges while some clearing occurs at the center. The tinea does not affect the scrotum.

Inguinal Area

Herniation in the groin (the direct hernia) was covered in Chap. 20 when the patient is supine. Testing for this "direct" form of hernia during that part of the examination is an expedient when the groin is easily assessable. In general, however, detection of the three forms of inguinal hernia is most successful with the patient standing.

Inguinal Hernia

Herniation of bowel occurs where a weakness in the abdominal wall or scrotal canal exists. There are three potential areas in the lower abdomen: (1) the groin, (2) the femoral canal, and (3) the scrotum (Figs. 27.1, 27.2, and 27.3).

1. Groin. A direct inguinal hernia is a protrusion of bowel (or fat, bladder, urethra) into a weakened area of fascial sheath of the abdominal muscle. The hernia may be palpable as a bulge in the groin or as an impulse on straining by bearing down, coughing, or performing a Valsalva's maneuver. The incidence of a direct inguinal hernia increases with age and is common in elderly men.

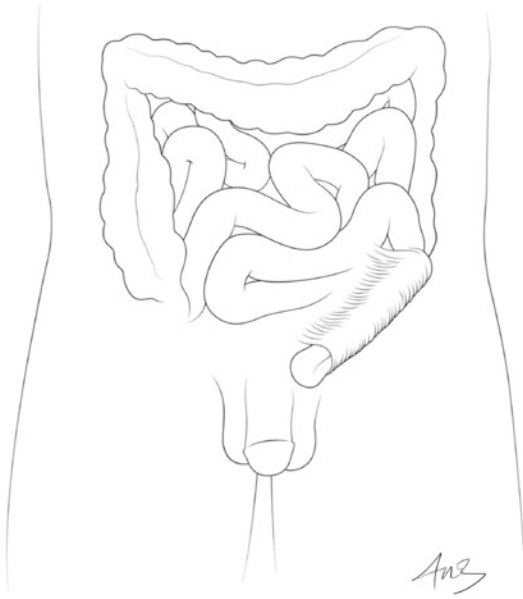

Fig. 27.1 Direct inguinal hernia

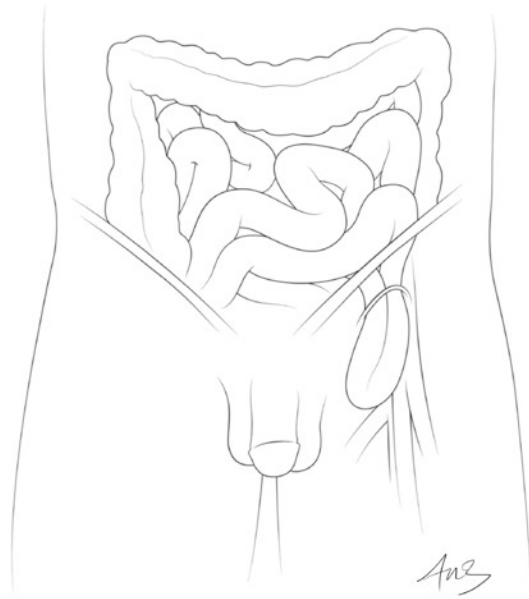

Fig. 27.2 Femoral hernia

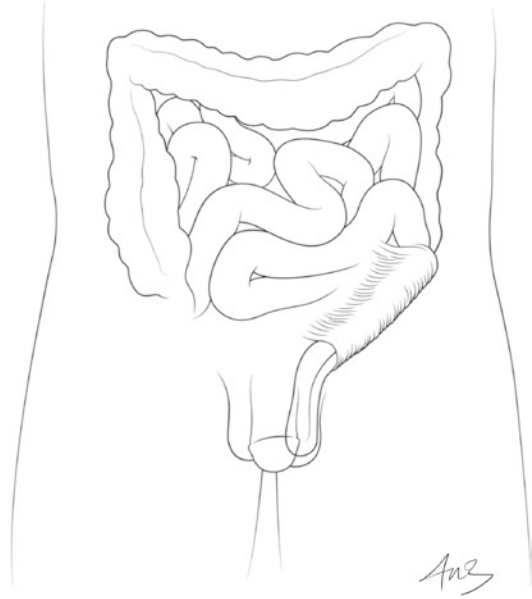

Fig. 27.3 Indirect (scrotal) hernia

A small, protruding kink of bowel in a direct hernia may simulate a lymph node with similar firmness and no discoloration. If incarcerated, its detection could be the key to the diagnosis of an acute abdominal syndrome.

2. A femoral hernia is a protrusion of a hernia sac distal to the inguinal ligament. Here, the femoral artery and vein emerge from the pelvis and pass into the leg, creating a relatively weak abdominal wall and a potential access for herniation of the bowel. This form of inguinal hernia may be more common in women than in men.
3. An indirect inguinal hernia allows passage of bowel (or other intra/retro-abdominal structures) through the inguinal canal as far as the scrotum. This form of hernia is described later in examination of the scrotum.

Phallus

Inspect the penis for the status of circumcision. The uncircumcised foreskin is retracted to inspect the glans, meatus, and corona. Always see that the foreskin is left in the reduced position. The uncircumcised phallus has an increased incidence of penile cancer.

Phimosis

Phimosis (Greek for "muzzle") is a constriction of the foreskin preventing the retraction over the glans. The genital examination must be considered incomplete if the glans is not inspected. When release of phimosis proves resistant to reduction, consultation with a urologist is appropriate.

Edema and circumferential entrapment can occur if phimotic skin retracted for examination of the glans is not reduced to its previous anatomical position. Denoted "paraphimosis", it is the condition frequently complicated by pain, excoriations, and bleeding of the involved skin and terminal phallus. Examination-induced paraphimosis is fraught with medical-legal ramifications. The liability is further increased if the complication follows a medical procedure such as placement of a Foley catheter.

Balanitis

Balanitis, an inflammation of the glans penis, is commonly associated with phimosis. In the uncircumcised male, it is usually a reflection of inadequate hygiene. In the circumcised, balanitis is so rare as to require detailed workup for contributing pathology.

Candida balanitis is common in diabetes, especially in those patients with urgency incontinence. The primary mechanism may be a form of diabetic bladder dysfunction with underlying neuropathy of the autonomic nervous system. It is uncommon for candida balanitis and an overactive bladder to be the presenting symptoms and signs of diabetes mellitus.

Balanitis may also be the mucocutaneous complication of "reactive arthritis" (formerly known as "Reiter syndrome"). The inflammatory signs appear circumferentially on the glans. Systemically, they may include conjunctivitis and arthritis. Without making the connection of the three conditions, the diagnosis could be easily overlooked in a young man with primary symptoms suggesting prostatitis [4]. The reactive arthritis syndrome with urologic involvement is being reported with increasing frequency following installation of bacillus Calmette-Guerin (BCG) in the urinary bladder as treatment for a bladder tumor [5].

Discharge

Any penile discharge is, of course, suspect for an infectious disorder. A meatal discharge in the sexually active man should be studied for gonococcal or chlamydial disease. The finding in turn calls for a search for other sexually transmitted diseases, specifically HIV and syphilis. A discharge may also represent a benign chronic disorder such as prostatitis.

Discoloration

Vitiligo is an autoimmune disease that may cause depigmentation of the glans, foreskin and shaft of the penis. While benign, it is often associated with atrophic diseases of the thyroid and adrenal glands and of the islet cells of the pancreas.

Lichen sclerosus (Gr. *moss* + *hard*) is an atrophic condition of skin that has a predilection for the genital area, especially for the tip of the penis. Most notable are white spots and patches where the skin is thin and wrinkled, usually on the glans and prepuce. Keratotic plugs that are black with erythematous halos may be present. The lesions can be extremely itchy. Lesions heal into firm and depigmented scars.

Stenosis of the urethral meatus as a possible complication of lichen sclerosus is possible. Known as "balanitis xerotica obliterans" in men, it usually presents with urethritis with or without a urethral discharge.

Ulcer

An ulcer from primary syphilis is typically superficial and round. The chancre has a fairly clean surface with an indurated rim. Notably, the lesion is not painful unless complicated by secondary infection when weeping vesicles and regional adenopathy are likely to be present.

The specialty of urology, incidentally, was originally called "syphilology," recognizing the prevalent genital manifestations of a systemic disease. Put another way, the multiple expressions of syphilis could be considered systemic manifestations of a genital disease. The diagnosis was a major concern among physicians in the pre-antibiotic ages

The ulcer of chancroid differs from that of primary syphilis by being painful. Caused by *Haemophilus ducreyi*, the chancroid ulcer brings to mind the mnemonic "You do cry with H. ducreyi." In addition to being painful, the chancroid ulcer has a necrotic surface, is deeper, and has undermined edges – all helpful signs in distinguishing chancroid from chancre. Inguinal lymphadenopathy occurs in both conditions.

A typical ulcer in secondary syphilis is condyloma latum (Gr. *knuckle* + *flat*; *pl. condylomata lata*). Appearing as reddish-brown, warty plaques, the lesions tend to occur in groups most especially in the skin folds of the anogenital area. They have a broad flattened surface. On eroding, they present a weeping surface. The plaques may congregate and form a mushroom-shaped dome. These lesions, swarming with spirochetes, are highly infectious.

Condyloma acuminatum (known as "venereal warts") presents in small groups of papillomas in the perineal area, particularly around the anus. They may aggregate to form a cauliflower-like mass. Human papillomavirus is the inciting cause; the potential for malignant changes occurs with HPV genotype 8, 16, and 18.

The physical examination of the uncircumcised phallus must include documentation of the underlying glans to rule out cancer. Penile cancer usually is associated with chronic irritation in the uncircumcised phallus. It may begin as an erythematous patch of friable tissue of the glans or foreskin. A bump will eventually appear that may be denuded, bleeding, and sore. Some forms of penile cancer include papillary or wart-like lesions. Nearly all cancers of the penis are squamous cell carcinomas.

Scrotum

The scrotum has redundant skin which may be in different states of contraction at the time of the examination. A cold room, an anxious patient, or uncomfortable bedding may induce brisk contractions of the cremasteric and dartos muscles, making palpation of the testes and adnexa difficult. Optimally, the examination is performed on a patient who is warm and relaxed.

Inspection of the normal scrotum will reveal fullness without bulging bilaterally. Any exception warrants further investigation, especially to recognize a missing or an enlarged testicle.

Ulcer

Finding an ulcer on the scrotal surface should bring to mind the classic triad of Behcet's vasculitis. Aphthous sores of the mouth and uveitis make up the other immune-mediated components. The lesions are painful and tend to heal on the scrotum with scarring and frequent recurrences. The ulcers may appear on the shaft of the penis as well as on the scrotum.

Hereditary Hemorrhagic Telangiectasia

A strong clue to this rare and multi-organ disease is the finding of multiple vascular tufts on the scrotal surface. Telangiectasias are also common on the mucocutaneous surfaces of the mouth and gastrointestinal tract. Recurring epistaxis, hemoptysis, and neurological mini-strokes from diffuse arteriovenous malformations are frequent complications.

Varicocele

A varicocele is a tangle of varicose veins in the scrotum that – when very large – feels like a "bag of worms." Reflux in the more proximal antigravitation valves is responsible. A furrowed outline of the distended veins on the scrotum may be obvious (Fig. 27.4).

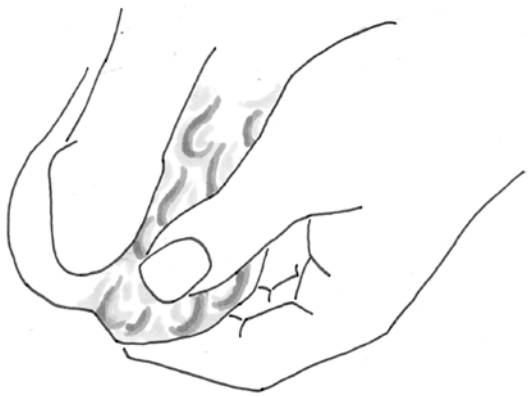

Fig. 27.4 Varicocele

When bilateral, varicoceles are almost always benign and without symptoms. The clinician must ascertain, however, that there is a no secondary testicular atrophy. Infertility, although decidedly unusual, can be a complication of bilateral varicoceles.

A unilateral varicocele has a different implication. An anatomical variant accounts for it being far more common on the left. The right gonadal vein empties into the inferior vena cava. The left gonadal vein enters the left renal vein and is much longer. The lower aspect of the vein is thus subjected to greater gravitational force than the right when the individual is standing. This difference favors thrombo-occlusive disease of the gonadal vein on the left.

Venous Thrombosis

The left gonadal vein is also more likely to be thrombosed by a renal mass on that side. Thus, finding a left varicocele that has developed within a few months is highly suspect for a renal cell carcinoma and prompts further investigation. While a new right-sided varicocele has the same implication, it is far less common.

Edema

In anasarca, total enlargement of the scrotum occurs in which tenting and thinning of the scrotal skin is evident. The cutaneous edema is contiguous in the dependent tissues of the lower extremities. Of course, the edema of anasarca will be evident throughout the body, including the eyelids.

Diffuse scrotal edema not due to anasarca should prompt investigation for pelvic pathology while ruling out intrascrotal lesions. Edema that is readily dissipated by gentle manual compression is more likely to be related to transudation of fluid from a volume overload. Lymphatic obstruction produces a tense, difficult to compress scrotal edema that can have a malignant origin or be from a chronic infection, most notably filariasis.

Hydrocele

Enlargement of the scrotum by a hydrocele, on the other hand, is usually a benign accumulation of fluid on one side that displaces the testis medially. A hydrocele can be bilateral and occupy virtually the entire scrotum, producing scrotal tension. They vary in size from marginally detectable to massive (Fig. 27.5).

A hydrocele is derived from the tunica vaginalis, a membrane that covers the lateral aspect of the gonad as it descends from the retroperitoneum to the scrotum during fetal life. The "communicating hydrocele" occurs virtually only in childhood. It tends to expand when the subject stands and shrinks when lying down. The "noncommunicating hydrocele" will maintain its enlarged size despite changes in body position. In both instances, the fluid sac closely encapsulates the testicle. A large cystic structure that feels separate from the testicle is more likely to be a spermatocele or a cyst of the epididymis. A lipoma of the spermatic cord is less common than the hydrocele.

A hydrocele will not create a palpable impulse in response to a cough. This finding helps differentiate the mass from bowel protruding into the scrotal sac. Here, with an indirect hernia, an impulse felt on coughing is a diagnostic sign.

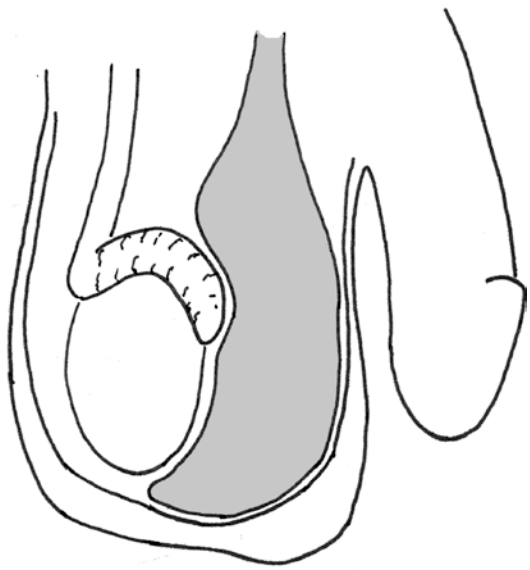

Fig. 27.5 Hydrocele

Transillumination with a penlight will help differentiate a hydrocele from a solid tumor or hematocele. The light must be pressed against the skin in a room that has been darkened. Light diffuses through a hydrocele readily; in contrast, a solid mass, a hernia, a hemorrhagic cyst, and a spermatocele will be relatively opaque.

Scrotal Hernia

An indirect hernia may produce unilateral enlargement of the scrotum. It will be associated with fullness in the internal ring. It is this deep ring leading to the inguinal canal that normally becomes obliterated at the time of birth after the testes have descended. The canal contains the vas deferens and the blood vessel that make up the spermatic cord. When the inguinal canal fails to close, there is a potential tract for segments of bowel to protrude into the scrotum.

Evaluation for the indirect hernia is not wholly reliable unless the patient is standing. To check for a "sliding" hernia, invaginate the scrotum with the fifth finger and introduce the digit into the superficial inguinal ring, the terminal point of this canal. Normally, the ring is just wide enough to allow the fifth finger. A wider ring, however, does not mean that a hernia is present. Have the patient cough. A tap on the tip of the examining finger indicates that the sudden increase in intra-abdominal pressure forces a kink of the bowel to slide through the inguinal ring. As noted above, this response to a cough does not occur with a hydrocele.

Bowel sounds heard within the scrotum signals definitive evidence of intestinal hernia extending through the inguinal canal. An attempt to reduce the hernia manually beyond gentle compression is not generally advisable. The incidence of indirect inguinal hernia increases with age. It can also be hereditary and is often bilateral because the internal ring fails to close.

This hernia is the "rupture" known throughout the ages. It was the impetus for inventing a myriad of "hernia belts" to contain the scrotal bulge. The old literature is replete with descriptions of massive hernias and ingenious devices to contain them.

A large mass in the scrotum that is easily kneaded is most likely a segment of herniated bowel. The impression is confirmed by hearing bowel sounds with the stethoscope. If the patient lies down, the hernia content may be completely withdrawn spontaneously into the abdomen. Reduction can be enhanced by gentle pressure, a technique that the patient may already have been practicing. If the bowel does not easily slide entirely back into the abdominal cavity, the attempt should be abandoned.

If the segment of bowel contained within the scrotum cannot be reduced, it is called "incarcerated." The "strangulated" hernia refers to bowel that has had its blood supply compromised. This condition, in which tenderness, nausea, and vomiting are prominent, is a surgical emergency.

An indirect hernia in the scrotum can simulate a hydrocele. Unlike bowel protruded into the sac, a hydrocele will not move on coughing. A hernia is typically superior to the testis while the hydrocele is almost always lateral to the testis. When differentiation is questionable, transillumination may reveal greater transmission of light in the hydrocele than in the protruding bowel.

Testes

Examination of the testes is critical in any general physical examination regardless of age. Men seldom self-examine and a testicular tumor can be remarkably large before a patient calls attention to the matter.

In general, the testes reside within the scrotum although they can be withdrawn into the inguinal canal by a brisk cremasteric reflex. This temporary relocation is practically limited to younger and anxious individuals in a cool environment. A common "cause" of the undescended testicle is an examiner with cold hands.

The left testis is slightly lower than the right when standing. The right testis that will be lower is one exception: situs inversus. Identifying this rare condition, described in Chap. 20, in this way could prove a godsend in the patient's lifelong medical and surgical care.

Size

Testicular size is measured in cubic millimeters. The size of the normal testicle is similar to a pigeon's egg. The ancient Greeks likened the human gonad to an olive (Gr. *orchidos*). A pragmatic estimate of size should be made if the testes appear large or small; formal measurement with an orchidometer is left for the specialist. There is little change of size with aging aside from acquired hypogonadism, either primary or secondary.

Orchitis

Orchitis is the usual cause of testicular enlargement. The predominant cause is mumps. In the majority of cases, the orchitis is unilateral, and the testicle is tender. Other infections producing enlarged testes are secondary syphilis and tuberculosis; in neither case are they likely to be tender.

Neoplasm

The most common neoplasm of a solid organ in men 20–35 years of age is testicular cancer. The enlarged testis is often painless and non-tender unless complicated by hemorrhage. Most characteristic is the rock-hardness of the tumor. Virtually all are diffusely enlarged and unilateral, stemming from germ cell anomalies. If testicular enlargement from a neoplasm is bilateral, the most probable diagnosis is lymphoma (Fig. 27.6).

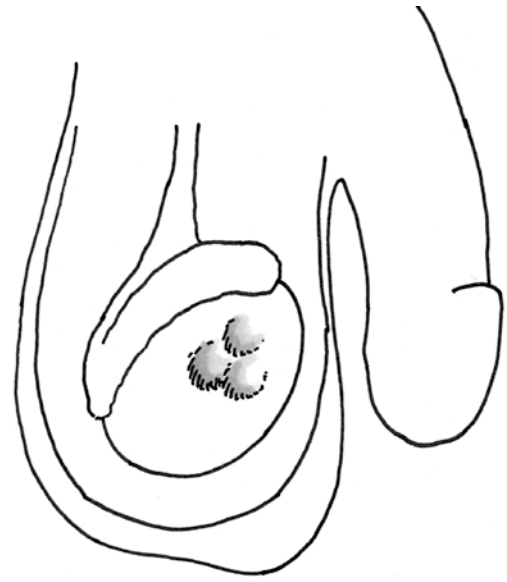

Fig. 27.6 Neoplasm of testes

Atrophy

An atrophic testicle is less than half the size of its counterpart. A reminder of an obvious point of confusion is an enlarged testis that gives the false impression that the contralateral testes is atrophic.

Bilateral testicular atrophy is associated most commonly with primary hypogonadism, abuse or overuse of testosterone drugs, and the anti-prostatic cancer drugs such as GnRH agonists and antagonists. It is emphasized that the clinician appreciate that normal-sized testicles do not rule out either primary or secondary hypogonadism.

Klinefelter syndrome, clinically apparent after puberty, is a rare congenital defect associated with present but small testes. Evidence of hypogonadism includes gynecomastia and sparse body hair that takes on a female pubic distribution.

Undescended Testicle

The inability to palpate a testis indicates that it has either not descended into the scrotum or it may have not developed. In cryptorchidism, the testis is present but resides at the internal ring or within the abdomen deep to the ring.

Unilateral cryptorchidism often goes undiagnosed into adulthood. A high-riding, palpable testis within the scrotum may be normal with rates of neoplasm equal to the descended side. A non-palpable testis within the inguinal canal represents true cryptorchidism and an increased risk of neoplasm. Requiring further urologic evaluation is a non-palpable or atrophic testis or a new-onset retractile testis (Fig. 27.7).

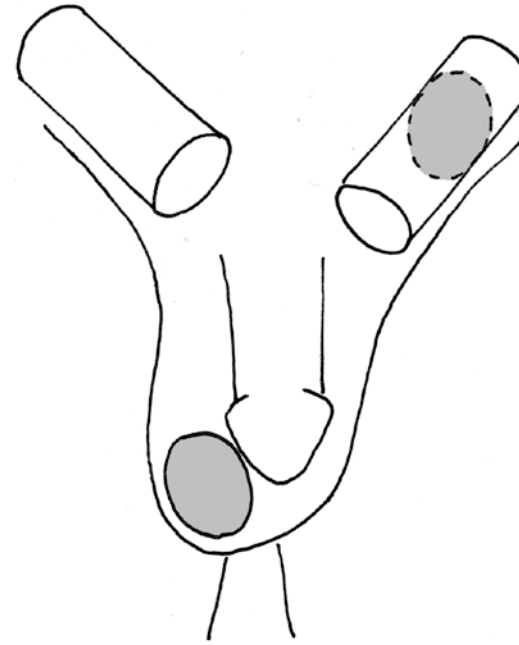

Fig. 27.7 High-riding testes

Texture

The normal testicle is firm but somewhat compressible, likened to the texture of a hard-boiled egg. To palpate, support the testis in the palm of one hand and gently compress it with the forefinger of the examining hand.

A hard, non-tender mass is highly suspect for a testicular tumor even if the testicle is not enlarged. Testicular cancer is described in the previous section. When suspected, the testicular mass must be promptly and fully evaluated – as the urologists say – "before the sun sets."

Pain/Tenderness

Testicular Torsion

The loosely suspended testicles can rotate on the spermatic cord, compromising the arterial supply. Those most likely to develop testicular torsion have congenitally lax suspension of the testicles, referred to as the "bell-clapper defect" (Fig. 27.8).

A high-riding testis of new onset associated with acute pain over the inguinal canal may be a sign of intravaginal testicular torsion. Ischemic damage occurs within hours, a testicle twisted around is truly a urological emergency.

The torsion causes severe pain that is worsened by elevation of the scrotum, further strangulating the vascular supply. In epididymitis, by contrast, pain is alleviated by raising the supported scrotum (known as Prehn's sign) [6].

Spontaneous detorsion is known to occur. The urgency for definitive management is more pressing when the torsion is painful. Palpation will reveal asymmetry of the scrotal contents and a tender, visibly withdrawn testis that lies higher into the inguinal canal. The so-called high-riding testis that is painful is a sign of torsion of the spermatic cord and the testicle.

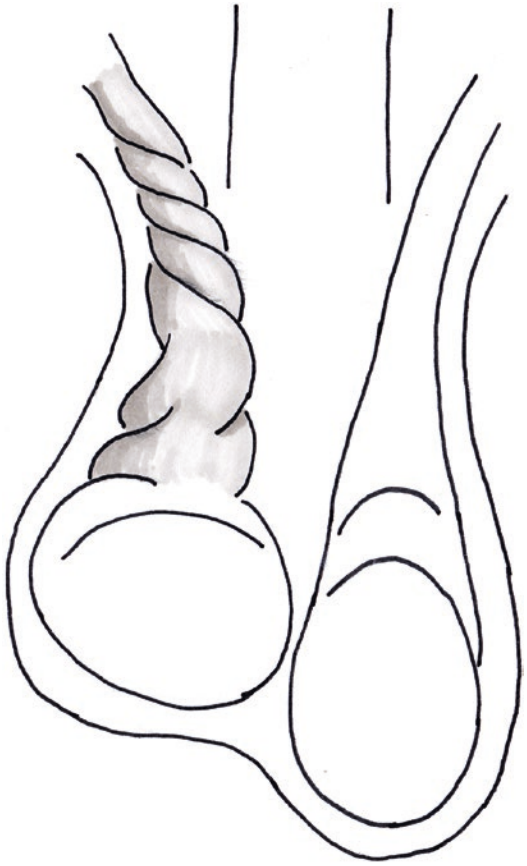

Fig. 27.8 Testicular torsion

Clinicians confronted in the emergency situation of spermatic cord torsion may attempt reduction manually, using repetitive medial to lateral motions "like opening a book." There are anecdotal successes, and this method may relieve pain and ischemia in short order. The effort, however, can quickly escalate into complications. Recommended is prompt detorsion by a urologic specialist.

Reference has been made of the epidemiology of testicular disease. The murky but interesting etymology of the word "testicle" deserves some mention. The word *testes* came from the number "three" in the Indo-European language. It came to mean "witness" in Latin, referring to a third person who would witness a solemn declaration between two others; that is, a person to make a testament to the agreement. Possibly the connection was made between the original meaning and the notion that the testicle was assurance of the virility of the witness. In another version, only those with testicles could testify in court, thus prohibiting the testimony of women.

Adnexa

The spermatic cord is a complex bundle containing the vas deferens, the blood supply to the testes, lymphatics, and nerves (sympathetic, parasympathetic, and somatic to cremasteric muscle). It is bound by a tough, multilayered fascia. After formation, sperm is transported from epididymis through the vas deferens to the prostate gland and from there to the phallus.

Epididymis

The epididymis, as the name implies, overlies the testis. (The suffix "-*didymis*" is from Greek, meaning twin or double.) It is palpated along the posterior vascular axis. The texture of the normal epididymis feels like a knotted strand of spaghetti. The epididymis can be isolated manually by shifting the fingers posteriorly from the testis. The head of the epididymis sits at the upper pole of the testes, its body descending along the lateral margin and its tail at the lower pole where it joins with the vas deferens (Fig. 27.9).

Epididymitis

In epididymitis, the epididymis but not the testis will be tender. The pain from epididymitis may be relieved on elevation of the scrotum, in contradistinction to the pain of testicular torsion in which elevation increases pain. Fever as well as discomfort on voiding are expected with or without urethritis (Fig. 27.10).

Causes of acute inflammation of the epididymis include a voiding dysfunction, a viral infection, or direct trauma. Amiodarone has also been implicated in the etiology of epididymitis [7].

Vas Deferens

The vas deferens is next palpated. The vas (also known as the "ductus") arises from the tail of the

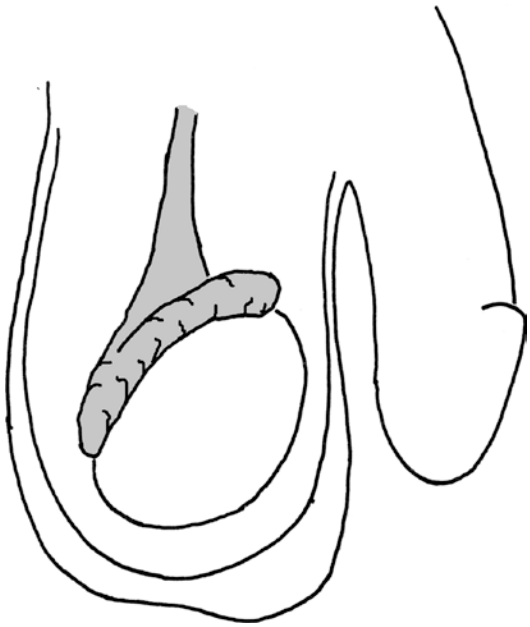

Fig. 27.9 Epididymis

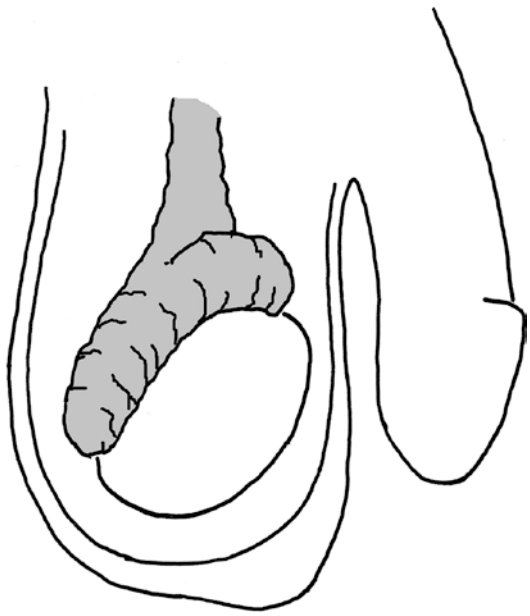

Fig. 27.10 Epididymitis

epididymis before joining the spermatic cord. It is a rubbery firm and freely mobile tubular structure that is clearly separated from adjacent structures. It can be manually rolled away from the spermatic cord so that it is poised just below the surface of the skin. This easy separation facilitates vasectomy (Fig. 27.11).

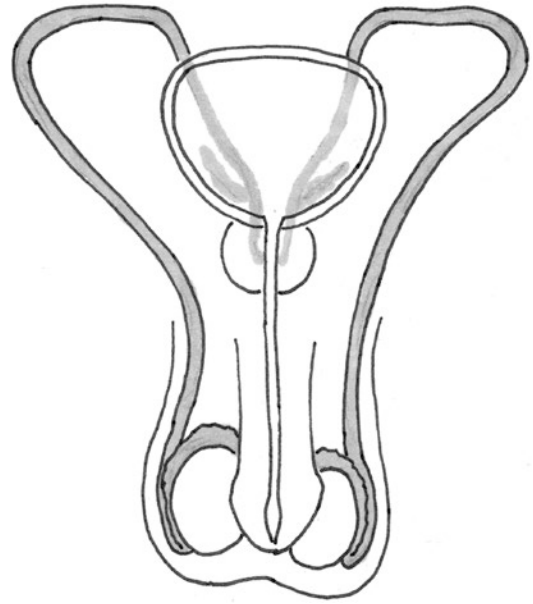

Fig. 27.11 Vas deferens

Lack of a palpable vas deferens should raise suspicion that there is no kidney on that side. If the vas deferens is absent bilaterally, the diagnosis of at least subclinical cystic fibrosis is likely [8]. Nearly all males with cystic fibrosis have a congenital absence of the vas deferens and produce no spermatozoa. Thus, a young male patient with asthma or respiratory compromise should undergo a genital examination in addition to the pulmonary evaluation and definite diagnostic testing.

Extra-testicular Mass

A mass around the adnexa of a testis that is mobile is probably benign. The most common adnexal mass found on physical examination is the lipoma. A lipoma of the spermatic cord does create a palpable impulse in response to a cough. Other possibilities are a spermatocele or an epididymal cyst.

When a solid mass is fixed to the tissue surrounding the testicle, suspicion of malignancy is raised, a finding that deserves further investigation without delay. Most common of the adnexal neoplasms is rhabdosarcoma [9].

Rectum

The rectal vault and prostate examination is performed taking advantage of the patient standing, now leaning over with elbows on the bed or examining table. Alternatively, many clinicians, including urologists, prefer to perform the rectal examination with the patient lying on one side with knees drawn up. The legs are covered with a warm sheet, and the upper body clothed or covered as well.

Anal fissures, especially active ones, will lie laterally along the anal verge. These can be tender. External hemorrhoids never occlude the rectal os and should not preclude the digital examination. If intestinal obstruction is present, the ampule of the rectum may have ballooned out. In peritonitis, the rectum is usually somewhat tender, even when pain is mainly experienced in the mid- or upper abdomen.

The digital rectal examination (DRE) can almost always be performed without any patient discomfort. As it is typically the last part of a general physical examination, the clinician must guard against the natural tendency of conducting the DRE in a hurried and perfunctory manner, thereby causing more discomfort than need be.

Discomfort from a digital examination of the rectum is largely from a rapid introduction of the lubricated, gloved finger. For easing entry, the index finger is placed slowly on the anal verge and pressure from side to side is then gently applied. So approached, the natural spasm of the sphincter will release within a few seconds; thereafter the finger is slowly advanced into the rectal vault. Should a second wave of spasm of the anal sphincter occur, the inserted finger can be paused until the spasm subsides. Further advance will now not require any additional sphincteric dilation and will allow for palpation of the deeper rectal structures.

Appendicitis may be suspected when the tip of the examining finger is directed toward the right lower abdominal area and causes pain. The rectal examination is an expected procedure when the diagnosis of appendicitis is considered.

Prostate

The prostate gland should be evaluated by rectal digital examination in every comprehensive examination of a man of 50 years or older. It is appropriate to offer it to a younger adult male with any of the following: a voiding disorder, a close relative with prostate cancer, a recent history of hematuria, or pelvic trauma such as from a deceleration injury.

Position

The normal prostate gland lies anterior to the rectum, about 6 cm from the anal verge. In some morbidly obese men, it may extend farther in; the standing patient position in these corpulent individuals is decidedly a disadvantage (Fig. 27.12).

Texture

The tip of the examining finger reveals the normal prostate gland to be smooth and firm but slightly compressible. Each lateral lobe of the prostate should have a “rubbery” firmness that is symmetrical. The texture is likened to that of the hand’s muscular thenar eminence, not the hardness of the thenar joint.

A hard nodule palpated on the prostate must be documented for size and location. While prostatic

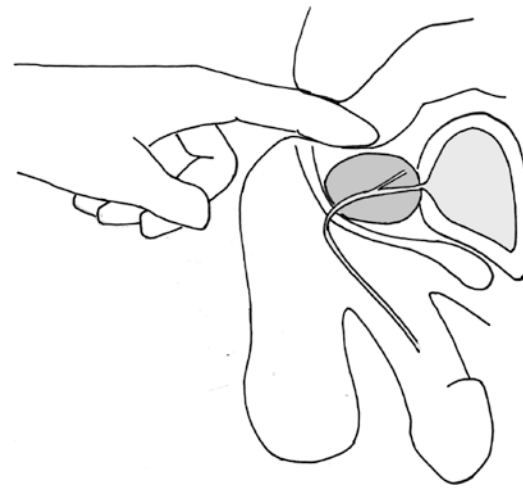

Fig. 27.12 Prostate examination

nodules are benign in nearly 50% of cases, the finding merits further investigation. If the tissue sampling of a palpable prostatic nodule proves neoplastic by histologic examination, it is at least a stage II cancer. Any sense of nodularity or induration of the prostate is a red flag that signals a screening for cancer in the workup.

There is no established correlation between the impression of the prostate on digital examination and the Gleason system of grading by histological features of neoplasm.

Size

The normal prostate gland is 3–4 cm wide (about one fingerbreadth on each side of midline) and is about as long as a fingerbreadth. Measurement is best documented for general clinical purposes by estimating such metrics rather than the commonly used by highly variable estimation of 1+ to 4+ for enlargement.

Reports vary on the ability to estimate the volume of the prostate gland on digital exam, but some studies support their accuracy [10]. Correlation of prostatic enlargement with the degree of urinary retention and frequency of nocturia is more limited [11]. Urinary outflow is affected by multiple factors including enlargement of the median lobe of the prostate gland, not the posterior lobe that is palpated on digital examination.

Tenderness

Palpation of the normal prostate gland will not elicit tenderness. Instead, the procedure may induce a feeling of urinary urgency or cause dribbling. Even slight tenderness from the touch of a fingertip indicates an inflammatory prostatitis. Causal bacterial infection can be of recent origin or of a recurring nature.

Acute bacterial prostatitis is characterized by distress on voiding with potential urinary obstruction, fever, and marked tenderness of the prostate gland. The impression is redoubled in a man who is febrile and who also has back pain. The clinician is urged to palpate the tender prostate lightly to minimize shedding of bacteria with resulting septicemia. Cancer of the prostate, it is noted parenthetically but with emphasis, is almost never tender.

Chronic prostatitis, on the other hand, is of uncertain etiology. It can be entirely asymptomatic or may be associated with difficulty in urination and ejaculation [12].

Mobility

There is a slight mobility of the prostate anteriorly and laterally. Should the gland seem fixed to the sidewall of the pelvis, further evaluation for a possible malignancy is indicated.

The 1-Minute Genitourinary Examination in Men

There are several components of the external male anatomy that can be evaluated individually with practiced carefulness and speed. Of course, identifying or being suspicious of one abnormality means a more detailed exploration. This express examination does not include the digital rectal examination for prostate size and texture, a test that takes a few minutes. Specific issues for the mental checklist, itemized in probable sequence, include:

Maturation (mismatch: real v. apparent)
Perineal skin (dermatophytosis, edema)
Inguinal ring (Indirect hernia)
Foreskin (phimosis/paraphimosis)
Glans (balanitis)
Phallus (venereal ulcer/discharge)
Testicles (orchitis, atrophy, mass)
Epididymis (inflammation)
Vas deference (absence)
Veins (varicosity)
Inguinal canal (indirect hernia)

Female

In the routine physical examination, examination of the female genitourinary system is often deferred for convenience to another time or to a specialist. Yet, it is part of the general physical examination and is all the more pertinent when

any symptoms are present that suggest an abdominal or pelvic origin. "Pelvic exam deferred," so often written in hospital charts, always poses the risk of not getting done.

Sexual maturity has been defined by the clinical "Tanner staging criteria." These guidelines are especially useful if there is discordance between the patient's stated age or gender and the physical findings. Discerning glandular breast tissue from fatty tissue in obese girls can be difficult by palpation. According to the criteria for breast development, Tanner I indicates no glandular tissue; the fully mature breast is Tanner V in which the areola is flush and the papilla is projecting [13].

The distribution of pubic hair in the fully matured female is horizontal at the upper edge. If the pattern resembles the male escutcheon, pointing cephalad, look for other signs of androgenicity. Signs of virilization include hirsutism, male-pattern baldness, acne, masculine voice and muscular development, mammary atrophy, and menstrual irregularities.

The pelvic examination – like the female breast examination – is not amenable to the "survey" type of physical examination. A limited urogenital examination can be performed, however, when the patient does not have symptoms of genitourinary disorders and when it is performed carefully and methodically as part of a comprehensive physical workup. Such is the approach outlined here.

As the clinician proceeds with the examination, the patient may be set at ease by explaining each step along the way and by incorporating a vocabulary of using "we" rather than "I." For example, "next, we will take a look at …" In this way, the patient, in a reassuring sense, participates in the procedure.

A useful evaluation of the female genitalia can be accomplished without the stirrup lithotomy position; a modified "frog-leg" position can be quite adequate for the asymptomatic subject having no risk factors for gynecologic disorders. The importance of a warm room, with a well-lighted field and – if the examiner is male – with a female chaperone cannot be overemphasized.

Habitus

Virilization

Masculine characteristic in a woman include receding hair in the forehead, facial hair (not including vellus hair), deepened voice, male muscular development, and acne. These signs can represent an androgenic tumor of the adrenal cortex.

Turner's Syndrome

The physical signs of Turner's syndrome should be recognized in the patient with "delayed adolescence." These are short stature, webbed neck, puffy hands and feet with shortened digits, hypertension, and cardiac and renal abnormalities. Late diagnosis of Turner's syndrome, unfortunately, misses the optimal opportunity to address the issues of growth and secondary sex characteristics [14].

Perineum

In addition to signs of masculinization, look for issues of hygiene, noting soiling or stains from hematuria, incontinence, urinary tract infections or cutaneous disease. The presence of trauma anywhere near the genital area (abrasions or contusions) must be documented as these have medical-legal implications. Evaluation for cases of recent rape must follow a strict protocol which is beyond the scope of this text.

Intertriginous areas are inspected for tinea infections. These areas are particularly susceptible to fungal infections in the diabetic and in the very obese who have fold-over adipose tissue at the groin.

Evidence of intense scratching in the perineal area may be initiated by keratin plugging of hair follicles. The initial lesions are papules developing over areas that are warm, humid, and subject to repeated friction. The blocked sweat glands and hair follicles that are torn open by scratching cause their content to create an inflammatory area that is intensely pruritic. The condition is far more common in women than in men, particularly in those of middle age.

Of the inguinal hernias, women are most susceptible to the femoral hernia. It is recognized as a bulge just distal to the inguinal ligament or within or neighbor to the labia majora. Because of the high risk of incarceration and strangulation of bowel, repair of these hernias in girls is recommended once identified.

Vulva

By vulva is meant the region of the external genitalia. Included are the labium, the clitoris, the vaginal vestibule, and the urethra orifice.

Genitalia

The genitalia are inspected for the stage of sexual maturation, to include the labia majora and minora, the clitoris, and urethral orifice. Look for evidence of trauma, lesions, warts, blood, or soiling. Prolapse of the uterus, rectum, or bladder must be identified. In the elderly, eversion of the uterus can be severe enough to cause it to protrude from the vagina (a condition called "procidentia"). Uterine prolapse, if present, should be reduced as the digital examination begins.

With the labia gently separated with one hand, the lubricated, gloved index and middle fingers of the examining hand are placed vertically at the introitus and slowly rotated to horizontal with the palms facing down. Then the clinician advances gently to gradually enter the vaginal canal. This procedure engenders little discomfort or spasm in all but the very exception.

The hand is then supinated to palpate the urethra and anterior vaginal wall, while gentle pressure is applied over the suprapubic region with the other hand. Vaginal masses are documented. The examining fingers are then advanced to the fornix of the vagina and the cul-de-sac. Tenderness of the cul-de-sac is abnormal and suggests occult cervicitis, such as caused by infection with *Chlamydia*.

A tender mass along the anterior vaginal wall (and thus posterior to the urethra) is suspect for ureteral diverticulum. The cervix must be palpated in any patient who has a uterus. Here, the bimanual approach is essential. Cervical cancer will be non-tender but has a hard consistency, prompting immediate gynecologic follow-up. Blood in the menopausal woman must be followed by further investigation, to include a Pap smear. After hysterectomy, the cul-de-sac will have a blind ending that must be documented.

Spots, Bumps, and Patches

A painful, cystic lump in the labia minora is usually an infection of the Bartholin gland. The infectious agent is usually *Chlamydia*. Further evaluation for alternate diagnoses is indicated if the labial finding is unilateral or if there is associated regional lymphadenopathy.

The tiny papillae of condylomata acuminata usually appear as aggregations and can occur anywhere in the vulva. These are "venereal warts" that may consolidate to produce a verrucous, horny mass. The infectious agent is the human papillomavirus. A single genital wart increases the likelihood of cervical cancer fivefold. It should also prompt a workup for occult chlamydial infection and all other STDs.

Discoloration

Spots and patches of blanched skin that is thinned and wrinkled are probably those of lichen sclerosus (also called "kraurosis vulvae"). The disease tends to affect small areas of the perineum, the labia minora, and the clitoral head. The condition, which is extremely itchy, affects women in much greater frequency than perineal disease in men.

Ulcer

Condylomata lata, an expression of secondary syphilis, consists of broad reddish-brown ulcers that are flat and clean (that is, they have little overlying necrotic tissue). They tend to appear in groups along intertriginous folds of the anal-genital area. These warty plaques are swarming with spirochetes.

Vesicular eruptions from herpes zoster can occur in the vulva. The woman having an immunocompromised state for whatever reason is the most susceptible.

The clinician can appreciate that there is no female equivalent to the "1-minute genitourinary examination" proposed for men. Yet, the limited approach to the pelvic examination outlined here, when well organized and practiced, can be expedient for the general physical examination until a timely, comprehensive gynecological examination can be performed.

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Part VII

The Diabetic Foot

Appendix: The Clinician's Guide to Examination of the Diabetic Foot

28

This section focuses on one clinical entity, a condition that is common and challenging in the everyday practice of medicine. While the "diabetic foot" is a complication of a specific diagnosis, it is such a common problem that challenges clinicians with its diverse presentations. The guidelines presented here are meant to provide a comprehensive approach to the evaluation of the diabetic foot at the bedside that can be accomplished rapidly and with a reasonable degree of accuracy. Supplemental information includes the reasons, in brief, for performing these various tests. So that this addendum can be used as standalone guidelines, there will be some repetition from the previous chapter.

To control hyperglycemia carefully, the patient with diabetes must confront a wide assortment of challenges. The diagnosis dictates a daily adherence to a prescribed diet, to physical activities, and to medications while managing coexisting conditions optimally. The permanency of the diagnosis, the persistence of some symptoms (such as paresthesias), and the increased susceptibility to trauma and infection are inherently discouraging. Often, the most troubling of the complications involves the foot. When these occur, for example, an ulcer, there is often a sense of hopelessness along with a crushing disability and the frustration of slow healing.

Even so, the modern clinician has at his or her fingertips a deeply stacked armamentarium for treating diabetes. The patient and caregiver can derive some solace and motivation in knowing that fastidious, long-term control of hyperglycemia may prevent or at least minimize the foot complications that are the subject of this article. A basis for this confidence depends, in part, on the scrupulous attention the clinician can devote to the periodic examination.

These guidelines are meant to facilitate the "hands-on eyeball" examination of the foot of diabetic patients and of those with impaired glucose tolerance. This assessment will not be replaced solely by imaging techniques within the foreseeable future. Certainly, identifying the precise cause of complications early is necessary for implementing effective care. In addition, well-documented data indicating stability of findings and recorded over time can serve as profound encouragement for the patient to maintain good glucose control; where there is a lack of good control, the periodic evaluation that may demonstrate advancing complications will serve as a blinking red light. The findings can be recorded conveniently on a "CHECK-OFF LIST" provided at the end of this section. It is recommended that the comprehensive physical examination be conducted at least once a year on all patients with diabetes.

What Is the "Diabetic Foot"?

The term "diabetic foot" as a specific diagnosis deserves rethinking. It is not an etiologic entity. Rather the condition is often, if not usually,

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a constellation of pathological issues. Put in perspective, "The magnitude of the clinical picture and morbidity [of the diabetic foot] mirrors the severity and complexity of the underlying pathology [1]."

The term "diabetic foot" brings to the mind of many clinicians a singular abnormality. By widespread opinion, the "diabetic foot" is believed to be the result of neurogenic insensitivity to pain, in turn leading to unawareness of minor injuries (such as may be caused "by a pebble in the shoe"). The consequence, ultimately, is a foot ulcer.

It is true that persons with any of the various complications of diabetes will likely have some degree of sensory neuropathy. It is also true that a lack of sensation in the foot can result in tissue injury. Yet, considering the "diabetic foot" to be just a sensory deficit may lull the clinician into overlooking other important conditions that are the basis of or contribute to problems of the feet.

Surely, there are many people with diabetes who have problems with their feet. And there are many of causes of the problems. Often two or more causes are present in the same person – and in the same foot. It is the responsibility of the clinician to sort out the causes and to direct management accordingly. A careful examination may, in addition, disclose problems unrelated to diabetes that could mimic those often attributed directly to diabetes. In practice, identifying all causes and applying measures to lessen their impact on the foot are effective in greatly reducing limb-threatening complications.

The Diabetic Foot in a Nutshell

The incidence of complications of the foot in Type 1 and Type 2 diabetes is about equal. These complications lead to more hospital admissions than all the other complications of diabetes combined. One person in eight attending a clinic specializing in diabetes has a major foot complication [2]. The incidence is much higher if the patient has hypertension or peripheral neuropathies or if the diabetes is of long duration. For the individual, the extent of symptoms and the disability from the complications can adversely affect everyday life ranging from inconveniences to major disabilities.

The overwhelming burden – including the cost – of caring for the diabetic can simply be described as colossal. Most of this burden comes down to care of its complications [3]. Emphasized here is that the majority of these complications involve the foot. Medicare dedicates a huge fraction of its budget to diabetes in general; the cost of individual care triples when there is a foot ulcer [4].

Evidently, the incidence of Type 1 diabetes (called the "sweet urine disease" in Chinese) has changed little in the population over the ages. Before insulin was available, patients with diabetes did not live long enough to develop complications. On the other hand, Type 2 diabetes has become the new pandemic, affecting huge numbers of people, a trend that parallels the emergence of obesity in the contemporary population.

While an answer has long eluded our understanding of the fundamental cause of complications from diabetes, there is strong evidence supporting the concept that they are related to elevated blood sugars sustained over years. One evocative clue is the finding that endothelial cells in a hyperglycemic environment tend to absorb glucose, an action not dependent upon insulin. The result of the glucose overload is disruption of the normal microcirculation in those vascular beds most notoriously susceptible to diabetic vasculopathy: the retina, the kidneys, and the nerves. Proposed mechanisms are summarized below under section "Pathophysiology."

In Type I diabetes, lack or ineffectiveness of endogenous insulin is recognized early by the evolution of distinctive symptoms; treatment is then initiated soon after discovery as a life-saving intervention. Thus, the duration of sustained, untreated hyperglycemia is necessarily short. How well the hyperglycemia is managed after discovery is another matter.

Type II diabetes, in contrast, evolves over time (perhaps over many years). Hyperglycemia may be overlooked or neglected for long periods, often in the presence of well-known risk factors for vascular disease such as obesity, smoking, hypertension, and hyperlipidemia. Thus, in Type

II diabetes, the complications (including those involving the foot) may be long established by the time the diagnosis is appreciated and treatment initiated.

Complications of diabetes in the feet involve neurological (sensory, motor, and autonomic), circulatory, immunological, and dermatologic systems, as well as those affecting musculoskeletal tissues. The clinician, mindful of these complexities, will use all measures readily available to identify them and to minimize the potential harm of each. He or she – as one pundit put it when it comes to the diabetic ulcer – will mind "the whole and not the hole" [5].

Pathophysiology

Before going further, a brief description of metabolic and neurovascular problems is included that are thought to make the person with diabetes more susceptible to complications.

A century of massive research to understand the basic and clinical problems of diabetes comes down to persistent hyperglycemia that eventually, perhaps through multiple biochemical abnormalities, damages blood vessels and nerves. Both the magnitude and the duration of hyperglycemia are major determinants in the pathogenesis of cellular injury. This concept implies that a strategy for day-to-day control of blood sugar is the bedrock foundation for averting complications.

Research on the causal mechanisms of diabetic complications suggests the following:

1. *Osmotic stress*: In the hyperglycemic state, glycolytic enzymes will be saturated. Excess glucose is then processed by the polyol pathway (also known as the "sorbitol-aldose pathway"). Glycolysis then proceeds as glucose is reduced into the alcohol compound, sorbitol, by the enzyme aldose reductase. Excessive sorbitol accumulates in the cell walls, causing osmotic stress. In effect, the basement membrane is thickened by "sugar coating" while its permeability increases by osmosis. In the process, protein-rich plasma leaks from damaged capillary walls into the interstitial spaces [6].
2. *Oxidative stress*: Free radicals of oxygen (such as HO, H_2O_2, and O_3, as well as oxygen itself) are released in excessive amounts owing to the incomplete pathways of glucose catabolism. These free radicals can exert a direct harmful effect on cells and their organelles and enzymes [7].
3. *Protein fragment stress*: Non-insulin-dependent absorption of glucose by endothelial cells produces excessive glycoproteins in a hyperglycemic environment. From these glycoproteins glycosylated, end products that normally combine with hemoglobin A1c (hereafter, written as HbA_{1c}) are released. Free floating in excess in blood, these protein fragments cause injury to endothelium [8].

The common denominator of these (and perhaps additional) pathological events is excessive glycoproteins causing injury to vascular surfaces. This excess in turn induces a thickening of the basement membrane while at the same time weakening its integrity. Protein-rich serum weeps into the soft tissue of the vascular wall, causing destructive changes (microangiopathy). Eventually, an inflammatory response (vasculitis) occurs along with accumulated lipoprotein deposits that impede blood flow (ischemia). The destructive results affect both large and small blood vessels.

In Type 1 diabetes, an autoimmune process targets insulin-producing beta cells in the pancreas. The ablation of beta cells occurs over weeks or months so that symptoms from hyperglycemia are of fairly recent onset. Hence, the diagnosis is discovered rather soon. The natural killer (NK) cells of the innate lymphocyte system appear to be the chief major cytotoxic mediator [9]. Children and young adults are those most susceptible. Type 1 diabetes, however, can affect older adults. The trigger that starts the autoimmune reaction against insulin-producing cells remains uncertain.

Type 2 diabetes is defined by an acquired resistance of receptor cells to respond to insulin. The resistance is gradual in onset, so that hyperglycemia can precede symptoms by years. Obesity is the most notable risk factor although genetic and environmental factors increase susceptibility.

The greater susceptibility to infection puts the diabetic at further risk of complications. There is a distinct correlation between elevation of HbA_{1c} and a defect in the natural killer (NK) cell-activated receptors [10]. In this way, sustained hyperglycemia places stress on the endoplasmic reticulum system. It appears that there is a fault of the intracellular transport network within this system – mainly for transporting lipids – that plays a critical role in the development of complications in diabetes.

Because it is the microvasculature in the various organ systems that appears to be the principle site of injury from hyperglycemia, effective prevention of complications depends on maintaining control of glucose levels [11]. "Tight" blood sugar control protects against such complications, demonstrated by extensive and long-term studies beginning soon after diagnosis in younger patients. Evidence is suggestive but less firmly established for any benefit of halting progression by tight control once the complications have already set in.

Examination

When feasible, examination of the diabetic foot is most proficiently conducted with the patient sitting on a table or bed facing the clinician and of sufficient height to allow convenient access to the lower legs. The legs dangle with clothing adjusted to exposure of the entire lower leg with dressings and hosiery removed. Adequate lighting is, of course, essential. A combination of incandescent and neon sources (which together simulate "white light") is optimal for evaluating color.

The perspective of symmetry for each observation and test is best appreciated in this direct-facing position. In addition, a habit that provides a powerful aid for remembering each point of attention is to perform each observation and test in a consistent manner: (1) compare the finding at one site with its contralateral match and (2) always move in the same direction, whether left-to-right or right-to-left. The examination best proceeds in an organized sequence from one functional system to another.

Of course, a skillful evaluation of the feet of a diabetic requires practiced experience. Once having acquired some degree proficiency, the examiner can accomplish a comprehensive evaluation within a few minutes. A worksheet is provided at the end of this section. The format is meant to allow the clinician to accurately and rapidly record his or her observations.

For standardized reference, pertinent regions of the foot are depicted (Fig. 28.1).

Of interest is the name for the great toe, the hallux. Of all the toes, only the hallux has an official name. It was thought to be derived from the ancient Greek work "allomai" or "halmos," meaning to leap and alluding to the importance

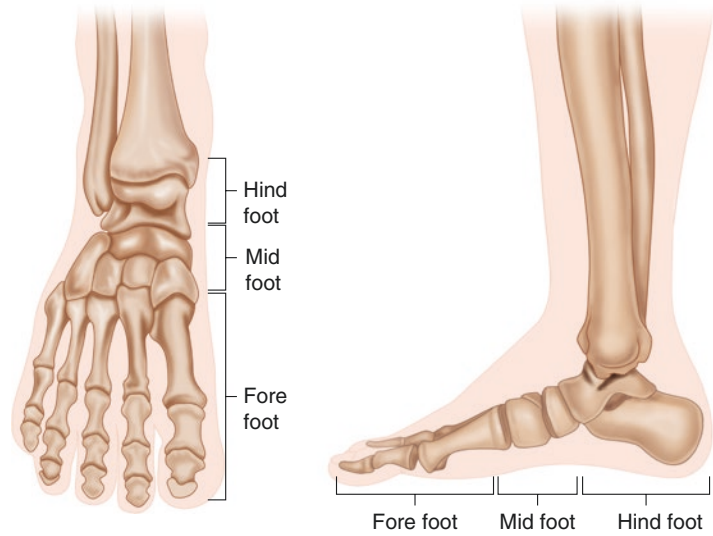

Fig. 28.1 Regions of foot

of the big toe in jumping. The name was altered in Latin to "*allus*". To match the fingers, all of which were designated long ago by anatomical names, *The New England Journal of Medicine* published a modern nomenclature for each of the toes, proposed by a medical student. From great toe to pinkie, they are *porcellus fori*, *p. domi*, *p. carnivorus*, *p. non voratus*, and *p. plorans domum* [12].

With respect for a universal, nursery favorite, "This little piggie …," a more easily remembered and recorded designation for the digits, is suggested: R (right), L (left), F (finger), and T (toe) with a number. For example, a lesion on the right little finger is recorded as RF5 and on the left second and third toes as LT2–3. This system is short, specific, and easily communicated and recorded.

Infection

That diabetics are susceptible to all kinds of infections – bacterial, viral, fungal – has long been recognized [13]. In the foot, they can appear in ways that are quite unusual in the nondiabetic. This introduction to infection of the foot will be expanded under the various organ systems involved.

Infections in the foot of diabetics are notorious for their spreading rapidly. A minor puncture wound can soon blister, become an ischemic pustule, develop cellulitis across the foot and eventually ulcerate. To follow oftentimes is necrosis of the skin and deeper tissues. Within a few days, the infection can extend up the entire leg. Such overwhelming infection, then, may lead to considering amputation as a life-saving intervention.

The astonishing rate with which a seemingly minor lesion in a diabetic becomes complex is well documented in a remarkable series of photographs that appeared in *The New England Journal of Medicine* in December of 2013 [14]. Here a patch of erythema on the dorsum of a foot rapidly evolved into a blister and then, within days, into a large necrotic, infected wound. It is noteworthy that diabetes and peripheral neuropathy were not discovered in the obese 50-year-old patient until this complication emerged. The initiating lesion was thought to be from the trauma of new, ill-fitted shoes.

Infection within the urinary tract, the gastrointestinal tract, or nasopharyngeal area may present as the first manifestation of diabetes, as may skin abscesses. Or it may be that an infection in the foot or elsewhere sets into motion the development of diabetic ketoacidosis or hyperglycemia. Furthermore, diabetics are more likely to have medical procedures than the general population, and, in this way, they are exposed more often to a variety of opportunistic microorganisms.

The reasons for the diabetic's increased susceptibility to infections are complex and not fully understood. Multiple deficiencies within the natural immune system are certainly involved. The increased glycosylation of hemoglobin inhibits the ability of B-cell lymphocytes to produce interleukins, the proteins that mediate interactions between cells. This protective function is particularly important in controlling inflammatory responses, including infection.

Diabetes is also shown to increase the production of other interleukins that tend to enhance the inflammatory responses. The effect promotes the destruction of insulin-producing beta cells of the pancreas and blunts sensitivity of cells in general to insulin activity. Reduced sensitivity to insulin is the cardinal lesion in type 2 diabetes. In these patients, the population of natural killer cells was increased although these cells have impaired cytotoxic activity [15].

Systemic Infections

Under "foot infections of the diabetic," a few words should be included on systemic infections. Such conditions are hardly separable in a clinical evaluation.

Localized infections that may be occult should be considered in any diabetic with otherwise undetermined febrile illness or constitutional symptoms. These include an abscess that can be subcutaneous or in the deeper tissue: retroperitoneal, in the psoas muscle, or perinephritic. Respiratory infections such as community-acquired streptococcal pneumonia or influenza generally hit the diabetic with increased ferocity. A lung abscess is certainly more common in diabetes.

Diabetics are particularly susceptible to systemic infections that are typically indolent in the early stages. Tuberculosis, histoplasmosis, and actinomycosis are preeminent examples. The site of clinically evident infection is notoriously in atypical places, such as in an extremity (including a finger or a toe) and the kidney. Infections of the gastrointestinal tract are more common and organisms include oral or esophageal candidiasis, hepatitis C, and cholecystitis. Emphysematous cholecystitis (from *Salmonella* or *Campylobacter*) occurs virtually only in the diabetic. Infection of the bone and supporting tissues (including fascia) is covered in the section on "Bones."

Skin

Examination of the diabetic foot proceeds with a system-by-system approach. It logically begins with careful observation of the skin.

Color

Whether local or diffuse, obvious or subtle, abnormalities of color in the foot of a diabetic have important implications. Looking at the nail beds and soles is preferred in heavily pigmented patients.

Erythema is an abnormally bright red skin. The cause may be a flush from reflexive vasodilation or venous distention; in contrast, it may reflect an underlying inflammatory condition, including infection. Increased localized warmth, swelling, tenderness, and pain in the erythematous area are addition signs of infection.

Pallor of the nailbed and soles suggests anemia. The clinician can compare this impression with color in the fingernails, palms, or the subconjunctival sac. Giving the impression of anemia, however, is severe vasoconstriction from anxiety, cold, or systemic hypotension.

Cyanosis of the nails indicates a critical level of deoxygenation of hemoglobin. It may have a central cause: either pulmonary or cardiac insufficiency. Cyanosis can also be confined to the peripheral circulation from stasis of blood, as occurs with intensive vasoconstriction from exposure to cold, shock, and in Raynaud's phenomenon. Also causing cyanosis are abnormalities of hemoglobin, namely, the met- and sulf-hemoglobinopathies. To produce cyanosis, the degree of deoxygenation must be lower if the patient is anemic. Conversely, a greater degree of oxygen desaturation is required to cause cyanosis if the patient has polycythemia.

Rubor is a purplish hue somewhere between erythema and cyanosis. In arterial insufficiency with the legs dependent, rubor may be observed in the tips of the toes. It tends to disappear, as the patient lies supine. Rubor may change to pallor when the ischemic leg is elevated.

Spotty *hyperpigmentation* in chronic venous insufficiency tends to occur in the distal lower leg while sparing the foot. The skin becomes stained by iron deposited when blood leaks from the plethoric microcirculation. The discoloration is associated with lipodermatosclerosis, as described in the vascular subsection on venous diseases of the leg. Other causes of hyperpigmentation, rare but important to recognize, are primary adrenal insufficiency and hemochromatosis.

A slight *yellowish* hue of the skin can sometimes be found in patients with long-standing diabetes. It is often most prominent on the soles. The discoloration is probably not caused by deposits of bilirubin but rather by the products of glycosylation.

Black is a sign of ischemic necrosis. In diabetes, necrosis is typically distal and involves the smallest arteries and the arterioles. It usually occurs in one or more toes. Necrosis may be confined to the skin alone, or it may extend into the muscle, tendons, and bone. Certainly, a black toe – revealing gangrene – demands urgent attention. As in frostbite, the vitality of the underlying tissue immediately proximal cannot be ascertained by inspection of the surface alone.

Texture

Increased Thickness

Edema

The principal cause of leg swelling is from venostasis. The edema can be simply a phenomenon

of osmotic gradient from prolonged sitting, in which case the swelling is likely to be symmetrical. It may be a chronic condition resulting from series of defective venous valves, resulting in venous insufficiency. The swelling may be confined to one leg or, if bilateral, decidedly more in one leg. In this condition, edema is most prominent in the ankle and the lower calf while the foot is largely spared. Usually, pitting of the edematous tissue is easily demonstrated. Swelling of the lower legs from lymphedema, in contrast, tends to involve the foot as well while it resists pitting. Descriptions of these conditions are expanded under "Peripheral Vascular System."

Callus

Thickened skin over the metatarsals of toes R or L 1, 2, or 3 suggests points of increased weight-bearing pressure. The formation of calluses in unusual places of the sole is common in diabetics; it is a sign of neuropathic injury that compromises the muscles and architectural support [16]. Calluses are lesions that can develop necrosis in the subcutaneous tissue and eventually ulcerate.

Myxedema

Skin that has a thickened and pasty texture suggests myxedema. This condition with its unique feel can occur in the feet although it is usually more evident in the face and forearms. Pitting of the doughlike skin of myxedema, incidentally, is minimal; it is not to be confused with that of edema in which pressure from a fingertip causes a deep impression. For presumptive confirmation, deep tendon reflex are slower, particular on their return after stimulation.

Necrobiosis Lipoidica Diabeticorum

Necrobiosis lipoidica diabeticorum appears as waxy or porcelain-like lesions that are raised and erythematous. Lesions tend to coalesce, forming patches that have a yellowish-brown coloration with a violaceous border. Over time, the involved skin atrophies, leaving a hardened surface that tends to crack and ulcerate. Multiple telangiectasias are likely to be present and local anesthesia demonstrable. At this stage, the edges of ulcers in necrobiosis lipoidica diabeticorum characteristically are undermined, differing in this way from the typical "venous ulcer."

Lesions of necrobiosis lipoidica may occur in any region of the body, including the foot. They are most common in the lower legs, usually in the shin, and can cover a large area. The lesions are often bilateral.

Fewer than one in a hundred diabetics will ever develop necrobiosis lipoidica. Yet this condition happens almost exclusively in diabetes. Its underlying etiology is uncertain. Perhaps the cause is multifactorial with degenerative changes in collagen tissue that is infiltrated with inflammatory cells. One presumptive cause is that the cutaneous tissue damage is due to weakness in the walls of the smallest blood vessels, perhaps caused by the same mechanism that produces microvascular changes in the retina and in the glomeruli.

Decreased Thickness

The turgor of skin provides clues to circulatory adequacy. In arterial insufficiency, the skin becomes smooth with loss of hair, particularly noticeable just above the ankle. Chronic venous insufficiency also causes thinning of the skin. At the same time, the skin hardens from loss of subcutaneous adipose tissue (hence the term "lipodermatosclerosis"). Hyperpigmentation from iron deposits is a conspicuous feature of the venostasis syndrome.

Temperature

Using the back of the hand is probably the most sensitive way of feeling small differences in temperature between the feet and within areas of the foot.

Increased Temperature

Neuropathic Vasodilation

In the diabetic, a warm foot does not necessarily indicate adequate blood flow. Indeed, the neuropathic loss of sympathetic vascular regulation favors vasodilation of the small blood vessels and with it increased warmth. Arterial-venous

shunting in the deeper tissues, in addition, can be quite extensive. These changes greatly increase blood flow in some areas of the foot while masking areas of ischemia in other areas. Surely, evaluating temperature to evaluate the health of the diabetic foot challenges the clinician.

Infection

Cutaneous infection – bacterial or fungal – occurring anywhere in the foot produces vasodilation and increased temperature. The imminent danger for diabetics is the inherent possibility of extending rapidly, as described earlier in this section.

On finding an overly warm foot of a diabetic, the clinician must rule out cellulitis and consider deeper pathology: abscess, arthritis, and infection of the bone. Vasculitis leading to arterial-venous shunting in the small vessels and vasodilation from impaired sympathetic vasomotor control may be superimposed. Sensory neuropathic defects can eliminate pain and tenderness in affected areas. Even when the foot feels excessively warm, at least in some places, the foot can be ischemic in others. The clinician must remain vigilant to this paradox.

Decreased Temperature

Thrombosis/Emboli

(a) Acute. When, in the foot, a cool area occurs rather abruptly, an arterial thrombosis or embolism must be ruled out expeditiously as a medical emergency before tissue injury is irreversible. With coolness of the whole foot, an obstruction in the superficial femoral artery at or above the knee is likely.

A sharp demarcation from warm to cold anywhere in only part of the foot suggests arterial thrombosis or an embolism of moderate size, compromising one of the three arteries that supply the foot. In the normal foot, however, the collateral circulation among the three major feeding arteries is so extensive that obstruction of one or even two of the arteries is unlikely to lead to ischemic changes provided that the other arteries are fully patent. The presence of ischemia in a part of the foot implies that all three of the pedal arteries have at least some degree of stenosis (Fig. 28.2).

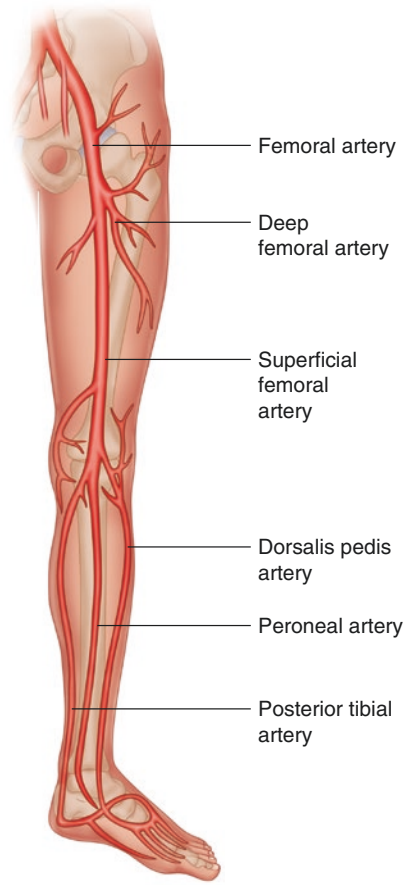

Fig. 28.2 Arterial supply

If some but not necessarily all the toes are cool and blanched or cyanotic, small emboli should be suspected. Emboli may arise from an arterial source upstream such as a disrupted cholesterol plaque or platelet aggregation. An intracardiac thrombosis, vegetation on a valve, or, rarely, a disintegrating intra-atrial myxoma may be responsible. Fat emboli can occur from liposuction procedures. Arteriolar spasm must also be considered, usually with the more obvious manifestations in the hands.

Atherosclerotic Stenosis

(b) Chronic. When coolness in the foot (or part of the foot) occurs slowly over weeks or months, underlying stenosis of an artery is suspect. Often, this event is associated

with pain on effort, a form of claudication. This subject is covered later under section "Peripheral Vascular System."

Moisture

Running the back of the hand along the palm or sole normally gives the sensation of slight traction owing to moisture from insensible sweating. The degree of moisture is closely controlled by the autonomic nervous system. Sweating is a parasympathetic function. Because the efferent fibers run in close proximity to sympathetic nerves, a strong adrenergic stimulus can activate the sudomotor system.

Increased Sweating

Sweating is our major conveyance for dissipating the heat of metabolism. Those areas where sweating is most effective are the hands, scalp, and soles. Fever, extended vigorous exercise, and a hot environment all drive this critical function to an accelerated degree. Increased sweating is an expected consequence of hyperthyroidism. It is also a feature of early neuropathic changes in sudomotor innervation. In each condition, the skin remains warm and ruddy.

Diaphoresis

The term "diaphoresis" is used to denote episodes in which markedly excessive sweating occurs in response to an abnormal condition. Beads of sweat appear in response to strong adrenergic stimuli, as from severe fright or profound hypotension. These states are accompanied by vasoconstriction in which the skin is cool and pale.

Hyperhidrosis

Persistent and excessive stimulation of the sudomotor system in the diabetic may be an early sign of neuropathy involving autonomic nervous system control. A common symptom in this situation is sweating that is easily precipitated by overwrought emotions and by eating spicy foods. Presumably, hyperhidrosis is an early phase of neuropathic injury to be followed by excessive dryness as the condition progresses.

Decreased Sweating

The lack of normal moisture in the palm and sole produces a surface that the examining hand passes over with little or no traction. While the difference between the normal and the dry hand may seem subtle, the clinician will be able to detect it after some practice.

Xerosis

Dryness of the sole occurs naturally in the elderly patient, a condition called "xerosis." It can also be a prominent complication of diabetes owing to degenerative changes in the autonomic nervous system, resulting in sudomotor neuropathy. Because the long tracts to the foot are more susceptible to neuropathic changes than are those in the hand and scalp, xerosis may occur in the sole while, paradoxically, excessive sweating occurs in the palm and forehead, an earlier manifestation of the same process.

Dry skin is fragile and easily cracked, allowing microorganisms to invade more easily. Neuropathic pruritus, more common in diabetics, increases the possibility of the skin breaking down through the abrasions of scratching. Indeed, scratch marks on the foot may be telltale signs of such a neuropathy.

Bumps

Callus

A callus is an excessively thickened layer of the skin that is raised and flat-topped. Its horny texture occurs at points of repeated pressure or friction. The build-up of keratin is protective to some extent, as is evident in the palms of a manual laborer. In the foot, normally, there is a thickening of skin that is fairly evenly distributed over the sole where weight is borne. A callus will form on the plantar surface where there is excessive pressure and friction. Most commonly, calluses occur at the heads of the metatarsals of the first and second toes.

While a callus is insensitive and may appear benign, the tissue underneath is particularly susceptible to injury in the diabetic. Sometimes a necrotic lesion develops beneath a callus, out of

sight, and unfelt because of coexisting sensory neuropathy. Bleeding behind a callus is a telltale sign of such underlying necrosis. The result is an imminent breakdown of the skin with subsequent ulceration. The ulcer, of course, is exactly where the burden of weight is concentrated with each step.

Corn

Corns are bumps of the skin produced by friction in areas of the foot that are not weight-bearing and where the skin is normally thin. They have a firm core and can be painful when pressed against the deeper tissue. The most common site is the upper surface of a toe where the box of a shoe rubs against it. Deformities of the toes (such as the claw toe and hammertoe) increase the liability of developing a corn. A "soft" corn can occur between the toes (usually between the fourth and fifth toes) where it is susceptible to maceration of the skin and subsequent breakdown.

Bunion

A bunion is a bulge caused by lateral deviation at the base of the great toe. Called "hallux valgus," the misaligned head of the first metatarsal bone is vulnerable to inflammation from excessive pressure from a tight shoe. Bursitis with swelling, erythema, and pain is a frequent complication. Sometimes a bunion will cause a callus between first and second toes where the toes are pressed together, and this overgrowth can develop into a wound.

Neuroma

A bump on the ball of the forefoot is referred to as a "neuroma" and even more specifically as "Morton's neuroma." It is a misnomer, however, since it is not a proliferation of nerve fibers but rather an enlargement of ligaments. The growth, which may or may not be palpable, rubs against the adjacent nerve, causing tenderness and pain on weight-bearing. It can have the appearance of a callus but is more fleshy and is more dome-shaped than flat-topped. A neuroma is usually more laterally placed on the sole than is a callus, most commonly between the second and third toes.

Ill-fitted footwear is thought to be a prominent cause of the neuromas. High heels worn habitually are particularly culpable.

Blister

A blister appearing anywhere on the foot indicates a reaction to recent trauma, friction, or, more ominously, to an underlying necrotic process. Such must be taken very seriously in the diabetic. In particular, look carefully for a pressure-inducing irregularity inside the shoes. Without an obvious explanation and a readily correctable problem in the shoe, a concerted investigation for the cause is warranted.

Spots

Petechiae

Petechiae on the foot and elsewhere are telling signs. Petechiae that are non-palpable may be the result of thrombocytopenia or of malfunctioning platelets. Petechiae that are elevated and are therefore palpable, in contrast, are caused by inflammation in the microcirculation. Vasculitis or infected microemboli may be responsible. They define a pressing need to detect the source, whether cardiac, hematologic, vascular, or infectious.

Emboli

Fragments of vegetations on cardiac valves can migrate to the hands and feet, producing visible emboli. Janeway lesions are tiny, hemorrhagic lesions on the palms and soles. Osler's nodes are septic lesions, occurring mostly in the digital tips. They are red, often with a pale or white, purulent center. Both Janeway lesions and Osler's nodes can be painful and tender. Both dictate expeditious care.

Pigmented Maculae

Leaking capillaries can produce tiny reddish-brown spots that appear in splotches and may coalesce. Also known as "cayenne pepper spots," the pigmented lesions usually occur over the tibia, although they can appear anywhere. The discoloration comes from deposition of iron

in extruded hemosiderin that may persist for years. While the pigmented maculae are themselves innocuous, they are warning signs of important pathology elsewhere. Particularly in the diabetic patient, they should draw attention to the likelihood of vascular disease in the retina and the kidneys.

Dermatophytosis

The tightly apposed surfaces between the toes form a space of excessive moisture and heat. When blood glucose is elevated, the combination provides an environment that favors the growth of fungi. All interdigital spaces need to be inspected for fungal disease on each periodic examination of the diabetic.

Tinea Pedis

Sloughing skin that forms white, peeling patches between the toes is evidence of fungal activity. Also known as "athlete's foot" and "ringworm," these interdigital infections increase the need for oxygen in the adjacent tissues. When, in addition, circulation is compromised, the infected digits become more susceptible to damage from coexisting ischemia.

Disruption of the interdigital skin provides a portal for the entry of microorganisms. The presence of palpable lymph nodes in the popliteal or inguinal areas suggests that extension of the infection has already occurred, either by fungi or bacteria.

When tinea pedis extends beyond the toes, it often takes on a "moccasin" distribution over the sole. Of course, the fungal infection can become widespread, and it is particularly threatening to the diabetic. It tends to be more aggressive while facilitating the invasion of other infectious agents.

Edema

To evaluate edema of the lower extremity, pay careful attention to symmetry. Fluid retention from systemic abnormalities – whether of cardiac, hepatic, renal, or protein deficiency origin – causes symmetrical swelling. Pitting edema in these conditions is generally prominent.

Edema from venous valvular insufficiency and lymphedema is often unilateral. When bilateral, there is usually some difference in leg girth. Spotty hyperpigmentation, especially over the lower calf, is typical of venostasis, not of lymphedema. Lymphedema has a greater propensity to involve the foot than does venostasis. The turgor of skin in both conditions is more resistant to pitting than are the systemic causes previously mentioned. Sometimes, because of overlapping signs, venous insufficiency can be difficult to distinguish from lymphedema. Indeed, they can occur together.

If observation cannot distinguish a discrepancy between the girths of the calf or ankle, reliable measurement of circumferences is helpful. Guidelines for measuring maximum calf and minimal ankle for comparison can be found under Chap. 22.

Cellulitis

The combination of localized erythema, edema, and tenderness when limited to the dermis and subcutaneous tissue is called "cellulitis." It is an inflammatory reaction, usually from an infectious process that follows a break in the skin [17]. Trauma, interdigital tinea infections, drug injections, and venous stasis disease with lipodermatosclerosis are the most common underlying conditions, each more likely to result in cellulitis among the diabetic than in the nondiabetic population.

An area of cellulitis is generally considered superficial if it is less than 2.0 cm in the largest diameter. Such a lesion, however, must be monitored frequently in the diabetic because of the tendency to spread rapidly. Streptococcus organisms are the most frequent invader, although gram-negative organisms and fungi can also lead to cellulitis. If a break of the skin cannot be found in an area of cellulitis (as often happens), sterile aspiration for fluid to culture may be warranted.

When the area of cellulitis in a diabetic is greater than 2.0 cm across, it is deemed serious, wanting of urgent treatment. Significant ischemia in the area of cellulitis, inflammation of a neighboring bone or joint, or signs of systemic toxicity indicate that the condition is at a critical stage.

Often an inflammatory cause of cellulitis can be distinguished from venous valvular reflux disease simply by lifting the leg of the recumbent patient. The erythema of the raised leg does not fade appreciably when cellulitis is present. In contrast, the dilated cutaneous vessels of venous insufficiency empty rapidly and the intensity of the erythema will lighten or disappear.

Wound

Where the skin breaks down in the diabetic foot to form an ulcer, there is a real potential for rapidly progressing complications. Any break in the skin that fails to heal promptly is considered an ulcer. For effective management, it is critical to determine the underlying cause(s) of a wound, whether neuropathic, ischemic, traumatic, or of multiple etiologies.

A harsh realism is that an ulcer in the foot of a diabetic forewarns of the possibility of eventual amputation of the limb [18]. A foot ulcer in the diabetic is considered high risk for amputation if he or she is greater than 50 years old or if diabetes has been present for more than 10 years. About half of elderly people with a foot ulcer complicating Type II diabetes will eventually undergo amputation. Greatly accentuating the risk are smoking, hypertension, hyperlipidemia, and an abnormal ankle/brachial index.

The most common inhabitant of a diabetic ulcer is staphylococcus. Enterococci, streptococci, and enterobacteria are fairly frequently cultured from these wounds. A significant minority, however, are anaerobic bacteria. The longer the duration and depth of a diabetic ulcer, the more likely it is that multiple species of microorganisms are present. These chronically infected ulcers, in general, tend to harbor microorganisms that are more resistant to antibiotics.

Edema from any cause may promote the susceptibility to wound development by allowing protein-rich interstitial fluid to accumulate in skin and subcutaneous tissue. Lymphedema, for example, contributes to the persistence of diabetic foot ulcer [19].

The character of the wound is determined in part by the forces applied to the area. The pressure of weight-bearing with each step tends to increase the depth of the wound. On the other hand, walking imposes more of a shearing effect; this movement has more effect on increasing the size of the wound.

A strong relationship between diabetic foot ulcers and low serum magnesium levels has recently been pointed out [20]. Hypomagnesemia is most likely to occur in the alcoholic, after persistent diarrhea or associated with aggressive diuretic therapy. It also frequently coexists with conditions that cause hypokalemia [21]. Yet, blood levels of magnesium are not routinely performed so that hypomagnesemia may be greatly underdiagnosed.

Each wound should be fully described by observations and recorded measurements. The CHECK-OFF LIST at the end of this section provides a convenient format. Documentation involves the following characteristics of each lesion:

1. Position: An ulcer overlying the plantar heads of the metatarsals typically occurs from the repetitive stress of walking. When the ulcer is over the lateral or medial foot, a tight shoe is the more likely cause. An ulcer at the tip of a cocked-up toe is probably the result of the shoe's toe box being too small in vertical dimension. A wound can occur between the toes and might be missed if the toes are not spread for inspection.
2. Dimensions: Drawing an outline of each lesion's configuration and dimensions adds substantially to the quality of the record, and it facilitates follow-up evaluations. A see-through ruler is advised so that direct contact with the ulcer can be avoided. A photograph that includes a ruler lying alongside provides irrefutable documentation.

(a) Width and length. Measurement is made of the general dimensions.
(b) Depth. Is the wound superficial (only involving the cutaneous layer) or are underlying tissues (fascia and/or bone) visible (Fig. 28.3)?

3. Hydration: Is the surface moist or dry?
4. Exudate: Describe any discharge regarding amount, viscosity, and color.
5. Roof: Is there a scab (clotted blood) or cicatrix (clotted serum)?
6. Exposed subcutaneous tissue: Is the surface granular, fibrotic, or necrotic?
7. Margins: Are the ulcer margins adherent, undermined, macerated, or necrotic?
8. Odor: An odor emitted by a wound implies infection and dictates urgent attention in the diabetic. Some microorganisms give off a characteristic odor. For example, anaerobic bacteria produce a sulfhydryl that smells like rotten eggs. Pseudomonas aeruginosa gives a musty aroma that some liken to that of grape juice. *E. coli* has the putrid smell of sewerage. *Brevibacterium*, an organism that digests dead skin, produces the smell of "ripe" cheeses.
9. Surrounding tissue: Is the skin, including the subcutaneous tissue around the wound, swollen and indurated? Protein-rich fluid can accumulate, causing varying degrees of lymphedema and promoting wound development.

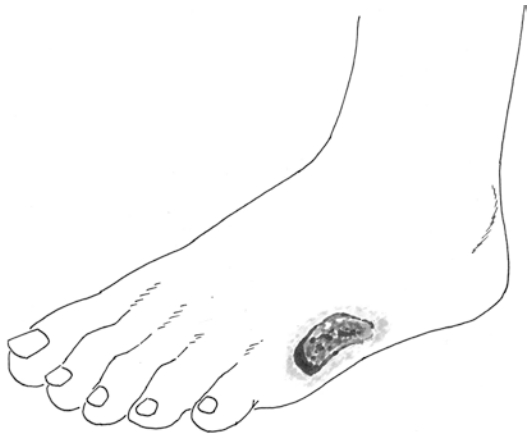

Fig. 28.3 Ulcer

Nails

Discoloration

Nails in long-standing diabetes tend to have a yellowish hue. The discoloration is probably a combination of a thickened nail (from slower shedding of normal keratin) and from end products of glycosylation. It occurs first on the hallux, later spreading to all the toenails.

Onychomycosis

Fungal disease of the nails is common among diabetics. It is a prevalent forerunner of diabetic foot complications. The most common causes of onychomycosis are *tinea* organisms. Indeed, the severity of fungal infections of the nails is a marker of the extent of other complications of diabetes [22].

Paronychia

A paronychia of the toe – either fungal or bacterial – presents the substantial risk of spreading to other areas of the foot. With compressing palpation, one feels a spongy nail bed. *Staphylococcus* is the usual opportunistic agent at sites where an ingrown nail breaks the skin. Infection by *pseudomonas* gives a greenish hue to the nail.

Subungual Hemorrhage

The nails of patients with neuropathy are more easily affected by relatively minor injuries. Subungual hemorrhage, as an example, is fairly common.

Periungual Telangiectasia

Dilated capillary loops form telangiectasias at the fold of the nail. They have a strong correlation with the incidence of microvascular abnormali-

ties elsewhere and therefore are perhaps comparable to the small blood vessel lesions best recognized in the retina and kidneys. While small, they are more readily seen by rendering the nail more translucent with the application of mineral oil. Adequate magnification is readily available with the ophthalmoscope, with the convex lens setting at +5 to +10 diopters.

Nervous System

Neurological complications of diabetes are all too familiar. Probably, at least one out of three diabetics – excepting those with recent onset – has some form of neuropathy, however minor. It may affect one out of four persons with impaired glucose tolerance [23]. Neuropathies seem to occur with equal frequency in Type 1 and Type 2 diabetes. The reason speculated for this similarity is that persons developing Type 1 diabetes from lack of insulin begin to receive treatment soon after the manifestations of severe hyperglycemia are discovered. Person with Type 2 diabetes, on the other hand, may have hyperglycemia to a lesser degree, but the hyperglycemia from resistance to insulin may have been present for years before the disease is recognized and treatment begun.

Now well established is the concept that achieving long-term glycemic control can prevent or slow the development of peripheral and autonomic neuropathy. It cannot be expected to reverse preexisting neuronal damage.

Anatomically, hyperglycemia-induced neuropathy is length dependent. That is, the longer the nerve, the greater the susceptibility to neural damage. Thus, the legs are more apt to be affected than the arms. The earliest neuropathic injury appears in the feet and creeps proximally over time, essentially in a symmetrical distribution. Neuropathies in the insensate foot, along with weakness of its intrinsic muscles and reduced coordination, subject the foot to repeated trauma while walking. They also greatly increase the risk of falling.

Painful sensations and weakness may also stem from inflammation in the blood vessels supplying nerves, causing an ischemic injury to the nerve [24]. Such obstructing lesions in the microvascular bed contribute in a major way to the development of neuropathies, injuring both the axon and the sheath. Any contributing form of vascular disease may go unrecognized in a diabetic patient with paresthesias or a wound when a neuropathy is considered the sole cause.

Concerning the distribution of diabetic peripheral neuropathy in the general population, a study of adolescents and young adults with diabetes revealed an incidence that is nearly as great as in the older adults [25]. The frequency of neuropathy in older patients with Type 2 diabetes was appreciably higher than in Type 1 diabetes.

In the broad spectrum of neurological manifestations of diabetes, abnormalities of the sensory, motor, and autonomic functions can occur. Some clinicians screen for peripheral neuropathy in the feet using a sampling triad of tests: light touch sensation, vibratory sensation, and deep tendon ankle reflex. A somewhat expanded outline for neural testing is presented here for a baseline evaluation and for follow-up of all three forms of neuropathy.

Sensory Nervous System

Neuropathy

Paresthesias from neuropathies are generally described as tingling, a "pins and needles" feeling, numbness, or the sensation of a limb "going to sleep." Others experience a prickling or crawling feeling. Some feel a persistent heaviness or coldness. Itching can be the dominant symptom of cutaneous neuropathy with evidence of scratching often obvious. Perhaps most distressing of all is the sensation of incessant burning.

Paresthesia from a neuropathy may be intermittent or unremitting. The sensation is often most disturbing at night. The intensity of paresthesias ranges from barely perceptible to upsetting in the extreme, resulting in a perpetual distraction, exasperation, and insomnia. Yet, about half of diabetics with sensory polyneuropathies identified in a large study had no neuropathic symptoms [26]. Nor had they been told

of having a neuropathic condition by their primary caregivers.

Sensory neuropathies in diabetes typically evolve in a "stocking-glove" distribution. That is, the sensory defects have a circumferential pattern in the extremity, not the segmental pattern of an isolated nerve distribution. Generally, the involvement is fairly symmetrical in the lower legs.

As the neurological complications of diabetes progress, paresthesias gradually lessen with further injury to nerves, leading eventually to numbness. The resulting anesthesia may be welcomed by patients, long-suffering from paresthesias. It should also be noted that many diabetics develop loss of sensation without any preceding paresthesia.

The loss of sensation does not in itself pose a serious threat to general health. On the other hand, the insensate foot carries the burden of severely reducing the patient's awareness of regional pathology. The acronym LOPS is applied to mean "loss of protective sensation." It emphasizes the danger from the lack of telltale symptoms of discomfort or pain that could have led to corrective measures. There is inherent in the loss a greater propensity for falling. Certainly in the aging person, the combination of feet lacking sensations along with issues of balance, strength, and vision is a distinct threat for maintaining a self-sufficient lifestyle.

Trauma to the foot often goes unnoticed. It can come from dropping a fork on the unprotected foot, from stepping on a jagged object while walking barefooted, or from a wrinkled sock or pebble in a shoe, all dangers in the insensate foot. An abrasion, puncture, or break in the skin from concentrated pressure allows microorganisms to invade the subcutaneous tissue and beyond. As already noted, this series of events comprises, in the minds of many clinicians, the essential burden of the "diabetic foot."

Lumbosacral Radiculoplexus Neuropathy

In contrast to the stocking-glove pattern typical of diabetic neuropathy is a syndrome known as the "lumbosacral radiculoplexus neuropathy." Also known as diabetic amyotrophy, it is the result of inflammatory and microvascular stresses occurring at multiple levels in the nerve roots as they emerge from the lumbosacral cord [27]. Diagnosed mostly in diabetics, the syndrome is characterized by pain and the blunting of sensations. The distribution is notably asymmetrical and may extend from the toe to the area above the knee. That the pathology of this syndrome has an immunological basis is suggested by some lessening of pain with immune-mediated interventions.

In addition to sensory expressions, lumbosacral radiculoplexus neuropathy also affects the motor system. Weakness from muscle wasting with fasciculations is evident. In addition, commonly associated symptoms of intestinal and urogenital dysfunctions imply that autonomic neural control is also involved in the process.

Nerve Entrapment

Some people with early symptoms of neuropathy describe pain as having a stabbing quality. In such cases, the clinician must rule out the possibility of a nerve entrapment syndrome. This possibility may include compression of the tibial nerve, called the "tarsal tunnel syndrome." If the pain is in the heel, the calcaneal branch of the tibial nerve is compressed. If the pain is toward the forefoot, either the lateral plantar nerve or the medial plantar nerve is involved.

The sensory examination of the foot can be performed rapidly with fair reliability. Outlined here are tests for nerve conduction in three distinct sensory pathways. At the same time, it is acknowledged that the accuracy of testing for sensation requires a patient subjective response; it is certainly one of the most challenging aspects of the neurological evaluation.

For screening purposes, the focus here is on the tips of the great toes. If sensation is intact there, it can be assumed that the long trunk from toe to the sensory cortex of the brain is intact, although branches higher up may be damaged. Should a reduction of sensitivity be encountered here at the toe, then the site tested should be moved progressively to more proximal levels in short increments – proximal foot, ankle, and so on – until the stimulus applied is detected.

Immediately before applying each stimulus to the foot, give the patient some idea of what to expect by first demonstrating the test by applying it to the tip of a finger. Furthermore, it is critical that a stimulus is applied with uniformity for side-to-side comparisons. This suggestion is applicable to all tests of tactile sensations. Those sensory tests to be described here are (1) light touch, (2) sharp, and (3) proprioception. Each ascends in a separate pathway within the spinal cord to the sensory cortex.

Light Touch

The loose end of a facial tissue makes a convenient and effective stimulus for testing light touch. The patient with normal sensations of the toe will feel its brushing across the tip. He or she should have the eyes closed or looking away during the touch.

For greater standardization of light touch, a calibrated instrument has been developed. Perception is measured when enough pressure is applied to bend a nylon microfilament. Instruments calibrated for 1-gm of pressure are the most sensitive. The 10-gm version is the least sensitive but has proven useful in annual evaluations on large numbers of diabetics [28]. The precision that this method provides is probably not necessary to adequately follow up in the periodic, non-research physical examination.

Sensations for light touch are transmitted from the spinal cord to the brain by the ventral spinothalamic tract. The ability to feel the end of a facial tissue brushing against the toe-tip is an assurance that this pathway is intact.

Sharp

For providing a stimulus for sharp, the tip of a wooden tongue blade twisted apart along lengthwise is a safe and effective tool. Just enough pressure is applied to indent the skin slightly. Do not use a pin or other metallic point that may penetrate, even microscopically, the skin. The handle tip of a reflex hammer (commonly used for testing sharp) may be too blunt for optimal evaluation.

Pain-conducting fibers are carried in the lateral spinothalamic tract. Fibers sensitive to temperature are also carried along the same tract. Except in complex neurological cases, it is not necessary to include more time-consuming testing for distinguishing warm from cool if the sensation of sharp is intact.

For recording this test, the term "sharp" rather than "pain" is preferred. The issue is a matter of interpretation. The latter term implies purposeful testing with a technique that hurts.

Proprioception

Proprioception – that is, the ability to sense one's position in space – is a complex function. It can be evaluated is several ways, both with the patient sitting and standing. Tests include vibratory stimulation, position of toes, and station. It is recommended that two of these three tests be included in the general neurological assessment.

The neural pathways for proprioception are within the posterior columns of the spinal cord. These originate in the joint capsules, ligaments, and muscles and enter the dorsal horn of the cord as heavily myelinated fibers. Those from the legs follow a track in the medial part of the posterior columns (the fasciculus gracilis) and those from the arms track more laterally (in the fasciculus cuneatus). These pathways ascend and decussate in the medulla, terminating in the thalamus. From there, other neurons carry proprioceptive stimuli to the parietal cortex. The receiving cerebral cortex communicates with the cerebellum by several pathways.

Most affected in sensory ataxia from polyneuropathy or posterior column lesions are gait, the awareness of limb movement, and position and the perception of vibration. Nystagmus and dysarthria are not typically present.

Vibration

A useful and sensitive instrument for testing proprioception is the 128 cps tuning fork (the one with the two bulbous discs at the end of each

prong). The force used to initiate the vibrations must be uniform for each side-to-side comparison. For consistency, a moderately strong strike on one prong with a reflex hammer is recommended although using the lateral side of the hand may be more expedient. A more forceful strike should be applied if the first vibratory stimulation is not felt.

Should the vibration not be felt in the toe, try it again at the medial malleolus, then moving upward from there in steps up the tibia to the knee, if necessary, until the stimulus is perceived.

Neurologists favor the "time to dissipation" test of the vibratory sensation as a more precise measure of proprioception. The stimulus is standardized by striking the 128 cps fork with force sufficient to hear a "clang" from its bulbous ends (creating its maximum vibratory capacity). The patient indicates by voice or hand signal exactly when the vibration is no longer felt. Of course, the test requires sustained attention by the patient. The nonspecialist examiner may find this technique too time-consuming or too exacting in a screening evaluation for practical purposes.

An instrument, the Bio-Thesiometer®, provides vibrations that can assess sensitivity with greater quantitative precision than attainable with a tuning fork. Driven electronically, it is more appropriately used in clinical research.

Toe Position

Testing the sense of toe position needs to be completed only if the patient cannot stand for the test of station (described later). It, too, integrates proprioception, neural impulses ascending the posterior columns of the spinal cord. The test can be rather time-consuming and ambiguous in patients who do not quickly grasp the idea of what is expected of them, even with a careful explanation.

The patient, who is sitting or recumbent, is asked to recognize whether the passively flexed or extended great toe is pointed "up or down." A critical component of the test is that the toe is held between two of the examiner's fingers, one applied to each lateral aspect, not on the dorsal and ventral surfaces. The examiner's other hand is used to block the patient's view of the foot (Fig. 28.4).

Fig. 28.4 Toe position testing

Station

The standard test of proprioception is performed with the patient standing. Commonly referred to as the "Rhomberg test," assessment of station documents the ability of the patient to stand with the eyes closed. The proper stance requires that the heels touch and the toes spread outward at about a 45-degree angle. When proprioception is compromised, vision is the dominant sense by which the upright stance is maintained. In perspective, testing station is extremely effective in exposing a deficiency in proprioception.

To insure that proprioceptive sensitivity is intact, the patient needs to hold this station with eyes closed for at least 10 s. As a precaution, the clinician is advised to assure the patient that his or her hands, removed from the patient's shoulders once the eyes are closed, will be held close by in case of unsteadiness.

Sensory input that conveys proprioception is carried by way of the posterior columns of the spinal cord. In this simplified illustration of the cord in cross section, the three sensory pathways just described are designated (Fig. 28.5).

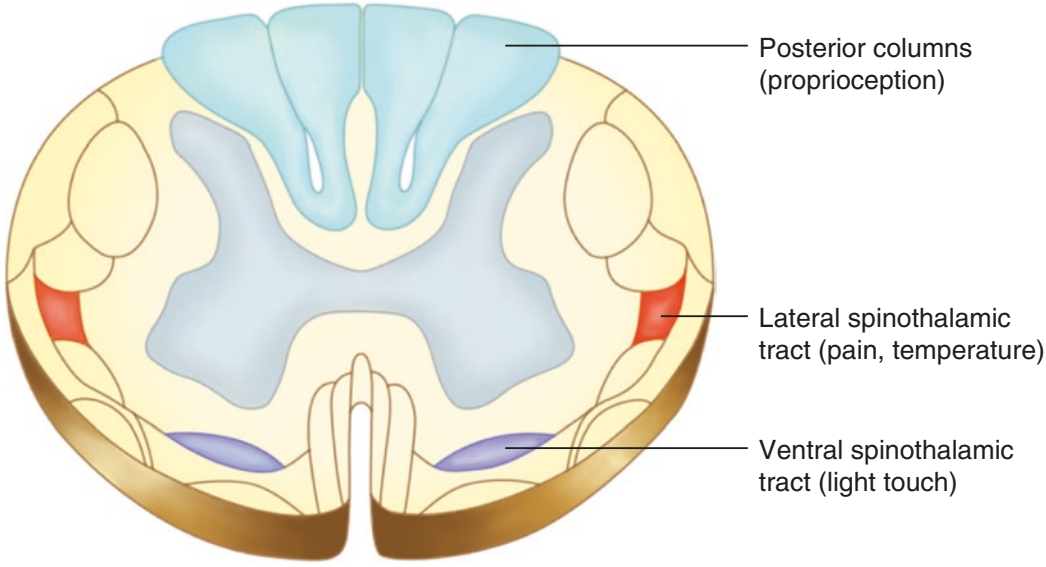

Fig. 28.5 Sensory pathways: composite

Tandem Walk

The tandem walk test does not assess an isolated component of the neurological system but rather integrate multiple components: proprioception in addition to coordination, muscular strength, and a sense of kinesiology (perception of where a limb is in space). The test consists of slowly walking in a straight line by placing one foot directly in front of the other, touching heel-to-toe. Using a straight line in the floorboard, carpet, or tile as a guide is helpful.

The patient should be made aware that tandem walking is not an easy test. For those patients with limited ambulatory agility, it can be very challenging, even when there is minimal or no neurological deficit. The examiner must stand very close to the patient but without touching in order to provide instant support, if needed.

A truly abnormal tandem walk is usually evident within the first few steps. Such abnormality does not indicate a defect in a specific tract but rather a problem in transmitting the neural input, assembling it in various areas of the brain and then performing the task. Hence, it is a useful screening test for assessing how the gears meshing in sensory-motor coordination.

Pharmacological intervention is appropriate when diabetic paresthesias are more than a trifling matter for the long-suffering patient. Reference to a recent and comprehensive review on this subject is available [29].

Motor Nervous System

Loss of motor function is a serious complication of diabetes. Neuropathy of the motor system, while perhaps less common than those affecting sensations, is generally more disabling [23].

Atrophy

Neuropathy within the motor system results in the atrophy of muscles. In the foot, it is the small, interdigital muscles that are most involved. While evidence of muscle atrophy in the foot may be subtle, it can be detected by careful observation. On the dorsum of the foot, grooves (or "guttering") between metacarpals may be apparent. It is likely that atrophy in the hand muscles is also present and is most obvious in the thenar, hypothenar, and or interosseous muscle groups.

Weakness

Of course, muscle atrophy results in weakness. The arch of the weakened foot tends to collapse on weight-bearing, accounting for the "fallen arch." This tendency can best be evaluated by placing the central three fingers beneath the arch as the patient "walks in place" on a stool. With experience, one can distinguish the difference between the normal instep and one exerting excessive pressure from flat feet (Fig. 28.6).

Weakness usually begins in the extensors of the toes. It slowly progresses to the flexors. With increasing territory, the extensors of the foot become weakened, followed by the flexors of the foot.

As in the sensory examination, it is prudent to begin testing for strength at the most distal site. This means first testing the strength of extension and flexion of the hallux against pressure of the examiner's hand. If weakened, the examiner moves up to assess the power of supination and pronation of the foot, then to ankle extension and flexion. Comparing symmetry of strength is important as one proceeds through the examination point by point, always testing in a consistent direction.

Should further evidence of strength muscle function be needed because of suspected slight weakness in the foot, evaluating walking on toes and heels is useful [30]. Both tasks place a high demand on the strength of the entire leg. They are a sensitive measure of weakness evaluated in a few steps.

Toe and heel walking (like testing for station and tandem walk) can be postponed until the end of the examination to minimize changing the patient's position.

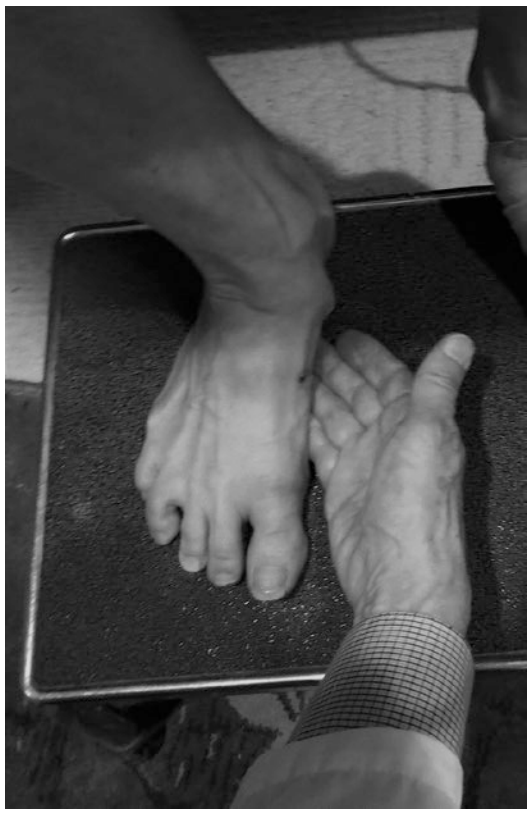

Fig. 28.6 Finger testing for fallen arches

Fasciculations

Fasciculations (twitches of the skin) are indicative of abnormal neural control of muscles. Their presence helps differentiate neuropathic damage from myopathy (primary muscle disease), a condition in which fasciculations do not occur.

In the legs, fasciculations are usually most visible in the anterior thighs. This sign of motor neuropathy is less apparent in the foot.

Hyporeflexia

Reduced or absent deep tendon reflexes, tested by knee and ankle jerks, represent neuropathy of either the afferent or the efferent limb of spinal cord innervation. When the reflexes of the limbs are reduced in the diabetic, it is likely to be both.

The ankle jerk is the more likely reflex to be diminished or disappear altogether when the lower lumbar and sacral nerves are involved. Absence of the knee jerk is usually a later complication, involving a more proximal level (and therefor shorter nerve length) of the spinal cord.

It must be kept in mind that these deep tendon reflexes may be barely detectable in normal persons. Often there is a discordant reaction between knee and ankle jerks, being strong in one set and weak in another. The technique, of course, requires uniform force from the reflex hammer when applied to comparable areas of the patella and Achilles tendons. Parenthetically, the head of a stethoscope, however convenient, is not an ideal substitute for the reflex hammer.

Deformities

The architecture of the foot becomes deformed from an imbalance of opposing muscle groups. That is, one side of a muscle pair involved in the flexion and extension of a joint dominates because of the weakness of its counterpart. The result, along with abnormal laxation of supporting ligament, is malformation of the foot and abnormal exposure to weight-bearing stresses. These conditions are described under the section "Skeletal System."

Autonomic Nervous System

Neuropathies involving the autonomic nervous system from diabetes are common but often overlooked or are attributed to other conditions, owing to their diffuse and nonspecific nature of autonomic functions. These complications may involve cholinergic, adrenergic, or peptogenic control.

Conversely, a wide spectrum of diseases may mimic the neurological complications of diabetes. Consider, for example, these confounding conditions in the diabetic: unawareness of hypoglycemia, gastroparesis, constipation, diarrhea, fecal incontinence (including nocturnal), erectile dysfunction, neurogenic bladder, sudomotor dysfunction, sinus node dysrhythmias, resting tachycardia, and orthostatic hypotension [31].

Neuropathies involving the autonomic nervous system are common to both. Type 1 and Type 2 diabetes. They may, in fact, be the first component of the nervous system to be damaged. It is not farfetched to conclude that individuals who have developed somatic sensory and motor neuropathies from diabetes may have already developed some degree of nerve derangement in the autonomic nervous system. Certainly, neuropathies in this system are more challenging to discern and difficult to document objectively.

How autonomic dysfunctions affect the foot is not easily appreciated, but they certainly must play some role in circulatory control and cellular viability. To assess involvement of the autonomic nervous system in the diabetic, one has to look well beyond the foot. An explicit patient history and some simple maneuvers performed at the bedside that involve various autonomic functions can be informative.

Sudomotor

Hyperhidrosis

Probably the most conspicuous disturbance of the autonomic neuropathies involves sweating. Rushes of sweating may occur in early stages of autonomic neuropathy, often brought on during eating (especially from spicy foods, a syndrome called "gustatory hyperhidrosis").

Anhydrosis

More important, however, is the reduction or absence of sweating ("anhydrosis") because it is a more advanced degree of neuropathy. The feet may be excessively dry, increasing the risk of developing cracks and fissures that invite microorganisms. Calluses are particularly susceptible to breakdown from drying. Paradoxically in autonomic neuropathies, dry feet may coexist with inappropriately excessive sweating occurring elsewhere such as in the face and on the hands, where neuropathy changes are of lesser degree.

Cardiovascular

Tachycardia

Neuropathic changes can affect heart rate by blunting automaticity and/or impulse conductivity. As an example, the heart rate at rest typically can be more rapid owing to impairment of the normal parasympathetic tone transmitted by the vagal nerve.

Postural Hypotension

Postural hypotension (with or without lightheadedness), for another example, may be demonstrated; observed is a sluggish acceleration of heart rate immediately on standing, along with an excessive fall in systolic and diastolic pressures from delayed peripheral vasoconstriction. (A procedure for evaluating postural changes has been described in Chap. 18 *Heart*.) A history of recurring light-headedness on first standing is corroborating evidence. The basis may be a failure of the heart to accelerate or of the peripheral vasculature to cinch up fast enough to adjust for the sudden change from a supine or sitting position to an upright position.

Vagal Hypotonicity

The normal, sudden sympathetic-driven acceleration of heart rate from deep inspiration may be blunted as may the subsequent slowing from increased vagal tone on sustained breath-holding. Also, the Valsalva maneuver does not induce the expected slowing of heart rate. Such changes have been documented in a third of diabetics who are still in their teens. Failure of the heart rate to slow on this stimulus has also been shown in hypertensive men who also had Type 2 diabetes; those with hypertension alone demonstrated a normal response [32]. The implication is that diabetes tends to impair control of parasympathetic reflexes, namely, on the tone of the vagus nerve.

Insensate Myocardium

A well-recognized liability of diabetics is the "silent" myocardial infarction or angina attack (i.e., a cardiac ischemic event without pain). The explanation lies in the impairment of sensitivity to pain from neuropathic complications. Furthermore, diabetes substantially increases the incidence of atherosclerotic disease of the coronary arteries.

Tolerance for exercise can be significantly reduced in the diabetic. Cardiovascular limitations certainly have a major role although adversities in other systems contribute.

Gastrointestinal

Disturbances of peristalsis and secretory function from diabetes may affect any portion of the gastrointestinal tract. Gastroparesis is fairly common in diabetes. It can be a prominent feature in the acute abdomen and, in fact, be the initiating event. Because it is often associated with nocturnal aspiration, gastroparesis is a risk factor for pneumonia. Diarrhea – sometimes nocturnal and alternating with constipation – reflects neuropathic involvement of the lower intestinal tract. Gastrointestinal neuropathy may have a center role in the acute abdomen.

Genitourinary

Incontinence in the diabetic suggests urogenital neuropathies with loss of bladder control. Other dysfunctions involve erection and ejaculation (which may be retrograde); failure of either is extremely common among older (but not necessarily old) diabetic men. A frequent complaint of women who have diabetes is vaginal dryness; this symptom may relate to neuropathic sudomotor dysfunction. Of course, there are other non-neuropathic conditions that merit consideration diagnostically.

Alternative Conditions

When visceral neuropathies are suspected in the diabetic, other primary causes should be considered as well. There are many such causes: pernicious anemia, alcoholism, chronic renal disease, heavy metal exposure, myxedema, and inflammatory demyelization syndromes such as syphilis and Lyme disease. Even the prolonged use of metformin in Type 2 diabetes is known to induce peripheral neuropathies, probably through depletion of vitamin B12 [33]. Contributing factors – such as aging, smoking, and peripheral arterial diseases – confound the expression of peripheral and autonomic neural dysfunctions.

Clues that suggest a cause of symptoms or signs not due to diabetic neuropathy are:

1. Motor dysfunctions appearing earlier and are of greater magnitude than sensory dysfunctions
2. Sensory or motor peripheral pathways having a highly asymmetric distribution
3. Neuropathic symptoms progressing rapidly

Peripheral Vascular System

Diabetes can adversely affect the major and minor blood vessels as well as the capillaries. Each component of the arterial and venous systems will be covered individually.

Despite the propensity of diabetics to develop ischemic arterial complications, blood flow in the foot may be several times that of normal. A curious paradox can thus be present: bounding pedal pulses in a warm foot where there are areas of localized deprivation of blood flow. These seemingly contradictory conditions, areas of hyperemia alongside areas of ischemia, are explained below.

Artery

Atherosclerosis

Obstructive arterial disease is a common contributing feature of foot ulcers in diabetes. Sustained hyperglycemia promotes the deposit of lipoprotein material within the atrial wall to form atheroma. Beginning with fatty streaks within the endothelium, this natural process accelerates in diabetes and progresses more rapidly to atherosclerotic plaques and ultimately to stenosis of the lumen.

Arterial insufficiency in the large vessels of the legs causes claudication and necrosis. Characteristic of claudication is discomfort (cramping, pain, or other discomfort) on exertion. The onset of claudication tends to occur with a predicted degree of effort, such as on walking a certain distance, known as the "time to claudication." Typically, the symptom subsides within a few minutes at rest, not like that caused by muscular overexertion or joint disease.

Claudication occurring in the calf is usually caused by obstruction in the superficial femoral artery in the thigh. Claudication in the foot is likely the result of stenotic disease in all three of the more distal branches. In the insensate foot, the symptom of claudication with activity may be absent altogether despite advanced arterial insufficiency.

Color, temperature, and texture – which are all affected in ischemic diseases – have been briefly covered earlier. Here, the focus is on the arterial pulses of the foot.

Branching from the superficial femoral artery in the popliteal area are three major arteries leading to the foot. Two are end arteries that are normally palpable: the dorsalis pedis (DP) and the posterior tibial (PT). The third is the peroneal artery; its branch to the surface lies over the lateral malleolus. This perforating peroneal (PP) is more difficult to palpate but is normally readily detected by Doppler ultrasonometry. The collateral circulation in the foot is so extensive that, normally, reduction of blood flow in one or even two of these arteries does not lead to foot ischemia. In diabetics, on the other hand, the arterial circulation may be so diffusely stenotic that collateral blood flow can be severely compromised.

The peripheral arteries can be graded roughly according to pulsatile dynamics:

+4 = Excessively bounding
+3 = Normal: robust
+2 = Normal: hypodynamic
+1 = Reduced
0 = Absent

For evaluation of vascular function, Doppler ultrasonometry can add a valuable extension of the physical examination. The bidirectional instrument provides a measure of the velocity and direction of blood flow, continuously responding to the reflection of high-frequency sound waves emitted from a handheld probe. The technique is relatively simple; a clinician can become competent fairly easily with the instrument. Its high degree of sensitivity provides an excellent method for assessing blood flow in the pedal arteries, even when flow is greatly diminished. For optimal waveform definition, it is important that the probe be held lightly but firmly at an angle to the skin at approximately 45 degrees and aimed cephalad.

Pulsatile flow in an artery can be observed in exquisite detail by Doppler ultrasonometry. During cardiac systole, the normal arterial pulsation exhibits a rapid upstroke from baseline (1), ending in a sharp peak (2) before descending in a somewhat slower downstroke (3) to baseline.

During cardiac diastole, the waveform dips slightly below baseline (4), representing the backward flow of blood caused by the elastic property of the artery distended in systole. Afterward, there is a small rise of the waveform (5) from resumption of forward blood flow from residual hemodynamic pressure (Fig. 28.7).

The waveform derived from Doppler ultrasonometry can disclose the characteristic appearance of a partially obstructed artery. In such cases, the waveform in cardiac systole has a more gradual upstroke (1); the amplitude is attenuated and the peak more blunted (2). Falloff toward baseline is somewhat slower (3). The reversed direction of blood flow may be reduced or absent altogether (4), owing to a reduction in wall elasticity. Late forward flow in the waveform may be attenuated or lacking (Fig. 28.8).

While waveforms can provide a sensitive profile of arterial function, they are not easily converted into a standardized format for measurement. Instead, a more practical technique is the systolic blood pressure determined by Doppler ultrasonometry. It is useful even when arterial pulsations are markedly diminished.

Systolic pressure determined by Doppler ultrasound is slightly higher than that obtained by auscultation. The explanation is simple: as the pressure in the cuff falls after arterial occlusion, the instrument reflects the sound of blood flow the instant it returns in the artery. Systolic sounds, in contrast, are not audible by stethoscope until there is enough blood flow to substantially distend the artery. Doppler ultrasonometry detects systolic blood pressure only; it is not useful for determining diastolic pressure.

Blood flow to the foot is readily assessed by comparing the arterial pressure in the ankle or foot with that in the arm. In the supine patient, the systolic pressure is determined over the posterior tibial or the dorsalis pedis artery. The pressure is then compared with that found over the brachial artery in the antecubital space [34]. This method is based upon the assumption that the brachial artery is not stenosed and therefore has normal blood flow (Fig. 28.9).

Normally, the arterial pressure in the foot is higher than in the arm (i.e., the ratio is greater than "1"). When pressure is lower in the foot, arterial obstructive disease is likely present [35]. A ratio less than 0.75% is diagnostic. An ankle-brachial ration that is less than 0.50% indicates severe ischemic disease of the legs. These criteria, while not infallible, do provide a high degree of reliability.

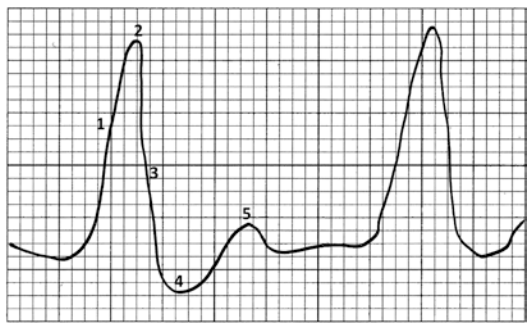

Fig. 28.7 Arterial waveform: normal

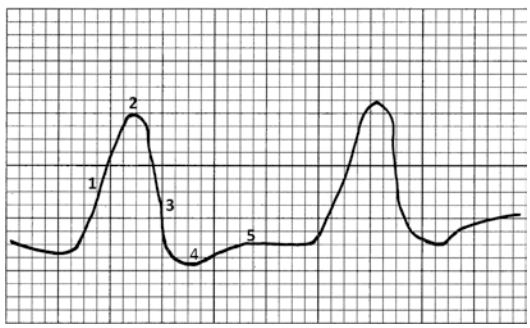

Fig. 28.8 Arterial waveform: partial obstruction

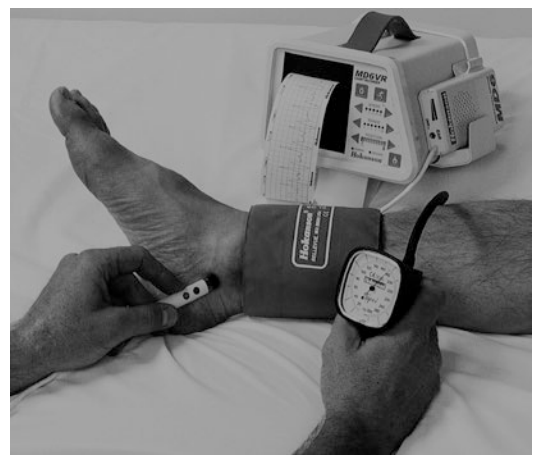

Fig. 28.9 Ankle/brachial index testing

The ease and reliability of establishing the ankle/brachial index provides a method of vascular function follow-up that is recommended on at least an annual basis (excepting the youngest patients). The procedure may reveal arterial insufficiency in the lower limb at a very early stage. It can provide serial measurements of established arterial stenosis. The periodic testing offers an objective incentive for the patient to maintain scrupulous glycemic control.

Thus far, this text covers stenosis of the artery from an atheromatous plaque. A second complication is disruption of the plaque. The consequences can lead to the formation of a thrombosis and/or to its release as emboli.

1. Thrombosis. A thrombosis occurring at the site of a ruptured atheroma may cause an acute obstructive ischemia. The site of ischemia can be at its origin or more distally where it may migrate. The subtended region within the arterial distribution will appear pale and cool. Arterial blood clots tend to be compact aggregations, largely of adherent platelets; they are quite different from the loose and friable, fibrin-rich clots that occur in veins.
2. Emboli. Clumps of the disintegrating plaque are released into the arterial circulation. Denoted "cholesterol emboli," they lodge into the distal tissues. A shower of emboli gives a clinical picture of spotty areas of pallor and cyanosis, most likely in the toes.

Arteriole

Vasoconstriction in arterioles is, of course, a physiological adaptation to adrenergic stimuli that alters the "resting" vascular tone. Having the greatest proportion of muscle tissue, the arteriole responds to sympathetic stimulation to a greater degree than do other segments of the arterial tree. They are distal to the palpable pulses in the hands and feet. Adrenergic stimuli causing vasoconstriction include exposure to cold, injury, hypotension, and high emotional stimuli. A form of extreme vasospasm occurs in Raynaud's phenomenon.

In neuropathy, impairment of sympathetic control of the arterioles alters vascular tone in favor of vasodilation. Vasoconstriction from norepinephrine stimulation is blunted. In addition, the damaged microcirculation is also subject to arterial-venous shunting. The result can actually be an increase in blood flow in a foot that feels warm but has areas, paradoxically, that are under perfused. In this complex vascular picture, the diabetic patient has a predisposition toward ischemia from stenotic arteries, while at the same time vasodilation occurs in smaller vessels in some areas of the foot. Adding further to the complexity, microscopic arterial-venous malformation may be present that increases the circulation in some locations while shunting it away from others.

Capillary refill time is a time-honored gauge for assessing blood flow to the nail bed. Apply pressure on the nail of the great toe with enough force to cause blanching in the vascular bed beneath and then observing the time for normal color to return. A delay exceeding 5 s has been considered an indication of peripheral ischemia, although this sign was found to have limited diagnostic value [36].

Capillary

We think of venous aneurysms and exudates in the retina and of glomerular disease in the kidney as organ-specific, microvascular complications of diabetes. Yet, the clinician should keep in mind that the damaging process occurring in the smallest blood vessels of these two tissues affects those throughout the body. It is simply that evidence of microvasculopathy visualized on fundoscopy and finding protein by urinalysis are the most readily detected. Furthermore, the severity of retinopathy and 24-h proteinuria is strong predictors of coexisting neuropathy.

Long-standing diabetes typically causes thickening of the basement membranes of capillary walls. The lumen of the capillary is thus narrowed, presumably by fragments of sorbitol and glucoproteins. The obstruction is aggravated by the leaking of carbohydrate-plasma protein that

tends to build up along (or "sugarcoat") the endothelium. At the same time, structural disarrangement of the capillary matrix weakens the wall, leading to the formation of microaneurysms and rupture.

Vein

A cardinal feature of venous disease is edema. Causes include (1) obstruction within or external to a vein or (2) reflux of valves within the veins. These conditions are mostly confined to the lower extremity and present critical issues in the evaluation and treatment of the foot in diabetes.

Of course, the microvasculature in the venous system is affected by hyperglycemia just as are the more well-recognized anomalies of the arterial system. We are familiar with diabetic venous aneurysms in the retina where they are visible but need to be reminded that the same phenomenon may be happening in other organ vascular beds.

Venous Thrombosis

Acute edema implies swelling of the ankle and calf within the past few days, with or without injury to the leg. When new-onset and unexplained swelling occurs in the lower legs and is unilateral or is asymmetrical, deep venous thrombosis should be considered. A recent leg injury or prolonged sitting increases suspicion. The condition could easily be passed off as a direct complication of the diabetic foot, therefore overlooking a life-threatening condition. Even so, the incidence of deep vein thrombosis is more frequent in the diabetic [37].

Venous Valvular Insufficiency

Chronic edema is typical of defective venous valves that normally function as our antigravitational system. There is a predilection of chronic venous insufficiency to cause edema predominantly in the lower calf, often sparing the foot in contrast to lymphedema. Concurring findings are cutaneous hemosiderosis (iron staining from capillary breakdown) and lipodermatosclerosis (thinning of skin over a tough fibrous subcutaneous layer). Inflammatory activity with cellulitis is often a complication of this condition. It is the potential for incurring infection in the swollen, inflamed leg, that is, of particular concern in the diabetic.

Documentation of edema – whether of acute or chronic onset – is well served by measuring the circumferences of the limb for serial comparison. Convenient and highly useful for serial follow-ups are measurements taken at maximum calf and at minimal ankle, described in Chap. 22.

Lymphatics

The relation of the lymphatic system – whether congenital or acquired – and diabetes is coincidental, not causal. Lymphedema results from a defect in the ability of the lymphatic system to pick up proteins within the extracellular fluids and return them to the circulation. Excessive extracellular proteins coupled with the hyperglycemia of diabetes mutually contribute to the injury of capillaries and perhaps to larger blood vessels.

Blood Viscosity

When viscosity of blood is increased, flow is reduced. This association becomes of increased significance in ischemic vascular beds.

In hyperglycemia, erythrocytes become stiffer as the HbA_{1c} increases. This reduced pliability interferes with their squeezing through capillaries. In addition, hyperglycemia is associated with an increase in fibrinogen, further increasing blood viscosity while impeding capillary flow. Hypoxia of cells is likely involving various systems a result of these dynamics.

Muscles

The skeletal muscles, by nature of their bulk, take up much more glucose than any other organ system. It is not farfetched to assume that the metabolic consequences of sustained hyperglycemia injure myocytes in proportion to that of the cells

of other systems. Yet accessing any direct injury to muscle cells independent of vascular and neural factors is difficult. Certainly, weakness and reduced muscle mass from diabetic myopathy occur, but quantitative measurement is challenging. The extent of muscular pathology can be obscured by coexisting adversities in other organ systems. In addition, the more limited physical activities imposed by neurological and circulatory complications of diabetes further compromise muscular function and mass.

Despite the difficulties in evaluating the stress of diabetes on muscle, there is evidence of direct injury by sustained hyperglycemia. Exaggerated "oxidative stress" on myocytes has been demonstrated in diabetics, at least partly in response to overproduction of free cell-toxic radicals released during the utilization of oxygen. In the diabetic, creatinine phosphate and lactate levels in blood during rest are elevated, suggesting an ongoing metabolic myopathy [38]. Diabetes is also known to have an untoward effect on the growth and regeneration of skeletal muscle [39].

Atrophy

Intrinsic muscles of the foot tend to atrophy in diabetes neuropathy. The degree of atrophy is a measure of the severity of motor dysfunction in the foot [40]. Generally, loss of muscle – that may be half of the normal volume – precedes the development of ischemic changes in the toes.

Dystrophic changes of muscle in the foot lead to imbalanced support on bearing weight. Because the extensors of the foot are generally more affected by weakness than the flexors, there is a tendency of the foot to supinate. The deviation leads to abnormal points of weight-bearing on the lateral edge of the foot where pressure-induced calluses and skin injury occur. When the flexors are weaker, the foot tends to pronate, imposing pressure on the plantar surface, mostly on the medial phalangeal-metatarsal joint.

Weakened intrinsic muscles of the foot also cause the arch to splay on bearing weight. These muscles extend to the metatarsal and interphalangeal joints. The disrupted balance of flexors and extensors typically draws the toes upward to a fixed "claw" or "hammer" position.

At the same time, the subcutaneous fat pad that cushions the metatarsal heads on the plantar surface is gradually displaced forward. Pressure of walking is thus exerted more directly on the metatarsal heads. With effective loss of the pressure-absorbing fat pad (added to the reduction in sensation, to circulatory disruption, and to infection), the problem of muscle atrophy of the foot takes a center role in the genesis of a neuropathic ulcer.

Infection

Infection of the fascia of muscle is a serious complication of diabetes. It extends rapidly to tendons and neighboring subcutaneous tissue as microorganisms dissect along fascial planes, virtually overnight in some cases. When fasciitis extends proximally up the tendon of the Achilles heel, spreading quickly into the lower leg is virtually inevitable.

Necrotizing Fasciitis

Necrotizing fasciitis is a highly destructive condition in which microvascular thrombosis and systemic toxicity are common complications. Necrotizing fasciitis may be the result of the "streptococcal toxic shock syndrome" [41]. The infection could also involve multiple invasive microorganisms. Diabetics are more susceptible to these infections and more likely than a nondiabetic to lose a limb from them [42].

Joints

To preserve a normal joint, the integrity of those supporting structures must be properly distributed. An important complication in diabetes is some deformity of the normal suspension system of the foot, namely, in the tendons and ligaments that normally preserve its structure and flexibility through the repeated stresses of standing and walking.

Repetitive stress on walking involves two forces. One is the vertical pressure of stepping down; the other is the horizontal pressure or shearing effect of forward momentum. Together, these stresses are, of course, readily tolerated in the normal foot. In long-standing diabetes, the pliability and strength of collagen fibers decline. Tendons are shortened and become more susceptible to tearing. The foot eventually bears weight in an abnormal distribution and is thereby subjected to excessive mechanical strain.

Ankle

Shortening of the Achilles tendon reduces the ability to dorsiflex the foot. Eventually, the muscle of the anterior compartment of the shin weakens. Overbalance of the gastrocnemius and soleus muscles tends to point the foot downward. When permanent, the condition is known as equinas deformans or equinovarus (referring to the hoof of the horse). This risk is exaggerated in the foot accustomed to high heels, allowing the Achilles tender to shorten.

Excessive weight on walking in equinovarus is then exerted onto a small area of the forefoot. This abnormal distribution of weight greatly increases the possibility of callus formation and eventually ulceration.

Arch

In the foot with stiff collagen fibers, the normal elastic compression and the springback of the arch with each step are both compromised. In diabetes, these changes are generally accompanied by weakness of muscle and neuropathy. The resulting fallen arch causes early fatigue on walking as well as greater susceptibility to weight-borne injuries.

Toes

The main site of tendon contraction in the diabetic is at the metatarsal heads. Frequent complications are the "claw toe" or "hammertoe." These deformities are illustrated in Chap. 24.

In the claw toe, the extensor tendon has shortened, pulling the proximal joint of the toe upward while the flexor tendon pulls the distal joint downward. The prominent knuckle is highly susceptible to the rubbing friction from the toe box of the shoe, while the tip of the toe, digging into the sole of the shoe, develops a blister or callus.

A hammertoe results from a bend in the middle joint of the toe. In addition, both the tendons of the metatarsal-phalangeal and the distal interphalangeal joints may shorten. The deformity presents a cocked-up toe (like a hammer) and a prominent area on the plantar surface where the pressure of weight-bearing is exaggerated. With it comes greater susceptibility to injury from the repeated trauma on walking and development of a callus. Both dorsal and plantar surfaces of a hammertoe are common places for corns or blisters to develop, each with the prospect of ulcerating.

Bones

The causes of bony deformities in the foot that beset the diabetic are multiple. Among them are reduced sensory neural signals and lax suspension fibers, added to chronic inflammation and increased osteoclastic activity, resulting in osteopenia of small bones. The microtrauma that may occur from the everyday stresses of walking is not fully repaired.

Subluxation

When the bones of the foot shift because of weakened supportive structures, their functional position is greatly disrupted. Such partial dislocations may appear first as a superficial deformity caused by subluxation of the bones of the joints, most commonly in the hindfoot. The displacement of the bone can occur suddenly with or without obvious trauma or may develop insidiously. Over time, dislocations cause serious disruption in the architecture of the foot and, with it, disturbances in walking. The result can be

woefully disabling, a condition known as the "Charcot joint."

The Charcot foot occurs in the presence of sensory neuropathy. It is, in fact, the lack of sensory input that appears to be responsible for the destructive changes within the stressed joint. Likely, the foot examination in the Charcot foot will disclose not only as a sensory but also as a motor neuropathy along with the absence a deep tendon reflex in the ankle.

Acute subluxation of a bone in the foot causes erythema, swelling, and increased warmth. Pain will, of course, be absent if the foot is insensate. Bulging of a malleolus can be an early clue (here, the importance of looking carefully for symmetry is stressed). Eventually, the signs of inflammation will subside but the deformity persists.

With subluxation of its smaller bones in the mid-foot, the footprint may suddenly become shorter and wider. The arch collapses and the foot rotates into pronation. On weight-bearing, pressure is exerted on the metatarsal-phalangeal joint on the plantar surfaces, usually at the greater toe and the second toe. In such misshapen feet, it is a common site of the "plantar ulcer." In contrast, rotation of the foot into supination puts pressure on the lateral surface of the foot where greater mechanical stress occurs.

The eponym refers to Jean-Martin Charcot, a renowned nineteenth century physician in Paris, who did much to explain the mechanism of "neurogenic arthropathy." He emphasized that the significance of acquired abnormal structure of the foot or ankle was overshadowed by more predominant, disabling features of a disease, as was common in later stages of syphilis. It was not until the 1930s, however, that a connection between these deformities of the feet was made with the neuropathy of diabetes. Before the "Age of Insulin," diabetics (and these were Type 1 diabetics) did not live long enough to incur such complications of bony architecture.

It is now well established that a sensory neuropathy is the basis for the Charcot joint. Furthermore, the presence of this deformity is a definite risk factor for development of a foot ulcer, infection, and amputation [43].

Fracture

Fractures in the foot or ankle can occur more readily when the natural balance of weight-bearing structures is disturbed. These may be unsuspected if sensory neuropathies are also present. Redness and swelling are clues, but these, too, may be absent. In addition to inducing abnormal alignment of the bone, fragments of the bone provide edges that are apt to cause further injury of soft tissue with each step.

Generally, a standard x-ray is sufficient to demonstrate a fracture. Sometimes, though, the fracture is not the easily identified separation of bone fragments; rather it is a fissure. Such a "crack fracture" may not become evident on x-ray until a week or two after an injury when aseptic necrosis has widened the visible gap between fragments.

Infection

Osteomyelitis can occur in three ways: (1) skin-penetrating wounds, (2) spread from a local soft tissue infection, and (3) hematogenous seeding. The insensate foot of the diabetic foot lacks the warning symptoms of pain and the discomfort of infection. Fever and local signs such as erythema and warmth need not be present. Indeed, with neuropathic complications, osteomyelitis may be entirely asymptomatic. Furthermore, the sites of possible microbial invasion – abrasions, punctures, and cracks in the skin – may not be evident.

Sometimes, an infection of the bone is announced by becoming difficult to control diabetes, perhaps along with occult fever or other constitutional features. There should also be a high level of suspicion of osteomyelitis when cellulitis or a wound fails to improve with seemingly appropriate and comprehensive therapy.

By far, the most frequent organism causing osteomyelitis is *Staphylococci aureus*. Those species of staph that are resistant to antibiotics (MRSA) are being detected with increasing frequency. Yet, mixed flora in the diabetic osteomyelitis is nevertheless common. *Salmonella* is often the causing organism in sickle cell disease

and in locales where general sanitary facilities are sorely compromised. Anaerobic pathogens, including *Clostridium difficile*, are among the more unusual organisms sequestered in the bone. *Pseudomonas* is more likely in patients with open wounds exposed to polluted water.

The laboratory may provide some nonspecific yet often helpful information that suggests osteomyelitis. These findings include an elevated level of C-reactive protein (CRP) in the blood in the presence of leukocytosis and a rapid erythrocyte sedimentation rate (ESR). Samples for bacterial identification obtained by percutaneous needle or small incision biopsy are recommended where osteomyelitis is strongly suspected. Obviously, identification of the infecting organism is critical for effective treatment.

When the bone becomes infected, it takes about 2 weeks before osteal destruction is enough to give signs of osteomyelitis that can be identified on x-ray. Even when it is, the extent of bone destruction is often much larger than is radiologically apparent. Differentiation must be made between osteomyelitis and reactive edema in the bone marrow. For these reasons, magnetic resonance imaging (MRI) is advantageous to x-ray when osteomyelitis is suspected [44].

Footwear

Care of the feet in a person with diabetes requires fastidious attention to the shoes. They must be wide enough with the toe box high enough to prevent untoward pressure or rubbing. Athletic footwear with highly cushioning soles is desirable. The ideal shoe has vents or wicks for minimizing the buildup of moisture.

A comprehensive examination of the diabetic foot should include shoes or other footwear that is habitually used. Differences of weight-bearing dynamics can be quickly appreciated by inspecting the shoes. Look and feel for seams or bumps inside for irregularities that may prove injurious. Uneven wear of a shoe is a clue for abnormal weight-bearing surfaces. A place to compare wear is the back of the heels. Uneven edges of the shoe heel indicate a problem in distributing the weight, such as a leg length discrepancy. This observation is a highly sensitive reflection of an unbalanced load during walking.

A difference in leg length is a common but seldom recognized abnormality in the general population. Confirmation can be performed in seconds. With the patient supine, place the thumbs just distal to the medial malleoli. If the axial skeleton is lined up perfectly, the thumbs will be opposite to each other when legs are of equal length. If not, the thumb on the shorter leg is closer to the head. A difference of 2.0 cm or greater is highly significant.

Ill-fitted footwear is a major precipitating cause of ulcer formation in the diabetic. Aggravating factors are high heels and cramped toe boxes. The best fitting shoes are the ones that are comfortable the moment they are put on; they do not need "breaking in." Shoes should be checked frequently for tears, sharp edges, and foreign bodies.

Often, minor abnormalities of the foot arches can be corrected with simple arch supports. An inserted heel lift can help alleviate a leg length discrepancy. Caution must be exercised to avoid overcorrecting. At first, only partial correction of the fallen arch or shortened leg is appropriate.

For persons with major foot deformities, custom-made footwear is meant to off-load weight from vulnerable areas and distribute it more advantageously. Such individually designed shoes pertain especially for management of hammertoes and Charcot foot. For these special patients, professionally designed footwear – while expensive – can provide immeasurable benefit.

Contributing Factors

The mainstay of care of the diabetic patient has long been proven to be a meticulous control of blood sugar. There is a strong relationship between increased hyperglycemia and complications of all kinds with those of the foot most common. In addition to devising a strategy to maintain physiological glycemic levels related to activity, eating, and sleeping, the clinician is expected to

♫ DIABETIC FOOT: CHECK OFF LIST ♫

Name_____ _ RIGHT / LEFT Date ___/___/__

A. SKIN

1. Color	/	
2. Texture	/	
3. Moisture	/	
4. Temperature	/	
5. Callus formation	/	
6. Spots	/	
7. Wound	/	

site, area, depth, exudate, odor

B. NEUROLOGY

Sensory:

8. Light touch	/	
9. Sharp	/	
10. Vibration (128 cps)	/	
11. Station	/	
12. Tandem walk	/	

Motor:

13. Atrophy	/	
14. Strength: Toe	/	
Ankle	/	
Knee	/	
15. Knee DTR	/	
16. Ankle DTR	/	
17. Toe walk	/	
18. Heel walk	/	

Autonomic:

19. Symptoms _____

20. Cardiac rate _____

C. CIRCULATION

Arterial:	21. Dorsalis pedis pulse	/	
	22. Posterior tibial pulse	/	
	23. Ankle/brachial index	/	= ____ %
Venous;	24.Varicosities	/	
	25.Venostasis	/	

D. MUSCULO-SKELETON

26. Range of motion	/	
27. Deformity	/	

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manage a complex array of contributing pathologies: hypertension, hyperlipidemia, anemia, thrombogenicity, and obesity. The patient must assiduously attend to a lifestyle that incorporates healthy everyday issues: medications, diet, activity (including exercise), personal hygiene, footwear, tobacco abstinence, and limited alcohol consumption. All these have been accomplished by men, women, and children who have learned to lead a vigorous and productive lifestyle free of complications while expecting a normal expectancy of life.

Those who are diabetic and those who care for them have abundant resources of materials and information to apply them effectively. A symposium on the Standard Medical Care in Diabetes by the American Diabetes Association looks at this massive but realistic challenge [45].

An inter-disciplinary group of vascular and podiatric specialists has recently published a consensus statement on "Management of the Diabetic Foot." It appears in the Journal of the American Medical Association [46].

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